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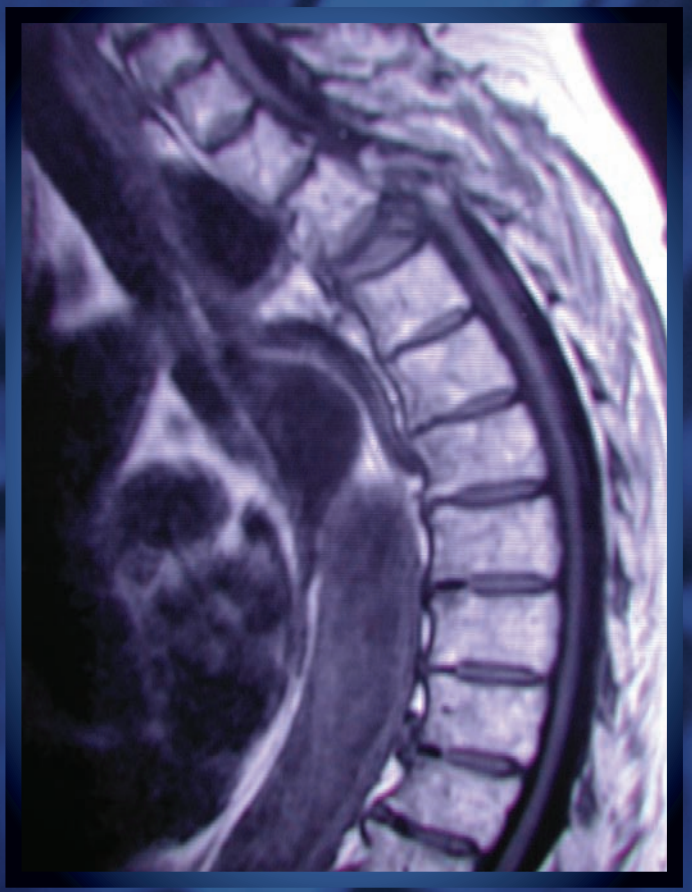
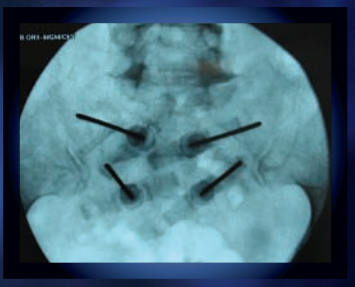
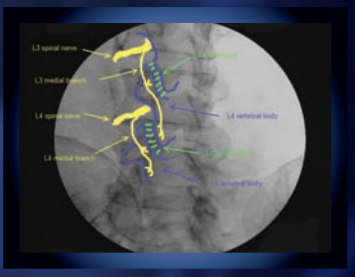
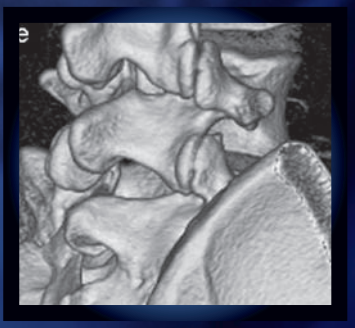
Evidence-Based Interventional Spine Care









MICHAEL J. DEPALMA

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Editor
Michael J. DePalma, MD
Associate Professor
Department of Physical Medicine and Rehabilitation
Medical College of Virginia Hospitals
Medical Director
Virginia Commonwealth University Spine Center
Director
Interventional Spine Care Fellowship
Virginia Commonwealth University Hospitals
Richmond, Virginia

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Contents

Preface

Recognition, awareness, and innovation are essential to reduce the costs associated with the prevalent painful spinal disorders facing all of us as practitioners, patients, and payers. The knowledge to critically evaluate and assign predictive meaning to data allows us to prescribe high-yield diagnostic and therapeutic care in a responsible fashion. By using best available evidence, spine clinicians can ideally accurately diagnose the source of symptoms in order to optimally treat and rehabilitate the patient. Within this process, supported interventions are promoted while needless ones can be discarded. Knowledge, skill, and cooperation are the prerequisites to advocate for our patients to protect their access to appropriate spine care.

The scope of *iSpine* reflects an algorithmic approach of interventional spine care to the diagnosis and treatment of a variety of painful spinal disorders. The book is organized to expound why certain conditions should be suspected and recognized by increasing the reader's awareness of the manifestations of these disorders. Key chapters highlight historical features that possess predictive diagnostic value, and others present physical examination findings that help discriminate symptomatic from non-symptomatic structures. The findings of these clinical evaluations help direct what diagnostic testing—imaging, electrical, or procedural—should ensue. The evaluating clinician can then assign clinical meaning to morphologic abnormalities revealed by imaging studies. The success of treatment interventions will be enhanced by appropriate technique and proper utilization after rendering tissue-specific diagnoses. Descriptions of acceptable spine biomechanics and ergonomic variables have been included to illustrate strategies to reduce spine strain and recurrent injury.

The list of contributing national and international authors—all of whom are leading experts in their fields—spans many specialties, including physiatry, anesthesiology, radiology, biomechanical engineering, physical therapy, orthopedics, neurosurgery, internal medicine, and rheumatology. This amalgam of specialists reflects the bedrock principles of interventional spine care: diagnose and treat the structural source of the painful spinal disorder in an outcomes oriented manner.

Michael J. DePalma, MD

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I would like to acknowledge and thank key individuals whose support, guidance, and investment fueled my pursuit of this project.

To my mentors, Ernie Johnson, who taught me the nuances of electrodiagnostic medicine, David Cifu, who taught me the art of bringing individuals to the table, and Curtis Slipman, who instilled in me the perspective of how to successfully practice a vastly challenging subspecialty and, more importantly, how to accomplish despite defeat.

To my parents, Ann and Jim, who fostered a respectable and productive work-ethic, which became my modus operandi. I could not have achieved what I have without your tireless love and support.

To my wife, Arpita: Her name means "devoted to" and how appropriate. You must understand that you have my utmost gratitude, respect, and love because of everything you are. Without you, many people would be incomplete, and I could not have persevered as I have.

To my children, Sarina and Nikhil, I hope this project exemplifies how I have learned to rely on others on so many fronts to accomplish something for the greater good. Let this notion be a guiding light.

To all of my colleagues and peers, thank you for your brotherly or sisterly advice and your selfless willingness to assist when I have asked. The integrity of our fraternity is immeasurable.

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Contributors

Divya Agrawal, MD
Clinical Instructor
Department of Orthopaedic Surgery
Stanford University
Redwood City, California

Venu Akuthota, MD
Associate Professor and Vice Chair
Director, Spine Center and Pain Fellowship
Department of Physical Medicine and Rehabilitation
University of Colorado School of Medicine
Denver, Colorado

Kenneth M. Alò, MD
Pain Management
The Methodist Hospital
Clinical Member
The Methodist Hospital Research Institute
The Texas Medical Center
Houston, Texas
Director
Section of Neurocardiology
Monterrey Technical University
Monterrey, Mexico

Ray M. Baker, MD
Director, Swedish Spine Program
Swedish Patient Center
Seattle, Washington

Les Barnsley, B Med(Hons) GradDipEpi, PhD, FRACP, FAFRM(RACP)
Head, Department of Rheumatology
Concord Hospital
Associate Professor
Department of Medicine
Sydney Medical School
Sydney, New South Wales, Australia

Douglas P. Beall, MD
Chief of Services
Department of Radiology and Interventional Pain Management
Clinical Radiology of Oklahoma
Oklahoma City, Oklahoma

Elizabeth Beck, MD
Department of Physical Medicine and Rehabilitation
Spaulding Rehabilitation Hospital
Wellesley, Massachusetts

Frank Bender, MD
Clinical Assistant Professor
Department of Physical Medicine and Rehabilitation
The Ohio State University
Columbus, Ohio
Neuromedical Center
Baton Rouge, Louisiana

Sarjoo M. Bhagia, MD, MS
Interventional Physiatrist
OrthoCarolina Spine Center
Charlotte, North Carolina

Amit Bhargava, MD, MS
Director
Advanced Interventional Pain and Sports Medicine Center
Owings Mills, Maryland

Nikolai Bogduk, MD, PhD, DSc
Newcastle Bone and Joint Institute
University of Newcastle
Newcastle, United Kingdom

Guiseppe Bonaldi, MD
Director, Interventional and Diagnostic Neuroradiology
Department of Endovascular and Spine
Ospedali Riuniti
Bergamo, Italy

Rachel Brakke Holman, MD
Senior Instructor
Department of Physical Medicine and Rehabilitation
University of Colorado School of Medicine
Denver, Colorado

Diane W. Braza, MD
Associate Professor
Department of Physical Medicine and Rehabilitation
Medical College of Wisconsin
Milwaukee, Wisconsin

Jonathan Carlson, MD
Clinical Assistant Professor
Midwestern Medical School
Glendale, Arizona

Anthony Chang, MD
Assistant Attending Radiologist
Hospital for Special Surgery
Assistant Attending Radiologist
New York-Presbyterian Hospital
Assistant Professor of Radiology
Weill Cornell Medical College
Hospital for Special Surgery
New York, New York

Larry H. Chou, MD
Medical Director
Sports and Spine Rehabilitation Division
Premier Orthopaedic and Sports Medicine Associates
Havertown, Pennsylvania

David W. Chow, MD
Medical Director
California Spine Center
Walnut Creek, California

Alessandro Cianfoni, MD
Assistant Professor of Neuroradiology
Director of Interventional Spine Neuroradiology
Department of Radiology
Medical University of South Carolina
Charleston, South Carolina

Steven P. Cohen, MD
Professor
Department of Anesthesiology
Walter Reed Army Medical Center
The Johns Hopkins School of Medicine
Washington, DC

Timothy Deer, MD
President and CEO
Center for Pain Relief
Charleston, West Virginia

Michael J. DePalma, MD
Associate Professor
Department of Physical Medicine and Rehabilitation
Medical College of Virginia Hospitals
Medical Director
Virginia Commonwealth University Spine Center
Director
Interventional Spine Care Fellowship
Virginia Commonwealth University Hospitals
Richmond, Virginia

Timothy R. Dillingham, MD, MS
Professor and Chairman
Department of Physical Medicine and Rehabilitation
Medical College of Wisconsin
Milwaukee, Wisconsin

Ron Donelson, MD, MS
President
SelfCare First
Hanover, New Hampshire

Omar El Abd, MD
Interventional Spine Director
Spaulding Newton Wellesley
Spaulding Rehabilitation Hospital
Wellesley, Massachusetts
Clinical Instructor
Department of Physical Medicine and Rehabilitation
Harvard Medical School
Boston, Massachusetts

Frank J. E. Falco, MD
Adjunct Associate Professor
Temple University Medical School
Philadelphia, Pennsylvania
Director
Pain Medicine Fellowship Program
Temple University Hospital
Philadelphia, Pennsylvania
Medical Director
Mid Atlantic Spine and Pain Physicians
Newark, Delaware, and Elkton, Maryland

Fabrizio Fasano, MD
Fellow in Radiology
Department of Neuroradiology
University Hospital S. Salvatore
L'Aquila, Italy

Sue A. Ferguson, PhD
Senior Research Associate Engineer
Department of Integrated Systems Engineering
Biodynamics Laboratory
The Ohio State University
Columbus, Ohio

Joseph D. Fortin, DO
Medical Director
Spine Technology and Rehabilitation
Fort Wayne, Indiana

Michael Frey, MD
Advanced Pain Management and Spine Specialists
Fort Meyrs, Florida

Michael B. Furman, MS, MD
Orthopedic and Spine Specialists
Temple Univeristy
York, Pennsylvania

Massimo Gallucci, MD
Professor of Radiology and Neuroradiology
Department of Neuroradiology
University Hospital S. Salvatore
L'Aquila, Italy

Stephanie Geffert, MLIS
Pain Medicine Fellowship Program Coordinator
Temple University Hospital
Philadelphia, Pennsylvania
Director of Research and Education
Mid Atlantic Spine and Pain Physicians
Newark, Delaware, and Elkton, Maryland

Michael C. Geraci, Jr., MD, PT
Director
Sports Medicine and Fellowship Program
Buffalo Spine and Sports Institute, PC
Buffalo, New York
Clinical Associate Professor
Department of Physical Medicine and Rehabilitation
College of Osteopathic Medicine
Michigan State University
East Lansing, Michigan

Lars G. Gilbertson, PhD
Director, Spine Research Laboratory
Center for Spine Health
Cleveland Clinic
Cleveland, Ohio

Jared S. Greenberg, MD
Interventional Spine Specialist
Department of Physical Medicine and Rehabilitation
Meriter Medical Group
Madison, Wisconsin

Anita Gupta, DO, PharmD
Assistant Professor
Department of Anesthesiology
University of Pennsylvania
Philadelphia, Pennsylvania

Steven R. M. Helper, MD
Clinical Instructor
Department of Physical Medicine and Rehabilitation
University of British Columbia
Vancouver, British Columbia, Canada

Patrick W. Hogan, DO
Clinical Assistant Professor
Midwestern Medical School
Glendale, Arizona

Christopher W. Huston, MD
Spine Consultant, NBA Phoenix Suns and WNBA Phoenix Mercury
The Orthopedic Clinic Association
Phoenix, Arizona

Zacharia Isaac, MD
Director, Interventional Physiatry
Brigham and Women's Hospital
Instructor
Department of Physical Medicine and Rehabilitation
Harvard Medical School
Director, Brigham and Women's Spine Center
Spaulding Rehabilitation Hospital
Boston, Massachusetts

Mary E. Jensen, MD
Director, Interventional Neuroradiology
Professor of Radiology and Neurosurgery
University of Virginia Health Systems
Charlottesville, Virginia

Ernest W. Johnson, MD
Professor Emeritus
Department of Physical Medicine and Rehabilitation
The Ohio State University
Columbus, Ohio

Gwendolen Jull, PT, PhD
Professor of Physiotherapy
School of Health and Rehabilitation Sciences
The University of Queensland
Queensland, Australia

Leonardo Kapural, MD, PhD
Chronic Pain Center
Wake Forest University Health Sciences
Winston-Salem, North Carolina

Mark Makumbi Kayanja, MD, PhD
Department of Orthopaedics
Cleveland Clinic
Cleveland, Ohio

John C. Keel, MD
Medical Director
Spine Center at Beth Israel Deaconess
Instructor
Harvard Medical School
Boston, Massachusetts

David J. Kennedy, MD
Assistant Professor
Department of Orthopedics and Rehabilitation
University of Florida
Gainesville, Florida

Wade King, MB, BS, MMedSc , MMed (Pain Mgt), GDMuscMed, FAFMM
Bone and Joint Institute
Royal Newcastle Centre
Newcastle, New South Wales, Australia

Sarah Knievel, MD
Pain Medicine Fellow
Department of Anesthesiology
Mayo Clinic
Rochester, Minnesota

Tim Lamer, MD
Associate Professor of Anesthesiology
Department of Anesthesiology
Mayo Clinic
Rochester, Minnesota

Izzy Lieberman, MD
Spine Surgeon
Texas Back Institute
Plano, Texas

Nicola Limbucci, MD
Assistant Radiologist
Department of Radiology
City Hospital
Pescara, Italy

Kaixuan Liu, MD, PhD
Chief of Endoscopic Spine Surgery
Atlantic Spinal Care, LLC
Edison, New Jersey

Gregory E. Lutz, MD
Associate Professor of Clinical Rehabilitation Medicine
Weill Medical College of Cornell
Physiatrist-in-Chief
Hospital for Special Surgery
New York, New York

Jean-Yves Maigne, MD
Head, Department of Physical Medicine
Hôpital Universitaire Hôtel-Dieu de Paris
Paris, France

Stephen May, PhD
Senior Lecturer in Physiotherapy
Sheffield Hallam University
Sheffield, United Kingdom

William S. Marras, PhD
Professor and Director
Department of Integrated Systems Engineering
Biodynamics Laboratory
The Ohio State University
Columbus, Ohio

S. Ashley McCowen, MD
Department of Non-operative Spine Care
The Orthopaedic Center of Central Virginia
Lynchburg, Virginia

Stuart M. McGill, PhD
Professor of Spine Biomechanics
Department of Kinesiology
University of Waterloo
Waterloo, Ontario, Canada

Douglas Mintz, MD
Clinical Associate Professor
Department of Radiology
Florida International University
Miami, Florida

Stefan C. Muzin, MD
Clinical Instructor
Harvard Medical School
Department of Orthopedic Surgery
Beth Israel Deaconess Hospital
Boston, Massachusetts

Wayne Olan, MD
Director of Neuroradiology
Suburban Hospital
Betheseda, Maryland
Director of Interventional and Endovascular Neurosurgery
The George Washington University
Washington, DC

Amish R. Patel, DO, MPH
Assistant Professor
Division of Physical Medicine and Rehabilitation
Department of Orthopedic Surgery
University of Toledo Medical Center
Toledo, Ohio

Rajeev K. Patel, MD
Associate Professor
Department of Orthopedics and Rehabilitation
University of Rochester Spine Center
Rochester, New York

Kevin Pauza, MD
Texas Spine and Joint Hospital
Tyler, Texas

Justin J. Petrolla, MD
Interventional Pain and Musculoskeletal Specalist
Colorado Pain and Rehabilitation
Wheat Ridge, Colorado

Peter E. Pidcoe, PhD, PT, DPT
Associate Professor
Department of Physical Therapy
Virginia Commonwealth University
Richmond, Virginia

Joel Press, MD
Professor and Medical Director
Spine and Sports Rehabilitation Centers
Department of Rehabilitation Medicine
Columbia Feinberg/Northwestern School of Medicine and Rehabilitation Institute of Chicago
Chicago, Illinois

Todd M. Reiter, MD, DC
Medical Director
Inpatient Rehabilitation Center
High Point Regional Health Systems
High Point, North Carolina

Erich O. Richter, MD
Assistant Professor
Department of Neurosurgery
Louisana State University Health Science Center
New Orleans, Louisana

Manuel Fanor Saavedra, MD
Neurosurgery Resident
Department of Surgery
Neurosurgery Section
University of Puerto Rico
San Juan, Puerto Rico

Thomas R. Saullo, MD
Interventional Spine Fellow
Department of Physical Medicine and Rehabilitation
Virginia Commonwealth University
Richmond, Virginia

Jerome Schofferman, MD
Director, Research and Education
San Francisco Spine Institute
SpineCare Medical Group
Daly City and San Francisco, California

Edward D. Simmons, MD, FRCS(C)
Clinical Professor
Department of Orthopaedic Surgery
State University of New York at Buffalo
Buffalo, New York

Curtis W. Slipman, MD
Miami, Florida

Matthew Smuck, MD
Assistant Professor
Orthopaedic Surgery
Director
Stanford Interventional Spine Center
Redwood City, California

Jeff Strakowski, MD
Clinical Associate Professor
Department of Physical Medicine and Rehabilitation
The Ohio State University
Associate Director of Medical Education
Department of Physical Medicine and Rehabilitation
Riverside Methodist Hospital
Columbus, Ohio

Philip Tasca, MD
Assistant Clinical Professor
Department of Rehabilitation Medicine
Columbia University
New York, New York

Joshua A. Thomas, DO, MBA
Attending Physician
Interventional Pain Management
Capitol Spine and Pain Centers
Fairfax, Virginia

Jayme Trahan, MD
Department of Neurosurgery
Louisana State University Health Science Center
New Orleans, Louisana

Brian S. Trussell, MD
Medical Director
Attending Physician
Department of Interventional Spine
Interventional Spine and Sports Institute
Hattiesburg, Mississippi

Paul E. Verrills, MBBS, FAFMM, GDMM (Hons), MM (Pain Management), FIPP
Musculoskeletal and Interventional Pain Physician
Co-Medical Director
Metro Group of Pain Clinics
Melbourne, Victoria, Australia

Christopher J. Visco, MD
Assistant Professor
Department of Rehabilitation and Regenerative Medicine
Columbia University College of Physicians and Surgeons
New York, New York

David G. Vivian, MBBS, FAFMM, GDMM, MM (Pain Management)
Musculoskeletal and Interventional Pain Physician
Co-Medical Director
Metro Group of Pain Clinics
Melbourne, Victoria, Australia

Sayed E. Wahezi, MD
PC Fellow
Spine Technology and Rehabilitation
Fort Wayne, Indiana

John B. Weigele, MD, PhD
Assistant Professor of Clinical Radiology
Department of Radiology
Hospital of the University of Pennsylvania
Philadelphia, Pennsylvania

Wade Wong, DO, FACR, FAOCR
Professor of Clinical Radiology and Anesthesiology
University of California, San Diego
La Jolla, California

Yinggang Zheng, MD
Attending Physician
Desert Institute for Spine Care
Phoenix, Arizona

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1 Etiologies of Painful Spinal Disorders

Christopher W. Huston and S. Ashley McCowen

INTRODUCTION

The lifetime prevalence of neck pain has been estimated at 67% [1]. At any given time, the prevalence of neck pain is 44%; up to half of those experiencing neck pain may have chronic pain lasting for 3 months or more. Although motor vehicle collisions and falls are the most common traumatic causes of neck pain [2], less than 5% of neck pain can be attributed to trauma [3]. Although most individuals with neck pain recover within 6 months [2], neck pain can become persistent and is responsible for up to 11% of time lost on the job [4]. The socioeconomic burden in the United States on an annual basis is more than 29 billion dollars [5]. In addition, neck pain has been determined to contribute to depressive symptoms in up to 24% of individuals experiencing such pain [6]. Secondary fibromyalgia has been reported to develop in up to 21% of patients experiencing cervical trauma [7].

In 1990, there were approximately 15 million office visits to U.S. physicians for mechanical low back pain (LBP) [8]. This does not include visits to allied health professionals such as chiropractors, which could increase this number to 30 million visits [9]. LBP is the second most common reason for visit to a primary care physician. Chronic LBP is the third leading cause for disability in the 45-to-65 age group. Medical costs are estimated at 30 to 50 billion dollars [10]. Consequently, mechanical LBP is a major health care concern for affected individuals, employers, providers, payers, and administration.

In 1966, a general practice group reported 470 patients suffered an episode of LBP from a clinic population that grew from 5713 to 6920 clinic patients over the study period of 4 years. Within 2 weeks, 62% of the episodes resolved although 44.6% experienced a recurrence. The general practitioners noted no evident cause of LBP in 79.3% of males and 88.8% of females. However, the paper does not discuss the efforts to diagnose the cause of LBP. Even by 1990 Nachemson stated that the cause of LBP in the majority of patients is unknown [11]. In 2001, Deyo and Weinstein stated that 85% of patients with LBP cannot be given a precise pathoanatomical diagnosis [12]. They note that imaging studies are not reliable in diagnosing the cause of LBP. The often used diagnoses of strain and sprain have not been anatomically or histologically characterized, and patients given these diagnoses more accurately suffer from idiopathic LBP [12] caused by injury of an underlying spinal structure [13–16]. The ability to provide a more precise diagnosis could lead to a better understanding of chronic LBP. Furthermore, more specific treatment could be rendered leading to a higher success rate for treatment outcomes and less disability effectively reducing the financial and economic burden of chronic cervical and lumbosacral pain.

With the advent of fluoroscopy and advancements in interventional spine care, specific spinal structures can be targeted and assessed. Such an approach either relies on pain provocation by tissue stimulation or index pain reduction by injection of local anesthetic. In neck pain and LBP sufferers, diagnostic zygapophyseal joint and sacroiliac joint (SIJ) injections, for example, using local anesthetics, have been developed to identify the structure causing axial spine pain. To control for a placebo effect, a double block paradigm has been studied [13,16–18]. With the double block paradigm, patients are injected with anesthetic of different half lives with the patient blinded to the medication injected. A positive diagnostic injection would be 80% decrement in pain including provoking maneuvers with at least 30 minutes relief with 2% xylocaine and 3 hours relief with 0.5% bupivacaine. In addition, the correct response would be longer relief with bupivacaine than xylocaine. Fluoroscopically guided diagnostic blocks utilizing local anesthetic are currently the gold standard for identifying pain emanating from the zygapophyseal joint and SIJs.

To consider a structure to be a cause of axial spine pain, we support a modification of previously proposed criteria [19]. The structure should be: (1) innervated with nociceptive fibers; (2) able to produce pain clinically seen and able to induce pain in normal volunteers; (3) susceptible to disease or injury known to cause pain; and (4) a diagnostic test to identify the structure as a cause of pain—relief with injection of local anesthetic targeted to the structure or exact reproduction.

Various structures have been purported to cause axial spinal pain. The four criteria will be applied and discussed for each of these structures in the cervical and lumbar spine. There may be some redundancy with the format of separating data for the cervical and lumbar spine. However, one cannot assume that findings from studies in the lumbar spine necessarily translate to the cervical spine. In addition, many readers often use textbooks as references.

Dividing the cervical and lumbar spine allows the reader to quickly review subsections of interest.

CERVICAL SPINE

Muscle

The muscles of the cervical spine are dynamic actors that provide support, movement, and stabilization. Flexor muscles acting to flex the occiput include the longus capitis and rectus capitis; occiput extensors include the splenius capitis, semispinalis capitis, and longissimus capitis. Flexor muscles of the cervical spine include the scalenes and sternocleidomastoid; cervical extensors include the semispinalis cervicis, longissimus cervicis, and splenius cervicis. The multifidus muscles assist in flexion and rotation of the cervical spine. The medial branches of the dorsal rami innervate multifidis and semispinalis cervicis, whereas the lateral branches innervate the superficial muscles longissimus and splenius [20].

Cadaveric studies demonstrate lesions of the muscle after trauma [21]. Numerous in vivo studies document muscle dysfunction in patients with neck pain including decreased strength, muscular imbalance between cervical flexors and extensors [22], fatty infiltration of extensor muscles [23], decreased range of motion, decreased bulk, and increased activity of the accessory muscles [24–28]. However, there is no study demonstrating whether these are learned responses to or actual causes of cervical pain. Injection into cervical musculature has produced pain suggesting that muscle can be a source of neck pain [29].

Yet, there is no clinical or radiologic evidence that identifies muscle as the cause of neck pain. Although muscle pain may be a part of the 35% of undiagnosed neck pain, muscle injuries usually heal within 3 months [30].

Ligament

The upper cervical spine encompasses the occiput, axis, and atlas. Ligaments at these levels provide stability and absorb energy. The apical ligament originates on the posterior superior aspect of the odontoid process and inserts on the anterior wall of the foramen magnum. The transverse ligament stabilizes the odontoid process to the atlas. Alar ligaments originate on the odontoid process to insert on the lateral walls of the foramen magnum and lateral masses of C1 [31].

The lower cervical ligaments are classified as anterior, middle, and posterior. The anterior comprises the anterior longitudinal ligament (ALL), which consists of layers of fibers of several lengths and alignment that span the anterior surface of the vertebral bodies and are tightly adherent to the vertebral body as well as confluent with the anterior annulus fibrosus. Anterior ligaments protect against significant extension injury [32]. Further study is required to identify the cervical ALL as an independent cause of pain.

The posterior longitudinal ligament lines the floor of the spinal canal and comprises the superficial, intermediate, and deep layers, which span the intervertebral disc and attach to the vertebral bodies [33,34]. The posterior longitudinal ligament (PLL) is innervated by the sinuvertebral nerve [35,36].

Posteriorly, the capsular ligaments provide connection between facet joints of adjacent ipsilateral vertebra. These capsules are innervated by the cervical dorsal rami of the adjacent level above and below [20]. The ligamentum flavum are paired ligaments, which connect adjacent laminae. Interspinous ligaments are not well developed in the cervical spine but provide attachments between adjacent spinous processes. The supraspinous ligaments provide attachment between the tips of adjacent spinous processes and are continuous with the interspinous ligament along its posterior margin. These posterior ligaments provide the most protection against flexion injury and outside of the capsular ligaments do not appear to be well innervated [13,37].

Cervical spine ligaments are predominantly made of collagen with fibrocartilage at the bony attachments. Thus, ligaments can be irreversibly stretched or ruptured during injury such as whiplash. The structure and orientation of these ligaments determine their ultimate strength [38–40]. However, significant decreases in the strength of all cervical ligaments have been documented after whiplash injury [41]. Disruption of these anterior and posterior ligaments after trauma has also been established [21,42–44]. The ALL is more commonly torn in lower speed injury [44,45]. However, even with such severe injury as a bilateral facet dislocation, the PLL can remain intact [46]. Injection into the interspinous ligament in asymptomatic subjects created pain in the neck and referral patterns into the scapula suggesting the interspinous ligament as a potential source of neck pain [47].

Despite the evidence for the structure and potential for injury of the ligaments of the cervical spine, the evaluation remains challenging. Magnetic resonance imaging (MRI) does not adequately demonstrate the cervical ligaments in asymptomatic individuals and thus cannot be a reliable evaluation of ligament injury in symptomatic individuals [48]. Clinical evaluation including range of motion and manual palpation cannot specify the structure causing the pain [30,49]. Currently, there is not a reliable objective diagnostic study to determine whether the ligamentous complex of the cervical spine is in facet a cause of neck pain.

Dura Mater

The dura mater is innervated by both myelinated and nonmyelinated branches of the sinuvertebral nerve as it courses posterior [35,36]. Dural irritation, or meningitis, can be caused by infection or bleeding into the area. Brudzinski's sign can help identify meningeal irritation but not localize the site or mechanism of irritation. The role of disc pathology and dural irritation is described in the lumbar spine. The dura has not been fully evaluated as a possible cause of pain in the cervical spine.

Bone

With major trauma or in the presence of osteoporosis and just minor trauma, acute fracture in the cervical spine can

be a source of neck pain. However, despite the numbers of patients presenting to the emergency department after blunt trauma, neurologically intact patients have an incidence of acute fracture of only 1% to 2% [50,51].

Vertebral bodies are innervated by branches of the sinuvertebral nerve, which course though the periosteum [35,36]. In addition, the basivertebral nerve innervates the trabecular bone of the vertebral foramen along with a corresponding venous plexus. The presence of substance P has been demonstrated within these basivertebral nerves, which indicates a possible source of nociception; however, the exact function of this nerve has not been demonstrated [52].

In the absence of acute fracture, bone in the cervical spine has not been determined to be a source of neck pain.

Zygapophyseal Joint

As part of the three-joint complex of the cervical spine, zygapophyseal joints (z-joints), are composed of the superior articular process of the joint below and the inferior articular process of the joint above encased by a synovial joint capsule. These joints are shaped to best bear weight and prevent anterior translation. Side bending and axial rotation are not part of z-joint function as movement is allowed only in the plane of the joint itself, which is superior and posterior [53]. The z-joints are innervated by the medial branch of the cervical dorsal rami from the adjacent levels above and below [20].

Cadaveric studies have demonstrated capsular lesions, hemorrhaging [21], and spondylosis [54] in the facet joint over the course of natural aging and with associated trauma [55]. Biomechanical studies demonstrate mechanisms of flexion and extension at physical forces significant to compress facet joints and stretch the capsular ligament [21,56–58].

Pain referral patterns have been established for individual lower cervical facet joints based on stimulation studies in asymptomatic volunteers; these patterns were diagnostically correlated with symptomatic patients using the double block paradigm in which comparative blocks of lignocaine and bupivacaine are used on separate occasions to block the z-joint or medial branches supplying the joint [13,37,59–62]. Following the same technique the upper cervical, the atlanto-occipital, and the atlantoaxial joints have also been determined to demonstrate specific patterns associated with pain in the head and neck [63]. The aforementioned studies demonstrate that the cervical z-joint can serve as a source of neck pain [64].

Techniques to establish z-joint-related pain are not related to tenderness of structures [65] or range of motion [66,67]. However, an experienced manual practitioner noting abnormal end feel, resistance to motion, reproduction of pain with passive accessory movement of the z-joint was found to be accurate when all three criteria were met and compared to a single diagnostic intra-articular or medial branch block [68]. In the patient nonresponsive to typical rehabilitation strategies, further confirmatory diagnostic testing can be directed at the presumably involved joint based on the patient's history of pain referral pattern and physical examination using manual techniques [68,69]. Imaging studies have not been reliable in identifying the involved cervical z-joint as pathologic findings have been found in up to 34% of asymptomatic subjects with MRI [70].

The confirmatory test to diagnose pain emanating from the cervical z-joint is a diagnostic intra-articular or medial branch block. The comparative block paradigm is used to avoid a false-positive seen with 27% of single diagnostic blocks; 10 studies using the comparative block paradigm have established the prevalence of cervical z-joint pain as 49% to 55% of those with neck pain [59,60,71]. Of the positive responses, the most common levels causing upper neck pain and possible associated headaches were C2-3 (45%), and lower neck pain and possibly shoulder or scapular pain were C5-6 (41%) [59]. The majority of subjects have pain emanating from just a single joint and not multiple joints [72].

Intervertebral Disc

Cervical discs, unlike their lumbar counter parts, are heavily endowed in their anterior portion with a thick annulus that anchors the vertebral body above to the one below, serving as an interosseus ligament [53,73]. Laterally, this structure thins out and may form a transverse fissure as it courses posteriorly [74]. The sinuvertebral nerve stems from the ventral ramus of the vertebral nerve in the lower cervical segments and ascends through the vertebral foramen posteriorly around the disc so that its major branches innervate the annulus of the disc and the adjacent disc below [35,36]. Histologically, nerve endings and nerve fibers have been observed in the intervertebral disc posteriorly and laterally. These findings correspond with an ability of the intervertebral disc to be a source of pain [36,53,75].

Cadaveric studies have demonstrated lesions of the intervertebral discs after trauma including bleeding into the disc, avulsion of the annulus fibrosus, and fissuring of the disc [21]. Simulated biomechanical studies demonstrate forces significant to cause such disruption [76,77]. Injection of the cervical disc during discography has demonstrated that the disc can cause pain [78,79]. Referral patterns of the disc have been studied and mapped [79]. These studies indicate that the disc can serve as a source of neck pain.

History and physical examination have not been diagnostic of axial neck pain of discogenic origin. Unfortunately, MRI has shown abnormal disc pathology in asymptomatic individuals [70,80,81]. MRI studies have demonstrated false-positive rates of approximately 50%, where abnormal appearing discs on MRI prove not to be the source of an affected individual's neck pain. Conversely, more than 27% of morphologically normal appearing discs on MRI may demonstrate internal derangement and concordant pain [82–84]. In light of the poor specificity of MRI, further evaluation of axial neck pain was established with discography.

Provocation discography was initiated to further evaluate the cervical disc morphology and the cause of pain [85,86]. Using radiopaque contrast to stimulate a disc enables visualization of annular fissures, which produce

the patient's usual neck pain upon stimulation [85,87]. The goal of discography is to identify the pain generator preoperatively for fusion surgery with studies reporting 70% to 91% success rates indicating diagnostic accuracy in appropriate patients [88–93]. It is also, however, the pain provocation which can limit the accuracy of discography, because normal discs have been demonstrated to provoke concordant pain in some patients [78]. Strict operational criteria must be adhered to in order to optimize the diagnostic accuracy of cervical discography (Chapter 30). Of 173 patients who underwent cervical discography to evaluate cervical degenerative disc as a source of chronic neck pain, only 17 (10%) were determined to be actual surgical candidates based on their responses, suggesting that cervical discography is not only helpful in diagnosing discogenic neck pain but also in eliminating unneeded surgery [94].

With concerns over the accuracy of provocation discography, analgesic discography was developed based on the alleviation of the patient's usual pain with intradiscal anesthetic at the completion of the stimulation [95]. The cause of any internal derangement, whether traumatic or degenerative, cannot be appreciated through these procedures [89].

There has been significant controversy over the accuracy of discography, imaging, and clinical evaluation in the identification of discogenic neck pain. Concerns about discography revolve around false-positive results that could be ameliorated by establishing a threshold for concordant pain as well as universal morphological classifications [96,97]. Further refinement is necessary, but discography remains the standard to evaluate the intervertebral disc as the cause of a patient's neck pain.

Patients undergoing discography for axial neck pain caused by trauma were also tested via diagnostic z-joint injections. Following the double block paradigm, it was established that both discs and joints can cause neck pain in 41% of those studied. Of the remaining patients, 20% were determined to have discogenic pain while 23% were determined to have only z-joint-related pain [14].

The aforementioned data are summarized in Table 1.1.

LUMBOSACRAL SPINE

Muscle

The muscles of the lumbar spine consist of the erector spinae, multifidi, quadrates lumborum, interspinalis, intertransversarii, and psoas. The interspinales spans one segment adjacent to the interspinous ligament. The intertransversarii span the transverse processes [98]. The main muscles posteriorly are the erector spinae, multifidi, and quadrates lumborum, which are divided into compartments by the thoracolumbar fascia. The middle layer intervenes between the quadrates lumborum and erector spinae. The anterior layer covers the quadrates lumborum. The posterior layer encloses the erector spinae and multifidus.

The medial and lateral branches of the dorsal ramus supply the thoracolumbar fascsia [99,100]. The thoracolumbar fascial has free nerve ending consistent with nociceptive fibers [100].

The multifidus and intertransversarii are innervated by the medial branch of the dorsal ramus. The iliocostalis is innervated by the lateral branch of the dorsal ramus. The longissimus muscle is innervated from the intermediate branch of the dorsal ramus [99,101]. The quadrates lumborum is innervated by subcostal nerve and lumbar plexus [102].

Various studies have been performed to determine whether the fascia or muscle can be a source of LBP. Lumbar spine surgery performed with local anesthesia noted that the fascia may be touched or cut without pain [103]. Forceful stretching of paraspinal muscle particularly at the site of blood vessels and nerves produced localized LBP, which varied with the degree of stretch. The authors postulated that the pain could be due to stretching of neurovascular bundles as opposed to muscle [103]. However, injection of hypertonic saline into fascia and lumbar muscles resulted in pain [29,47]. Muscle injection often resulted in referred pain following a segmental pattern [29].

Tender muscular nodules have been noted as a secondary source of pain in patients with radicular pain from

Table 1.1 Cervical Spine Summary of Four Criteria and Prevalence

Structure	Nociception	Nerve	Clinical	Disease/Injury	Diagnostic Test	Prevalence
Muscle		Yes	Yes	Yes		
ALL				Yes		
PLL		Yes		Yes		
Ligamentum flavum				Yes		
Supraspinous ligament						
Interspinous ligament		Yes		Yes		
Capsular ligament		Yes		Yes		
Dura		Yes		Yes		
Bone	Yes	Yes	Yes	Yes	Imaging	1%–2% acute fracture
Z-joint		Yes	Yes	Yes	Diagnostic injection	23%–55%
Disc	Yes	Yes	Yes	Yes	Discography	20%
Disc and z-joint	Yes	Yes	Yes	Yes	See above	41%

Blanks in table represent lack of data. The nociception column represents studies evaluating presence of either nociceptive fibers or neuropeptides. The nerve column represents innervation of the structure is present. For bone, the data is for acute fracture. For bone there is no clinical, disease/injury, or diagnostic data in the absence of fracture.

herniated lumbar disc. Temporary relief was noted with injection of local anesthetic into the tender points [104]. These tender muscle nodules have been postulated as a primary source of LBP. Travell and Rinzler postulated that myofascial trigger points are hypersensitive points that result in referred pain through central pain mechanisms. Trigger points may be secondary to direct trauma to joints, muscles, chronic muscle strains, arthrtitis, nerve injury, dyskinesia, and hysteria [105]. They based their hypothesis upon clinical experience with 1000 patients. These patients had relief with trigger point procaine injection, dry needling, or use of ethyl chloride spray [105]. Simon postulated that the mechanism of trigger point pain is from trauma to the sacroplasmic reticulum. The injury results in release of calcium along with adenosine triphosphate (ATP) producing a continuous contraction. Subsequently, the prolonged contracture releases metabolites that sensitize sensory neurons. With continued contraction, ATP depletion occurs creating an energy deficit contracture. Stretching the locked myofilaments terminates the contracture [106]. Despite these eloquent hypotheses, there is a lack of histologic or biochemical studies to support the trigger point as a pathologic entity [107], and trigger points have not been systematically studied with reported prevalence estimates.

Muscle spasm has been considered a source of LBP. Whether muscle spasm results in a cycle of pain-spasm-pain is unknown [108]. Muscle cramps in the extremities are known to be painful. But whether muscle spasm in the lumbar region is a primary cause of LBP is not known [108,109]. Lumbar muscle spasms may be due to secondary causes. Increased muscle activity on electromyogram of the iliopsoas and paraspinal muscles was demonstrated with irritation of nerve root and dura with injection of hypertonic saline in subjects 10 days after surgical discectomy for disc herniation induced sciatica [110]. This study suggests muscle spasms may be secondary to an underlying pathology such as inflammation of a nerve root or herniated nucleus pulposus. However, little evidence exists supporting a role for muscle spasms as a stand-alone source of chronic LBP.

Compartment syndrome has been postulated as a cause of LBP (Chapter 18). A case report of severe LBP after exertion was attributed to the development of compartment syndrome. The patient did have elevated CPK and myoglobinuria corroborating rhabdomylosis. Computed tomography (CT) scan demonstrated enlarged paraspinal muscles with low-density lesion suggestive of necrosis. Unfortunately, compartment pressure measurement was not performed [111]. The paraspinal musculature is at risk of compartment syndrome as the paraspinal muscles are divided into fascial compartment [111,112]. A case report of compartment syndrome of the erector spinae in a patient with LBP induced only with exercise was diagnosed based on elevated compartment pressures. Symptoms were alleviated with rest. The patient had relief of symptoms with fasciotomy [113]. Compartment syndrome was considered rare because only one subject in 4 years was found by the investigator [113]. The prevalence of lumbar compartment syndrome is unknown.

Lumbar strain is a commonly wielded diagnosis for mechanical LBP but is without anatomical or histologic evidence [12]. Much of the knowledge of lumbar strain is extrapolated from peripheral muscle strains. Strains typically occur at the myotendinous junction because of eccentric loading [109]. Despite the widespread diagnosis of lumbar strain, objective diagnostic studies confirming the diagnosis have not been reported in the English literature.

Serologic evaluation of CPK levels along with MRI of lumbar musculature to diagnose acute lumbar strain has been proposed [114]. Muscle may be a source of acute LBP but if so, without known prevalence and which age group is typically affected. Currently, evidence does not support muscle as a cause of chronic LBP.

Ligament

The ligaments of the spine can be classified into four groups [115]. The first group consists of the anterior and posterior longitudinal ligaments. These ligaments connect the vertebral bodies. Both ligaments are innervated through an extensive neural plexus [116]. The ALL is innervated by a plexus consisting of fibers from the sympathetic trunk, rami communicantes, and perivascular nerve plexus [116]. The presence of small fibers and contribution from the sympathetic trunk suggest the ALL could be a source of pain. However, no diagnostic tests have been developed to identify the ALL as a source of LBP. Whether the ALL can result in pain is unknown at this time.

The PLL is innervated by the sinuvertebral nerve, which forms an extensive neural plexus [116–118]. Nociceptive fibers have been isolated in the PLL [119]. Nylon suture attached to the PLL at the time of spine surgery when pulled in the postoperative period produced LBP [120] as did mechanical stimulation [103]. The PLL is intimately connected to the outer annulus and could not be separated to determine which is a source of pain [103]. The annulus is a well-studied source of LBP. Current diagnostic studies are unable to separate pain from the posterior outer annulus versus the PLL. Although some may postulate that discography may be able to differentiate between the two, the cause of pain from discography has not been completely elucidated and is complex—neural pathways, dorsal root ganglion, end plate deflection, and chemical sensitivity [121–124].

The second group consists of the ligamentum flavum, supraspinous, and interspinous ligaments. These ligaments connect the posterior elements of the spine. The ligamentum flavum is poorly innervated with unmyelinated free fibers on the outer most layer of the dorsal aspect but not in deeper layers [125]. In vivo studies with mechanical stimulation of the ligamentum flavum did not produce pain [103,120]. The ligamentum flavum is unlikely to cause LBP.

The supraspinous ligament terminates in 95% of individuals before the L4–5 level. Its absence in the lower lumbar region makes the ligament an unlikely source of LBP [126,127]. The interspinous ligament is innervated by the medial branch of the posterior ramus [117]. The interspinous ligament at L5-S1 is supplied by the L4 medial branch. Dissections have revealed small myelinated nerves, which can transmit pain [117]. Stimulation of the interspinous ligament can cause LBP [47,128]. However,

injection of hypertonic saline into the interspinous ligament produced mixed results. One study did not [104] and one did produce pain [128]. In another study, nylon suture attached at the time of surgery found that the interspinous ligament produced no pain when the suture was pulled [120]. Forcible stretching of the interspinous ligament occasionally produced central LBP in surgical subjects [103,127]. Anesthetizing the interspinous ligament can relieve pain and can be utilized as a diagnostic procedure [129,130]. The validity of this test has not been well studied. Studies of the interspinous ligament have produced mixed results of whether the ligament can cause LBP. Injection into the ligament may be a potential diagnostic test (Chapter 16) but requires further testing.

The third group consists of the iliolumbar ligament. The ligament attaches the ilium to the lumbar spine with attachments to the lumbar 5 transverse process. The iliolumbar ligament receives innervation from the dorsal and ventral rami at L4 and 5 [19]. The ligament does not develop until the second and third decade of life. Although the iliolumbar ligament is often cited as a cause of LBP, there are currently no objective findings to confirm this assumption.

The fourth group are false ligaments. These consist of fascial planes overlying the foramen and transverse processes. The mamillo-accessory ligament is not a true ligament because its connections are on the same and not different bones. The fascial planes and mamillo-accessory ligament are unlikely sources of pain [115].

Dura Mater

Although innervation is sparse posterolaterally, the ventral dura is richly innervated by a plexus from the sinuvertebral nerve [116,117,131]. Blood and infection can irritate the dura resulting in pain. With bacterial meningitis, stiffness and pain occurs. Kernig and Burdzinki's signs are positive with stretching of the inflamed meninges.

The dura also may become inflamed with a herniated disc with or without nerve root irritation [132]. However, currently it is not plausible to selectively anesthetize the dura to differentiate pain emanating from the nerve root, annulus, or dura mater. An in vivo study suggests that the dura does not cause pain. Five subjects undergoing surgery for a herniated nucleus pulposus and sciatica had nylon suture anchored to the dura mater. Postoperatively, pulling on the thread did not cause any pain suggesting that the dura typically does not cause mechanical LBP [120]. At the time of surgery, stimulation of the posterior dura did not cause LBP [103]. However, the innervations of the posterior dura is sparse compared with the anterior dura and could explain the lack of pain observed in the two surgical studies [131]. Summers et al. postulated that reproduction of LBP in subjects with a central focal protrusion during the straight leg raise (SLR) physical examination manuever was due to tension on an irritated anterior thecal sac [132]. However, SLR with LBP still does not differentiate whether the pain is from the annulus or thecal sac. Furthermore, those suffering from LBP in which the dura is presumed to be a source of pain have demonstrated a concomitant disc protrusion. Hence one may hypothesize that the disc, and not the dura, is the primary source of pathology. Although dural irritation may explain why some individuals with discogenic LBP have relief with epidural steroid injection, primary treatment for long-term success would need to be directed at the intervertebral disc. For the patient suffering from nonspecific mechanical LBP, these symptoms cannot be attributed to the dura mater as the primary source of LBP.

Vertebral Body

The vertebral body is covered by periosteum innervated anteriorly by a nerve plexus consisting of fibers from the sympathetic trunk, rami communicantes, and perivascular nerve plexus [116]. The sinuvertebral nerve creates a nerve plexus covering the posterior vertebral body [116]. From both of these plexuses nerve fibers penetrate the vertebral body wall [116].

The basivertebral nerve innervates the vertebral body trabecular bone. Besides vasomotor involvement, substance P has been isolated suggesting the presence of some nociception. However, the function of the basivertebral nerve is not known [52]. Nerve fibers are seen throughout the trabecular bone of the vertebral body that originates from the basivertebral nerve and from nerves penetrating the anterior vertebral body [133].

The vertebral body endplate has both sympathetic and sensory nerve fibers. These fibers are proposed to be involved in neovascularization of the intervertebral disc resulting in disc degeneration. Further, supporting the endplates as potential source of pain is the presence of substance P, calcitonin gene-related peptide (CGRP) sensory nerve fibers [134]. Deep LBP has been created with pressure or curetting the vertebral end plate and with endplate deflection during discography [103,121]. The innervation of the vertebral endplate could explain LBP with acute disc herniation through the endplate [135].

Vertebral body compression fractures may or may not be painful. Diagnosis of a symptomatic fracture is based on history, physical examination, and imaging as discussed in Chapters 45 to 47. The patient should complain of pain overlying the fracture. Physical examination frequently demonstrates pain with percussion over the fracture site. Plain films will demonstrate the fracture but typically will not indicate acuity. MRI demonstrating bone marrow edema is consistent with an acute or subacute fracture that may be painful. The presence of a vertebral compression fracture with MRI evidence of bone marrow edema but no pain indicates an asymptomatic fracture. The presence of a compression fracture but normal marrow signal on MRI would suggest an old fracture. In this scenario, back pain could not be directly attributed to bone. An old fracture may indirectly result in pain from spinal deformity. Compression fractures may be traumatic or pathologic. In the absence of fracture, bone is usually not considered in the diagnosis of chronic mechanical LBP.

Baastrup's Disease

Baastrup's disease is also known as kissing spine disease. There is spinous process abutment with the development

of a neoarthrosis. The neoarthrosis does have sensory fibers that may be capable of transmitting pain as discussed further in Chapter 16. Cadaveric studies have demonstrated a pseudojoint with chondroid metaplasia, enchondral ossification, and osteoarthritic changes [136]. Bursa tissue develops and can exhibit inflammatory changes [137]. Surgical resection of a symptomatic level can demonstrate an interspinous bursitis, interspinous cyst, and a neoarthrosis [138].

MRI has demonstrated findings consistent with inflammation and edema within the interspinous bursa, interspinous ligament, and spinous processes [130]. The prevalence of interspinous bursitis on MRI in 539 subjects with LBP is 8.2%. Multiple level involvement was present in 47.7%. Of this group, 71.4% were with two-level and 28.6% with three-level involvement [139]. The authors suggested the term Baastrup's sign for the radiologic findings because imaging findings do not necessarily correlate with pain [139].

Injection of local anesthetic under fluoroscopic guidance has been proposed as a diagnostic test to determine whether the presence of Baastrup's sign is symptomatic [140]. Relief of pain would suggest the presence of symptomatic Baastrup's disease. In subjects with relief from a diagnostic injection, successful treatment with fluoroscopically guided corticosteroid injection into the interspinous ligament or surgical treatment has been reported [130,140]. Individuals are selected to undergo a diagnostic injection based on the presence of midline LBP, tenderness to palpation, with positive radiographic studies at the involved level [130,140]. The diagnostic injection is performed with local anesthetic injected under fluoroscopic guidance into the involved interspinous ligament. A positive test would be 80% relief of pain. 18F-fluorodeoxyglucose PET or CT has been proposed to diagnose Baastrup's disease, but further study is needed [141]. Until then, fluoroscopically guided diagnostic injection is used to confirm Baastrup's disease.

Baastrup's disease determined by radiographic studies and confirmed with diagnostic injection between the spinous processes had a prevalence of 1.8% with a mean age of 75 years in chronic LBP subjects presenting to an academic spine center [142].

Bertolotti's Syndrome

Bertolotti's syndrome is LBP attributed to a painful pseudoarthrosis created by a broad transverse process of a transitional segment abutting either the iliac crest or sacral ala. How the pseudoarthrosis results in pain is unknown. The abutment of the periosteum of the transverse process and sacral ala or ileum is a potential cause as the periosteum is innervated by nociceptive fibers.

Degenerative changes at the pseudoarthrosis have been noted on radiographs but not necessarily correlated with pain [143]. Nuclear single-photon emission computed tomography (SPECT) scanning has been proposed in the evaluation of Bertolotti's syndrome [144,145].

LBP subjects who had increased uptake seen on SPECT imaging at the pseudoarthrosis did not undergo diagnostic injection to determine whether pain was related to the increased uptake [144]. Nuclear bone scan has been found to be normal in subjects with symptomatic Bertolotti's syndrome diagnosed with relief from fluoroscopically guided injection of lidocaine into the pseudoarthrosis. The nine subjects with a positive diagnostic injection underwent resection of the broad transverse process and had complete relief in seven and only minor pain in two subjects. One subject had no relief from the diagnostic injection and subsequently had no relief with surgery. The authors recommended a positive injection before consideration of surgery [146]. Because of the poor surgical results in two out of six subjects after relief from a diagnostic injection, instillation of local anesthetic as a diagnostic test has been questioned. Another study noted favorable results from surgical resection of the transverse process in those with positive diagnostic injection with lidocaine [147]. Imaging studies are not helpful in confirming the diagnosis of Bertolotti's syndrome as the source of LBP. Diagnostic injection is the current standard for diagnosis. Improved results may be seen by using a small volume of local anesthetic, at least 80% decrement in pain, and following the double block paradigm to minimize false-positive responses.

The incidence of transitional vertebra on radiographic studies in 2000 LBP sufferers was 7% [143]. MRI in 769 subjects with LBP found the overall incidence of Bertolotti's syndrome on imaging studies of 4.6%. In this group, Bertolotti's syndrome was more common in those below 30 years with an incidence of 11.4% [148]. Unfortunately, these studies did not use diagnostic injections to determine whether the pseudoarthrosis was symptomatic. The current prevalence of Bertolotti's syndrome as a cause of LBP is unknown.

Spondylolysis

Acute pars stress fractures typically present in adolescent athletes. The athlete typically has focal pain just off of midline in the lumbar region. The pain is usually aggravated by lumbar extension. Physical examination reveals pain on extension with positive stork test. MRI is now the imaging of choice to evaluate for an acute fracture [149,150]. The acute fracture will demonstrate decreased signal on T1, increased signal on T2, and fat suppression STIR images. Fine cut CT with reverse gantry is used to confirm the presence and characteristics of the fracture, which will affect treatment. As mentioned earlier, acute fractures can be a source of LBP. The diagnosis is confirmed by history, physical examination, and bone marrow edema on MRI.

The absence of bone marrow edema on MRI or increased uptake on bone SPECT imaging suggests an old fracture with nonunion. The question arises in these cases of whether the spondylolysis is an incidental finding or the cause of LBP. Histologic evaluation of surgical specimens from symptomatic subjects found small myelinated and unmyelinated axons within the connective tissue of the fracture. Free nerve endings were additionally seen. These findings were consistent with nociceptive innervation of the pars fracture [151]. Another study had contrary results finding lack of innervation of the spondylolytic defect [152]. The studies did prepare specimens differently and may be a variable in the different histologic results.

Immunohistochemical study has found protein gene product, CGRP, substance P, vasoactive intestinal peptide (VIP), and C-flanking peptide of neuropeptide Y in the ligamentous structure and surrounding soft tissue of the pars defect [153]. Overall, the studies support the ligamentous tissue within the pars defect as a potential source of LBP.

The incidence of spondylolysis found on 2000 CT scans in a general Japanese population was 5.9%. Spondylolysis was more prevalent in those with than without spina bifida occulta at 16.2% and 5.0%, respectively [154]. The incidence in an American population of 510 CT scans done for reasons other than LBP was 5.7% [155]. The development of LBP in 6-year-olds with spondylolysis followed for 45 years had no difference compared to the general population [156]. The majority of spondylytic defects occurred at L5. The prospective study included follow-up radiographs and at final evaluation MRI. Clinical follow-up also included questions on pain, work status, medication, and disability with the addition of the SF-36 at final evaluation. Those with a unilateral defect had no spondylolisthesis or disability. Only 5% (1/22) with bilateral spondylolysis had symptomatic progressive spondylolisthesis. Overall there was no increase in disability and LBP compared to the general population [156]. Screening radiographs in 3988 Israeli policemen found 196 with a spondylolytic defect. The officers with spondylolysis had a similar incidence for absenteeism for LBP with the other officers. However, the duration of absence was 2.7 times longer for those with spondylolysis [157]. Because spondylolytic defects can be seen on imaging studies in asymptomatic individuals, a confirmatory diagnostic test is required. The diagnostic test to determine symptomatic pars defect is a diagnostic pars injection with local anesthetic.

Diagnostic injection of lidocaine under fluoroscopic guidance to relieve pain is recommended before consideration of surgical stabilization of the pars fracture [158,159]. Selection of surgical patients on the basis of an 80% decrement in LBP after diagnostic lidocaine pars injection improves surgical outcomes [159].

The pars defect is a potential source of LBP. The prevalence of symptomatic pars defects has yet to be established.

Zygapophyseal Joint

The lumbar z-joint receives innervation from the medial branch of the posterior rami. Each joint receives a medial branch from two levels [99,101,160]. For example, the L4-5 z-joint receives innervation from the L3 and L4 medial branches. In fetal dissections the medial branch supplies the z-joint capsule and periosteum [117]. In surgical specimens, PGP 9.5 immunoreactive nerve fibers, CGRP immunoreactive nerves, substance P, and VIP were found in facet joint capsules [161–163]. An in vitro biomechanical study on rabbits found high threshold mechanoreceptors activated at loads that could be damaging to the joint and were postulated to be involved in nociception [164]. These studies suggest that the lumbar z-joints can serve as a potential source of LBP.

Stimulation of z-joint capsule with needle or Cobb elevator in surgical subjects produced localized LBP and rarely buttock pain. This pain was relieved with injection of local anesthetic [103]. Injection of hypertonic saline into the lumbar z-joints of normal volunteers produced LBP [125,165,166]. Furthermore, subjects with LBP also had pain relief after injection of xylocaine into the joints [165].

Postmortem studies of subjects who died from trauma have demonstrated subchondral fractures, capsular tears, avulsion, and joint space hemorrhage in the z-joints suggesting the joints are injured sufficiently to become painful [167]. However, findings of facet joint degeneration can be seen in asymptomatic subjects or in those with negative diagnostic facet joint injections [168–170]. Hence, a confirmatory diagnostic test is needed to determine whether the z-joints are causing pain in patients suffering from LBP. With fluoroscopic guidance, the z-joint can be selectively anesthetized to identify whether the joint is a source of LBP [171,172].

Schwarzer et al. refined diagnostic z-joint injections to control for a placebo effect [18,173]. Using comparative anesthetic blocks or placebo injection with saline, a false-positive rate with single diagnostic injections of 32% to 38% was determined [18,173]. Hence, comparative or placebo-controlled injections are required to minimize a placebo effect and maximize specificity to diagnose LBP emanating from the z-joints.

Using comparative blocks, the prevalence rate in chronic LBP patients presenting to spine clinics has been estimated. Depending on patient age, z-joint pain prevalence ranges from 12% to 45% [173–178]. Controlling for age, the prevalence was 18% in the 31-to-40 age group and 44% for the 51-to-60 age group [179]. Z-joint pain is more common in older patients with 1.16 increased risk for each 5 years of age [178]. In fact, the most common source of LBP in adults above age 55 years is a z-joint [178] and the most effected level is L5-S1, followed closely by L4-L5 [173,178,180,181]. In those suffering from failed back surgery syndrome, the z-joint was the cause of pain in 2.7% [182]. For those with LBP after lumbar fusion surgery, 12.5% of subjects had pain emanating from the lumbar z-joint with 80% at the level adjacent to the fusion [142].

Sacroiliac Joint

The SIJ is an auricular-shaped diarthrodial joint with a joint capsule, synovial fluid, hyaline cartilage on the sacral side, and fibrocartilage on the iliac side. The SIJ innervation is not completely elucidated. The joint is innervated by the posterior rami of the lumbosacral roots [101,129]. The anterior aspect of the SIJ receives variable innervation from L3-S2 and the superior gluteal nerve [183]. The posterior aspect of the joint receives variable innervations from S1-2 and L4-S4 [101,183,184]. A study in rats has demonstrated sensory fibers from the L1 and L2 dorsal root ganglion passing through the sympathetic nervous system [185]. Histologic evaluation of the SIJ and posterior ligament found myelinated and unmyelinated nerve fibers [184,186]. Immunohistochemical studies found substance P and CGRP in cadaveric SIJ anterior capsular ligament and interosseous ligament [187]. Nociceptive fibers are postulated to be present in the adult SIJ [186].

Injection of contrast into the SIJ created pain in normal volunteers [188]. Seronegative spondyloarthropathy resulting in pain and relief with fluoroscopically guided corticosteroid SIJ injection has been reported [39,189]. SIJ infections also result in back pain [190]. A variety of degenerative changes occur in the SIJ [191–196]. By the third decade, degenerative changes may be seen [191,195]. By the fifth decade, 91% of males and 77% of females have degenerative changes [195]. By the sixth decade, all cadaveric specimens had degenerative changes and joint ankylosis occurred in 82% of males and 30% of females [195]. These studies suggest the SIJ is a potential source of LBP.

History and physical examination has not been accurate in diagnosing pain emanating from the SI joint [17,197–200]. Radiographic imaging is helpful in diagnosing SIJ pain from trauma, inflammatory, infectious, and metabolic conditions [201]. Radiographic plain films have demonstrated degenerative changes in asymptomatic individuals above the age of 50 [202,203]. Furthermore, the previously mentioned cadaveric studies have demonstrated degenerative changes commonly present by the third decade. Advanced imaging techniques have not been helpful in diagnosing SIJ syndrome.

Anesthetizing the SIJ with a small aliquot of local anesthetic injected under fluoroscopic guidance (Chapter 13) has been demonstrated to mitigate pain by more the 50% suggesting the joint as a cause of LBP [204]. Although pain provocation has been helpful in demonstrating that the SIJ can cause pain, provocation is not diagnostic [205]. Diagnosis is based on at least 75% relief with the injection of a local anesthetic [17,173,206]. False-positive injections due to a placebo effect is controlled by injecting different agents on separate days with the patient blinded to the agent injected [17]. To further decrease a false-positive test, injection should be done with a mixture of anesthetic and contrast under live fluoroscopy to avoid extravasation from the SIJ into adjacent tissues [207]. As history, physical examination, and imaging is not accurate in diagnosing LBP emanating from the SIJ, fluoroscopically guided diagnostic SIJ injections are the current standard for diagnosis 17,199,200,206,207].

Using a single diagnostic injection, a prevalence of 13% to 30% has been reported [205]. The higher 30% was based on 43 subjects suspected of having SIJ pain and the 13% based on the all 100 subjects who were part of the study [205]. However, the 13% may underestimate the prevalence because not all subjects were offered to undergo diagnostic SIJ injection. In another study utilizing single diagnostic injections, the prevalence of SIJ pain was 18% [178]. But the use of single diagnostic blocks may overestimate the prevalence by not accounting for the placebo effect. Using comparative blocks in 54 chronic LBP patients, 18.5% were found to have pain from the SIJ [17]. The false-positive rate from single diagnostic injection was 47% [17]. In 120 chronic LBP subjects randomized to participate in a study to evaluate the etiology of LBP, the SIJ was the etiology in only 2% of patients. The false-positive rate from single diagnostic injections in this study was 22% [177]. In a study by DePalma, SIJ pain occurred more frequently in older subjects and was typically unilateral [178]. In LBP sufferers after lumbar fusion surgery, 32.5% had pain emanating from the SIJ with increased risk when the fusion extended to the sacrum [142]. In LBP patients above age 55 years, SIJ was the second most common source after z-joints [178].

Intervertebral Disc

The sinuvertebral nerve from T12-L5 arises from the spinal nerve near the ramus communicans or with the ventral primary ramus within the intervertebral foramina and supplies the dura mater, posterior annulus, and PLL [117,118]. The sinuvertebral nerve in fetal dissections was found to have sympathetic and spinal components [117]. Nociceptive endings have been isolated in the intervertebral disc [208]. Surgical specimens demonstrated nonmyelinated fibers consistent with nociceptive fibers in the outer fibers of the annulus and PLL [119].

The nucleus pulposus has been demonstrated to be inflammogenic when exposed into the epidural space [209–213]. Inflammation has been demonstrated to be important in perpetuating pain by resulting in repetitive firing of nerve root in animal studies [214,215]. Inflammation of neural tissue results in impaired conduction [212]. Phospholipase A2, the rate limiting step in the liberation of arachidonic acid, has been found in human disc surgical specimens [216,217], and has been shown to be neurotoxic [218]. Leukotrienes and cytokinins are also involved in inflammation and pain mediation. Prostaglandin E2 found in surgical disc specimens sensitizes nociceptors to bradykinins [219]. Various cytokinins have been isolated from surgical disc specimens [220,221]. The cytokinins and matrix metalloproteinase are involved in disc degeneration. Nitric oxide in pathologic disc material promotes inflammation and is involved in immune regulation [221]. Immune responses to nucleus pulposus have been hypothesized to result in chronic inflammation [222]. Also, supporting an immune response promoting LBP is elevated IgM levels found in subjects with discogenic LBP [223].

Surgical disc specimens have been found to contain various neuropeptides involved in pain mediation—calcitonin gene-related peptide, substance P, VIP, and C-flanking peptide of neuropeptide Y [224,225]. Neuropeptides are involved in nociceptive activity. Discography has been found to elevate substance P and VIP of the dorsal root ganglion and modulate LBP [124]. In one study, an annular fissure was created by a stab wound and resulted in inflammation [226]. The inflammatory response along with release of cytokinins and neuropeptides could potentially explain discogenic pain from an annular fissure.

In vivo studies have been performed to determine if the intervertebral disc can cause LBP. Injection of hypertonic saline into the disc caused severe LPB [125]. Probing the disc at the time of lumbar spine surgery performed under local anesthesia in 100 subjects and another study of 193 subjects created LBP [103,104]. In another operative study, nylon suture was attached to different structures at the time of lumbar surgery [120]. The suture was passed through the skin to allow pulling on the attached structure up to postoperative day 14. The annulus was found to cause LBP [120].

Lindblom noted similar LBP created with diagnostic discography in subjects with a disc herniation [227].

Discography has been noted to create LBP in both symptomatic and asymptomatic subjects in the presence of an annular fissure seen on MRI [228]. Individuals suffering from infectious discitis develop severe LBP [229,230].

Studies have indicated that the intervertebral disc is the most common cause of adult LBP. Patients with discogenic LBP often complain of increased pain with trunk flexion, coughing, sitting, and lifting. They often feel better with trunk extension or lying flat with the knee bent or feet elevated. However, other subjects may atypically have more pain with trunk extension. Both history and physical examination will be discussed in more detail in Chapter 3 in evaluating pain emanating from disc versus other spinal structures.

MRI has been the imaging modality of choice to evaluate for disc pathology (see Chapter 4). Unfortunately, MRI has been unable to differentiate asymptomatic disc degeneration from symptomatic disc pathology. High-intensity zone (HIZ) lesions seen on MRI have been purported to correlate with LBP [231,232]. However, HIZ lesions have been noted in asymptomatic individuals [233,234]. Findings of disc bulging, protrusions, extrusions, and disc degeneration are also seen in asymptomatic population [70,170]. To determine whether an abnormal disc on MRI is the cause of LBP requires further diagnostic testing.

Discography has been utilized to determine whether a disc is the cause of LBP. The concordant reproduction of LBP at low pressure with disc injection with fissuring into the outer third of the annulus suggests the disc as the cause of LBP. The inter-rater reliability of discography ranges from 0.88 to 0.99 [235]. In this blinded, prospective study that included asymptomatic and symptomatic subjects, the false-positive rate was zero [235]. Colhoun et al. prospectively evaluated outcome with minimum of 2 years follow-up in 195 subjects undergoing fusion for LBP [236]. The success rate for subjects with positive discography was 89% compared to 52% for those with just abnormal disc morphology on imaging. Discography has been further refined with manometric discography [237]. Better surgical outcomes were found with concordant pain response at disc injection pressures < 15 psi above opening disc pressures [237]. However, discography is controversial [228,238–240] and further discussion is presented in the Chapter 5 on discography.

Noting the controversy surrounding provocation discography, discoblock has been proposed [241]. Discoblock is the fluoroscopic injection of 0.75 mL of 0.5% bupivacaine intradiscally. A positive discoblock is the relief of the patient's LBP. In a randomized, controlled study, discoblock was found to have statistically improved outcomes compared to discography for spinal fusion for LBP [241]. However, the sample size was small with only 15 subjects in each group. The study did not describe how to control for a placebo effect to minimize false-positive results. Further study is required regarding this diagnostic test. Discography has been studied extensively and is the current standard for diagnosing the intervertebral disc as the source of LBP.

Using discography, the prevalence of discogenic LBP in patients with chronic LBP presenting to three independent clinics was 26%, 39%, and 42% [175,177,178]. Discogenic LBP occurred more frequently in younger patients than older patients [122,178]. In those suffering from failed back surgery syndrome, the disc was the cause of pain in 31% [182]. After lumbar fusion, the intervertebral disc was responsible for LBP in 12.5% [142]. In 80% of subjects, the disc at the level of a posterior fusion or adjacent disc level was the cause of LBP [142]. The L4-5 and L5-S1 discs were most likely to result in exact pain on discography versus the L2-3 and L3-4 [122,175,178]. The intervertebral disc is typically the most common identifiable structure to cause chronic LBP [178,242] except for in one study facet joint pain was more common than discogenic LBP [177]. Typically only one disc is responsible for the patient's symptoms [243].

Table 1.2 Lumbar Spine Summary of Four Criteria and Prevalence

Structure	Nociceptive	Nerve	Clinical	Disease/Injury	Diagnostic Test	Prevalence
Muscle	Thoracolumbar fascia	Yes	Mixed	Yes	MRI?	
ALL		Yes				
PLL	Yes	Yes	Yes		No	
Ligamentum flavum	No	Poorly	No		No	
Supraspinous ligament			Ends L4–5			
Interspinous ligament	Yes	Yes	Mixed		Injection	
Iliolumbar ligament	Yes	Yes				
Dura		Yes	Posterior dura—No	yes	No	
Bone	Endplate-yes	Yes	Yes	Yes	Yes for fracture	
Baastrup's		Yes		Yes	MRI/injection	1.8%
Bertolotti's	Yes		Yes	Yes	Injection	
Pars Fracture	Yes	Yes		Yes	Injection	
Z-joint	Yes	Yes	Yes	Yes	Injection	12%–45%
Disc	Yes	yes	Yes	yes	Discography Discoblock?	26%–42%
Disc and z-joint	Yes	Yes	Yes	Yes	Yes	8%
SI joint	Yes	Yes	Yes	Yes	Injection	2%–32.5%

Blanks in table represent lack of data. The nociception column represents studies evaluating presence of either nociceptive fibers or neuropeptides. The nerve column represents innervation of the structure is present. For bone, the data is for acute fracture. For bone there is no clinical, disease/injury, or diagnostic data in the absence of fracture. MRI has been proposed as a potential diagnostic test for lumbar strain based upon use in limb muscles.

Only 8% of chronic LBP was found to be emanating from both the disc and z-joint [175,205].

The aforementioned studies are summarized in Table 1.2.

CONCLUSION

The differential diagnosis of axial neck pain and LBP is extensive. A careful history and examination is performed alerting the clinician to red flags that would suggest a neoplastic, infectious, rheumatologic, or medical cause of pain. Furthermore, shoulder and hip disorders can be difficult to separate from cervical and lumbar spine problems, respectively.

Axial neck pain is prevalent in today's society and carries a significant socioeconomic burden. It is often associated with nonorganic factors. Discogenic pain and pain emanating from the z-joints comprise the bulk of chronic neck pain. The symptoms of each of these etiologies of neck pain overlap significantly. Although the prevalence of facet pain is statistically greater than that of discogenic pain, the diagnosis is dependent on the appropriate use of available investigative techniques. Ultimately, 80% of patients with chronic neck pain can be given a specific anatomical diagnosis using appropriate evaluation techniques [244].

Various structures of the spine are potential sources of axial LBP. In the past, the majority of these structures could not be identified through history, physical examination, and imaging studies. Hence, patients were classified with idiopathic mechanical low back. Through the use of interventional spine procedures, many of these structures can now be identified. With the identification of the specific pain generator, more specific treatment may be rendered. In addition, more accurate epidemiologic studies may be performed for specific diagnoses to evaluate the natural history and comparative effectiveness of different treatments. Improved and more effective treatments may be developed. This may lead to improved outcomes with less disability and economic costs from persistent LBP.

By utilizing interventional diagnostic procedures, the structural etiology of chronic LBP can be identified in 68% to 90% of affected individuals [177,178]. The structural cause of chronic LBP could be determined in 64% of subjects [175,205]. In failed back surgery syndrome patients, the cause of pain was identified in 94.4% of 197 subjects [182].

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Part I
Lumbosacral Spine

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2 Pathophysiology of Painful Lumbosacral Spine Disorders

Lars G. Gilbertson and Manuel Fanor Saavedra

This chapter presents an overview of how painful lumbosacral spine conditions develop, recognizing the role of genetic, environmental, and biomechanical factors. Involvement of the intervertebral disc, facet joints, and sacroiliac joints is emphasized, laying the groundwork for subsequent chapters discussing diagnostic and therapeutic spinal algorithms.

CONCEPTUAL FRAMEWORKS FOR UNDERSTANDING SPINE FUNCTION

While there is no substitute for clinical experience and insight, along the way to acquiring such, residents, fellows, and post-training physicians routinely may benefit from the growing body of research that can help to systemize thinking and improve understanding of spine function in states of health and as altered by aging, disease, and clinical treatment. An influential conceptual framework advanced by Panjabi [1] represents the living spine as a complex neuromusculoskeletal system whose biomechanical functioning is rather finely dependent upon the interactions among and between three principal subsystems: the passive musculoskeletal subsystem (osteoligamentous spine plus passive mechanical contributions of the muscles), the active musculoskeletal subsystem (muscles and tendons), and the neural and feedback subsystem (neural control centers and feedback elements such as mechanoreceptors located in the soft tissues), as shown in Figure 2.1. The two-headed arrows between subsystems readily encourage "thought experiments" of how pathological changes in one subsystem might influence another—for example, prompting one to consider how painful arthritic changes in the facet joints might affect neuromuscular control of spinal movement.

As a neuromusculoskeletal system, the spine is relied upon to fulfill three principal (and usually simultaneous) biomechanical roles: load bearing, mobility, and protection of the spinal cord and nerve roots. The pathophysiology of the painful lumbosacral spine will be discussed in the context of these three roles.

Spinal Load Bearing

In vivo measurements of intradiscal pressure by Nachemson and colleagues [2] beginning in the 1960s revealed that axial compressive force on the

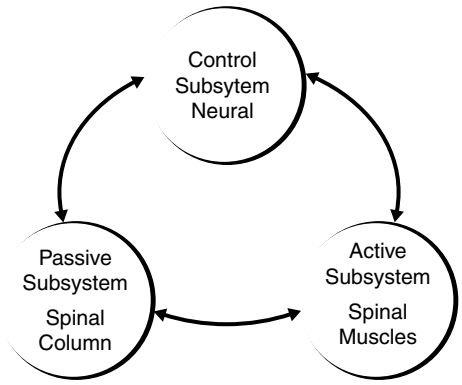

Figure 2.1 Conceptual framework of the living spine as a neuromusculoskeletal system whose biomechanical functioning is dependent upon the interactions among and between three subsystems. Adapted from ref. [8].

osteoligamentous lumbar spine can be several-fold greater than the combined weight of the body parts (head, upper limbs, trunk) and external objects being supported during various activities. This "force amplifier effect" is common to musculoskeletal joints, where large joint reaction forces can arise due to the poor mechanical advantage of the muscles as compared with that of the weights being supported. A simple biomechanical model based on an Archimedes lever system demonstrates how a relatively short lever arm requires the extensor muscles to generate high forces to equilibrate the flexion moment ("force times distance") produced by a hand-held weight and also the upper body weight—resulting in elevated reaction forces at the fulcrum (represented here by the disc), as shown in Figure 2.2. Such a model does not consider any inherent ability of the osteoligamentous spine to resist flexion/extension bending (greatest at extreme ranges of spinal motion), but otherwise can be useful for demonstrating ergonomic principles to patients (and for demonstrating biomechanical benefits of weight loss).

While the disc is important in transmitting axial compressive force from one vertebra to the next, experimental evidence indicates that the facet joints also are involved, and may support up to a third of total axial compression force depending on the spinal posture [3]. Under axial torsion (twisting about the longitudinal axis of the spine) the

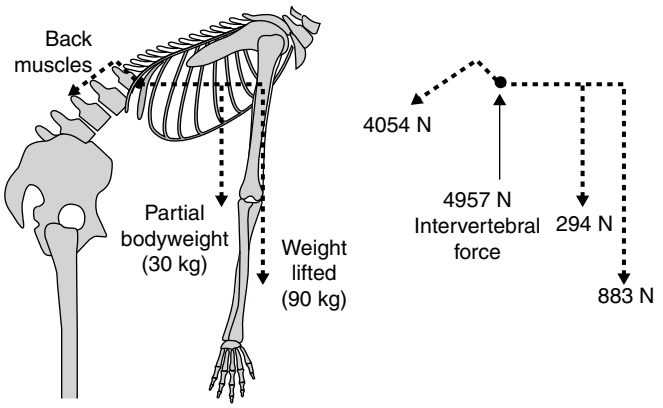

Figure 2.2 Simplified biomechanical model of the spine based on Archimedes lever system, illustrating high extensor muscle forces needed to equilibrate flexion moment produced by hand-held weight and upper body weight. Adapted from Scoliosis Research Society.

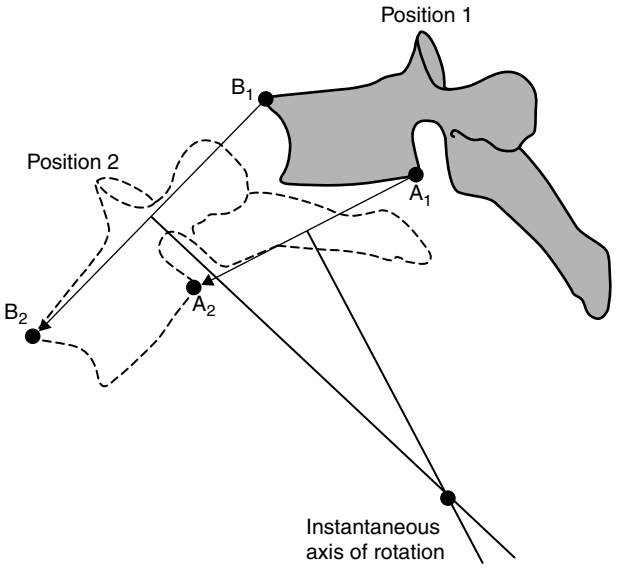

Figure 2.3 Spinal instantaneous axis of rotation (IAR). From ref. [6].

load sharing role of the facets (including capsular ligaments) can equal that of the disc [4]. Such evidence supports the concept of the spine as a "three joint complex" [5], and points to a complex mechanical interplay between disc and facets that is vulnerable to disruption under pathological conditions.

Spinal Mobility

Whereas spinal load bearing is described in terms of forces and moments, spinal mobility is described in terms of rotations and translations. Representative rotational ranges of motion for an individual lumbar vertebra-disc-vertebra unit are 15° for combined flexion/extension, 6° for lateral bending to one side, and 2° for axial rotation to one side [6]. Given that there are five such units within the lumbar spine (L1-2, L2-3, L3-4, L4-5, L5-S1), these add up to provide considerable overall range of motion for the lumbar spine, available for fulfillment of daily activities. Translations of a vertebra relative to another also occur—2 mm of anterior sagittal plane translation is considered normal in vivo, as observed in radiographical studies [7].

An important consideration of vertebral motion is the "virtual axis" about which motion occurs; for planar motions, this is termed the instantaneous axis of rotation (IAR) shown in Figure 2.3 (the three-dimensional analogue of the IAR is the helical axis of motion). Combinations of vertebral translations and rotation in a plane are efficiently described by defining the position of the IAR and reporting the amount of rotation occurring about that IAR. Changes in the location of the IAR can have dramatic effects on the functional roles of the muscles and osteoligamentous structures, in a similar manner as changing the location of the fulcrum alters mechanics of an Archimedes lever system—in other words, the IAR may be thought of as the "virtual fulcrum." The spinal ligaments develop tension as they are elongated—hence a spinal ligament resists any spinal motion (particularly sagittal plane bending) that would tend to increase the separation distance of the ligament's attachment points between adjacent vertebrae. The separation distance in turn depends upon the location of the ligament relative to the IAR and the amount of rotation occurring about the IAR. The tension-elongation relationship is such that ligaments initially offer little resistance to elongation, but develop greater tension as the ligament is stretched further—hence their stabilizing effect increases with increasing spinal motion.

Whereas the relatively short lever arm of the spinal extensor muscles is an apparent liability during load bearing (resulting in large forces experienced by the intervertebral disc and facet joints), an advantage is seen during mobilization—namely, that relatively small changes in the lengths of muscles so close to the axis of motion are able to produce large intervertebral rotations.

Neural Protection

Of the three principal biomechanical functions of the spine, protection of the spinal cord and nerve roots is foremost. For example, surgical decompression to alleviate pressure on the spinal cord or nerve root routinely is performed even at the expense of the removal of structures crucial to spinal load bearing. Once decompression is achieved, attention turns to restoration of load bearing—with restoration of mobility often a distant consideration (as is the case with surgical discectomy followed by fusion). Restoration of motion arguably might receive greater consideration for the cervical spine than the lumbar spine.

There are numerous potential sources of direct mechanical pressure upon the neural elements—such as a herniated disc, bone fracture fragments, and stenotic spinal canal. Often, a binary classification of "clinically

stable" or "clinically unstable" is made in the assessment of the threat posed to the neural elements under different clinical conditions—with the "unstable" determination often an indication for surgical intervention. The load-mobility characteristics of the osteoligamentous spine are believed to be highly important in maintaining clinical stability of the spine. White and Panjabi [6] have defined clinical instability as "the loss of the ability of the spine under physiological loads to maintain its pattern of displacement so that there is no initial or additional neurological deficit, no major deformity, and no incapacitating pain." A corresponding checklist for the diagnosis of clinical instability in the lumbar spine includes flexion/extension radiographical criteria in which sagittal plane translations in excess of 4.5 mm and sagittal plane rotations more than 15° (L1-2, L2-3, L3-4) or 20° (L4-5) or 25° (L5-S1) are contributors to, and potential indicators of, clinical instability. An even more generalized concept of clinical instability considers a widened neutral zone as an indicator of spinal instability [8], as shown in Figure 2.4.

PATHOPHYSIOLOGY OF THE PAINFUL LUMBOSACRAL SPINE

Discogenic Pain

As noted earlier, the intervertebral disc is subject to large forces in vivo—primarily axial compression, but also anterior-posterior shear. The healthy disc is considered the largest primarily avascular and aneural organ in the human body—the question, then, is how it can be a source of pain, even under large loads. An influential study by Freemont et al. [9] examined the relationship between degenerative changes of the disc, nerve ingrowth, and chronic low back pain. Histological and immunohistochemical analyses of disc biopsy samples revealed isolated nerve fibers expressing substance P deep within degenerated intervertebral discs, and correlating with presence of pain as established clinically by discography—suggesting a role for neoinnervation of the disc in the pathogenesis of chronic low back pain.

While much current research is focused on elucidating the role of neovascularization (angiogenesis) and altered cell and matrix biology as potential causal factors of neoinnervation, the importance of structural defects (annular tears, fissures) and associated zones of granulation tissue should not be overlooked, as they provide a physical pathway for ingrowth of sensory nerve endings deep into the disc. The precise etiology of annular injury is the subject of ongoing investigations, with mechanical loading of the disc continuing to be strongly implicated. Recent in vivo measurements of intradiscal pressure by Wilke et al. [10] further support Nachemson's earlier findings that the disc experiences high pressures in daily activities. In the Wilke study, measured intradiscal pressure ranged from 0.10 MPa (megapascals), while lying supine, to 2.30 MPa, while lifting 20 kg, bent over with round back (1 MPa is approximately 145 pounds per sq in). Large stresses can develop in the annulus fibrosus as it contains the disc contents under such pressurization. Computational modeling has revealed high stresses in the posterolateral region of the disc, demonstrating the susceptibility of this region to mechanical failure—and correlating convincingly with clinical observations of disc failure in this region [11].

Facet Pain

Likewise the facets routinely encounter large forces in vivo. Under spine axial compression and axial torsion, the articular cartilage covering the bone surfaces of the facet joints experiences high contact stresses. Osteoarthritic changes leading to loss of this cartilage layer can result in painful bone-on-bone contact. Beaman et al. [12] have reported evidence of substance P-containing nerve fibers in the subchondral bone of osteoarthritic facet joints. Moreover, the facet capsule is highly innervated, including both nociceptive and autonomic nerve fibers, and thus can also be a source of pain under abnormal loads.

Biomechanical and Environmental Factors

A recent meta-analysis of 33 studies examined the association between obesity and low back pain [13]. In

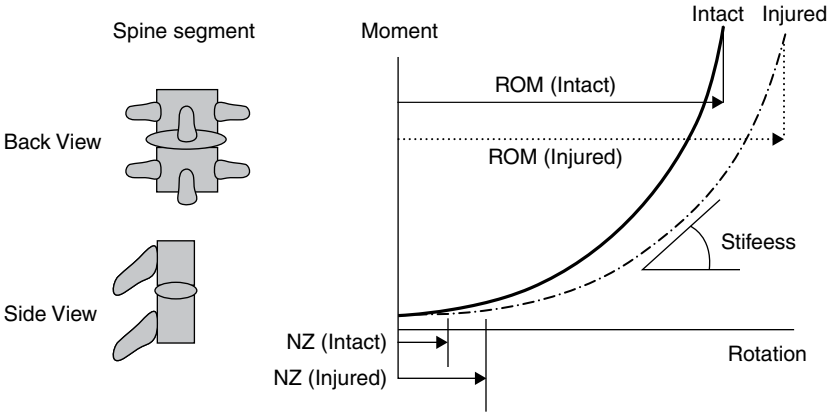

Figure 2.4 Widened neutral zone (NZ) as an indicator of spinal instability. Adapted from Kim DH, Cammisa FP, Fessler RG. Dynamic reconstruction of the spine. New York: Thieme Medical Publishers, Inc.; 2006:355.

cross-sectional studies, obesity was associated with increased prevalence of low back pain in the past 12 months, seeking care for low back pain, and chronic low back pain. If obesity increases low back pain, does weight reduction alleviate symptoms? Khoueir et al. [14] performed a prospective assessment of axial back symptoms before and after bariatric weight reduction surgery. Thirty-eight patients with morbid obesity and chronic axial back pain were assessed preoperatively and 12 months following bariatric surgery, using clinical measures for axial back pain and disability. Mean weight was 144 ± 41 kg preoperatively, decreasing to 106 ± 29 kg postoperatively. Over the 12-month interval, patients demonstrated a statistically significant 44% decrease in axial back pain (Visual Analog Scale) and significant improvements in both SF-36 and Oswestry Disability Index following bariatric surgery. Similarly underscoring the association between spinal loads and low back pain, a meta-analysis of 29 studies of 4173 patients by Chadbourne et al. [15] showed that reduction mammaplasty was associated with a statistically significant improvement in physical signs and symptoms including upper/lower back pain. On the other hand, Videman et al. [16] produced evidence of *positive* effects of greater body mass on disc degeneration. Lumbar magnetic resonance imaging and bone density measurements in 44 pairs of healthy male monozygotic twins with mean 13 kg discordance in body weight showed evidence of a delay in L1–4 disc desiccation in the heavier men as compared with their less heavy twin brothers, and 6.2% higher bone density in the lumbar spine. These findings challenge the common belief that higher body mass is always harmful to discs, although it should be understood that the previous evidence suggests that there is a limit beyond which the effect of increased body mass on the spine clearly is not benign.

Genetic Factors

While lifting heavy loads, torsional stress, and motor vehicle driving are among the most studied environmental risk factors for lumbar disc disease, evidence from family and twin studies suggests that genetic factors are also important—even to the extent of claims that "sciatica, disc herniation and disc degeneration may be explained to a large degree by genetic factors" [17]. Evidence includes the identification of two collagen IX alleles associated with sciatica and lumbar disc herniation, and the relation of disc degeneration to aggrecan gene polymorphism, a vitamin D receptor, and matrix metalloproteinase-3 gene alleles.

PATHOPHYSIOLOGY OF THE PAINFUL SIJ

The sacroiliac joint (SIJ) satisfies the criteria to be considered a pain generator because it has nerve supply and it is susceptible to disease or injuries known to be painful. Pathological conditions of the SIJ that might be the involved as a source of pain are spondyloarthropathies, infection, malignancy, and trauma [18].

Anatomical Principles

The SIJ is a diarthrodial joint with a joint capsule and synovial fluid [18]. It has two bony surfaces, the sacrum and the ilium. The ilium has a convex promontory at the second sacral vertebra (S2), whereas the sacrum is more concave. The joint surfaces are lined with hyaline cartilage, although the iliac cartilage appears thinner and more fibrocartilaginous. The superior third of the hyaline iliac cartilage is strongly attached to the surrounding stabilizing ligaments, forming wide margins of fibrocartilage. The inferior third of the joint along the iliac bone has some histological characteristics of a synovial joint [19].

The long arm of the joint is oriented posterolaterally and caudally, whereas the short arm is positioned posteriorly and cephalic. The morphology of the SIJ varies widely between individuals with respect to size, shape, and contour [20].

The anterior capsule of the sacroiliac is well formed, but the posterior capsule frequently possesses multiple rents and tears [20]. The ligaments supporting the SIJ are the anterior and posterior sacroiliac ligament, interosseus

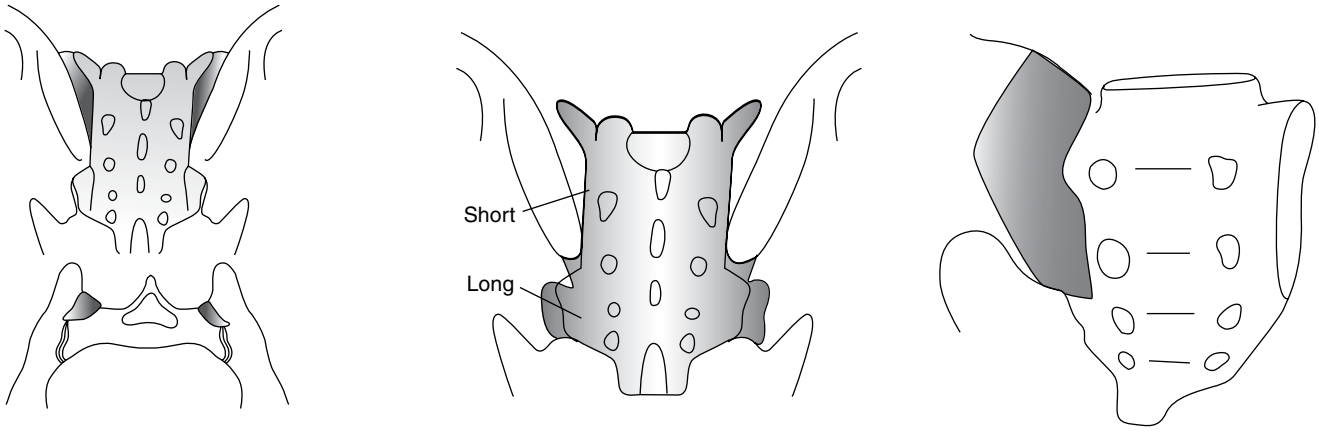

Figure 2.5 Sacropelvic ligaments. Adapted from Bogduk N. *Clinical Anatomy of the Lumbar Spine and Sacrum*. 4th ed. Philadelphia: Elsevier; 2005.

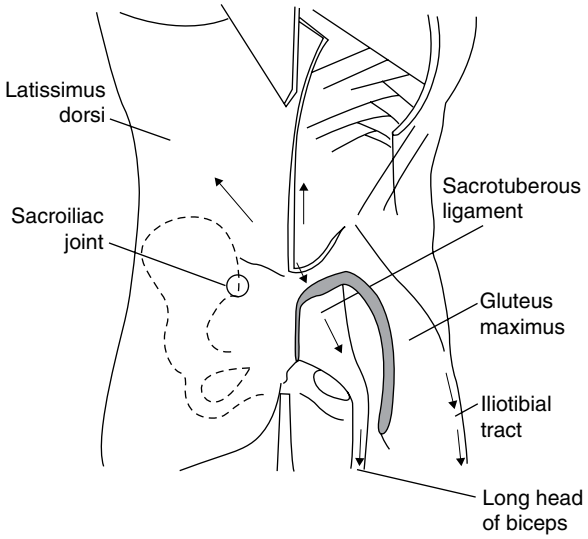

Figure 2.6 Mulculoligametous sling of the lumbosacral region. From ref. [24].

ligament, sacrotuberous ligament, sacrospinous ligament, and iliolumbar ligament [18,21], as shown in Figure 2.5. The interosseous ligament is the strongest ligament supporting the SIJ [18,20]. Structures that have connections or an intimate relationship with the mentioned ligaments are the piriformis, biceps femoris, gluteus maximus and minimus, quadratus lumborum, erector spinae, iliacus, latissimus dorsi, and thoracodorsal fascia [18,22–24], as shown in Figure 2.6. The joint space decreases with age and becomes rougher and filled with debris. As the joint fills and ages, it becomes stiffer and less effective as a shock absorber [25]. The joint does not truly fuse with normal aging [26].

The exact innervation is unclear. The anterior portion of the SIJ receives innervation from the posterior rami of the L1-S2 roots. Additional innervation to the anterior joint may arise directly from the obturator nerve, superior gluteal nerve, and/or lumbosacral trunk. The posterior portion of the joint is innervated by the posterior rami of L4-S3 with a particular contribution from S1 and S2. The S1 level may provide the greatest contribution to the SIJ [18,22,27].

Physiological and Biomechanical Principles

Forces from the lower limbs are transmitted to the trunk through the sacrum [26]. During axial loading, the upper sacrum is forced downward and anteriorly, wedging into the iliac bone. The SIJ acts as a triplanar shock absorber possessing motion that likely does not occur around a single fixed axis. The SIJ is surrounded by some of the largest and most powerful muscles of the body, but none of these muscles have direct influence on joint motion [22,26]. The precise model of SIJ motion is unclear. It seems that the SIJ physiological motion is limited to minute amounts of rotation and translation [28]. Rotation range is between −1.1° and 2.2° along the x-axis, −0.8° and 4.0° along the y-axis, and −0.5° and 8.0° along the z-axis. Translation range is between −0.3 and 8.0 mm along the x-axis, −0.2 and 7.0 mm along the y-axis, −0.3 and 6.0 mm along the z-axis [27].

The SIJ is the only joint in the body that has a flat joint surface oriented almost parallel to the plane of maximal load [21]. It has self-locking properties, occurring through two types of closure: form and force. Form closure represents how specifically shaped and closely fit contacts provide inherent stability independent of external load. Force closure represents the external compression forces that add additional stability, shown in Figure 2.7. Ligaments in this region provide additional support along with the fascia and muscles within the region. They provide significant self-bracing or self-locking to the SIJ due to their cross-like anatomic configuration [24]. Ventrally, this is formed by the external abdominal obliques, linea alba, internal abdominal obliques, and transverse abdominals. Dorsally, this is formed by the latissimus dorsi, thoracolumbar fascia, gluteus maximus, and iliotibial tract. Additional to the prior mentioned musculoligamentous structure, there appears to be an arthrokinetic reflex mechanism by which the nervous system actively controls this added support system [18,21,24].

Pathophysiology

The SIJ was first suggested as a source of lower back pain in 1905 by Goldthwaite and Osgood—but then largely ignored as the intervertebral disc became labeled as the major cause of back pain by Mixter and Barr in 1934 [21]. SIJ pain generally arises in relation to pregnancy, trauma, sports, spondyloarthropathies, infection, and malignancy. Pain is experienced between the posterior iliac crest and the gluteal fold, particularly in the vicinity of the SIJs. It may radiate to the posterior thigh and can also occur with/or separately in the symphysis. The endurance capacity for standing, walking, and sitting is diminished. The incidence in pregnancy may be as high as 20% [21,29]. The situation is different in the general population complaining of low back pain—here it might be up to 15% to 21% [21,22,26,30,31] (see Chapter 1).

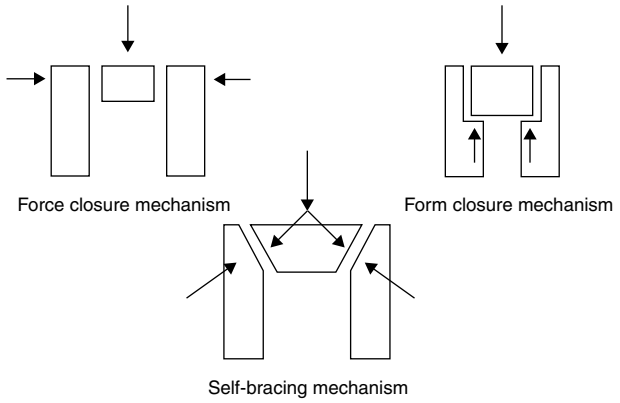

Figure 2.7 Diagrammatic representation of force closure, form closure and the self-bracing mechanism of the SIJ. From ref. [21].

History of trauma can be found in 44% to 58% of individuals. Possible mechanisms include sudden heavy lifting, prolonged lifting and bending, torsional strain, arising from a stooped position, falling onto a buttock, or motor vehicle accident with the ipsilateral foot on the brake [22,32].

SIJ dysfunction can develop in any sport that places significant biomechanical stress through the lumbar spine and pelvis—especially when the pelvis is relatively fixed while primarily transverse plane loads are applied through the lumbosacral region. Imbalances in muscle action may disrupt the normal equilibrium of muscle function around the pelvis and sacroiliac region. It has been found to be common in cross-country skiers and rowers [24], and after lumbosacral fusion [33].

In pregnant women the mechanism might be as a result of the release of the hormone relaxin, which allows pelvic expansion and increased motion [34]. Other factors, such as the trauma of childbirth, altered posture, increased lordosis, and weight gain, also may increase the risk of pain. Asymmetric SIJ laxity measured during pregnancy (Doppler imaging) is predictive of the persistence of moderate to severe pregnancy-related pelvic pain into the postpartum period [35].

Sacroiliitis often leads to typical inflammatory back pain of varying intensity but some patients may remain asymptomatic. Spondyloarthropathies comprise five entities differentiated mainly on a clinical basis: ankylosing spondylitis, reactive arthritis, psoriatic arthritis, arthritis of chronic inflammatory bowel disease, and undifferentiated spondyloarthropathy. Overlapping, transition, and coexistence of these subsets are typical and sacroiliitis can occur in all of them. The SIJs are either unilaterally or bilaterally affected with an intensity ranging from mild to very severe inflammation resulting in partial or complete ankylosis. Symmetrical sacroiliitis is found in more than 90% of ankylosing spondylitis patients and in two-thirds of patients with chronic reactive arthritis and psoriatic arthritis of longstanding disease. Spondyloarthropathies are genetically linked (90% of cases), the strongest contributing factor being human leukocyte antigen B27 [26,36,37]. Nonspondyloarthropathic SIJ pain is typically unilateral [38].

Pyogenic sacroiliitis is relatively rare, representing only 1% to 2% of all cases of septic arthritis. Initial symptoms are usually nonspecific and difficult to differentiate from sciatica or septic arthritis of hip, and sometimes may mimic acute abdomen and sepsis syndrome. Delay in diagnosis may lead to several severe complications such as abscess or sequestrum formation, prolonged period of sepsis, long-term joint deformity and disability, and even death [39].

Giant cell tumors, chondrosarcoma, and metastatic disease are the most frequent tumors involving the SIJ. The following carcinomas have been reported in the literature as having metastasized to the SIJ: hepatocellular, salivary gland, renal, colon, and lung. There are reported cases of malignancies mimicking a unilateral sacroiliitis at first presentation [40].

SUMMARY

The pathophysiology of painful degenerative lumbosacral conditions is indeed complex, as can be seen by the numerous ongoing clinical and scientific investigations of a highly interdisciplinary nature. Residents, fellows, and post-training physicians dedicated to the care of patients with painful lumbosacral conditions are faced with a rapidly growing body of published work written by a diverse group of clinicians and scientists—including bioengineers, biologists, epidemiologists, and geneticists—requiring an interdisciplinary mindset/level of comprehension in order to be able to rapidly assess what pieces of evidence should be considered in the clinical care of patients. While genetic research has been helpful in elucidating the roles of specific genes in lumbosacral disorders, broader genomics research efforts to identify the complex interactions between genes, biological and biomechanical pathways, and environmental factors would appear to be the trend toward future research. For now, the physician must integrate the disparate information streams into a cohesive treatment plan for their patients.

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3 Mechanical Assessment of Axial Lumbosacral Pain

Stephen May and Ron Donelson

INTRODUCTION

The term *nonspecific or mechanical low back pain* has been in common use for several decades and arose from the limited diagnostic acumen of physical examination procedures to identify specific anatomical diagnoses [1,2]. Pathoanatomical or structural diagnoses of low back pain (LBP) by clinical examination have traditionally lacked validity [3]. Most physical examination procedures have poor levels of reliability between clinicians, especially those based primarily on observation and palpation [4,5]. However, physical examination procedures based on symptom response, such as centralization, generally have higher levels of reliability [4,5]. Identification of clinically meaningful pathology via advanced imaging modalities is problematic, as morphologic changes, such as disc degeneration, disc herniations, and spinal stenosis, can be found in the asymptomatic population [6–8]. Nonspecific or mechanical back pain has been the preferred nomenclature for several decades [1,2]. Yet, more recent evidence has emerged that mechanical LBP can be further delineated into the specific structural source (disc, facet joint, sacroiliac joint [SIJ]) of LBP (see Chapter 1).

PHYSICAL IDENTIFICATION OF STRUCTURAL DIAGNOSES

It has been demonstrated that a structural diagnosis can be established in "nonspecific back pain" in some LBP patients (see Chapter 1) using precision, controlled, fluoroscopically guided diagnostic spinal procedures as a reference standard as long as particular diagnostic criteria are maintained [9–11]. Injections must be performed under fluoroscopic control to ensure accurate placement; double blocks are necessary as single blocks are associated with appreciable rates of false positives; in discography, concordant LBP must be produced at one segmental level, with no pain at an adjacent level. Studies on spinal procedure studies meeting stringent operational criteria provide the theoretical framework for understanding the prevalence of different structural diagnoses in chronic mechanical back pain; however, their specialized and intrusive nature reserves these interventions for certain patient populations.

Discography has been deemed controversial; yet, its proponents (see Chapters 4 and 5) have shown it to be the only valid and reliable method to detect primary discogenic LBP [12], and its detractors claim it to have poor specificity and be of limited clinical value [10,13,14]. Traditionally, the only physical examination finding consistently associated with discogenic pain, rather than lumbar radiculopathy, is centralization [3,10,15–17]. Centralization and peripheralization appear to be strongly correlated with discogenic pain, but from the evidence to date it is unclear if this correlation can be used to rule in or rule out the diagnosis. More recent findings support the predictive utility of sustained hip flexion (SHF) [18] and pelvic rock in detecting discogenic LBP. Reproduction of LBP during SHF in which the patient lies supine, hands resting on his or her abdomen, with knees extended lowering his or her limbs in a controlled fashion toward the plinthe (Patel/Slipman) is highly predictive of discogenic LBP.

Regarding SIJ dysfunction, SIJ tests that used palpation and attempt to detect movement abnormalities were consistently found to possess poor reliability, whereas SIJ tests that are based on pain provocation of the patient's concordant symptoms were found to have moderate levels of reliability, though not consistently [19]. In terms of validity, compared with a SIJ injection, no provoking or relieving movements or positions have been found that were unique or especially common to SIJ pain, either in the history or the physical examination [20]; pain provocation tests were not validated against reference standards [21–24], and false-positive SIJ tests are common in populations without confirmed SIJ pathology [25]. However, the use of multiple tests has been shown to be more reliable than single tests [17,26–28]. The diagnostic accuracy of the clinical examination is enhanced if lumbar spine and hip joint pain patients are first excluded—detected using a mechanical evaluation, noting centralization or peripheralization, following which three out of five positive pain provocation tests are used to determine SIJ problems with a reasonable level of validity [16,17,28,29]. The recommended tests are as follows: distraction, compression, thigh thrust, Gaenslen's, and sacral thrust tests [28,29].

In conclusion, it does seem that painful SIJ dysfunction can be diagnosed using clinical examination, but only if a staged differential diagnostic process is used involving

lumbar mechanical evaluation and pain provocation SIJ tests. Demonstration of centralization, peripheralization, or directional preference denotes a lumbar spine problem and discounts the need to examine for SIJ pathology—it should be noted that this may not occur at the initial assessment, but can occur subsequently [30]. LBP produced by SHF strongly suggests discogenic LBP and can be used to corroborate a more extensive staged examination. In the absence of a positive symptomatic or mechanical response to end-range repeated lumbar movements, and in the presence of unilateral LBP toward the buttock, pain provocation SIJ tests should be used. When three of these tests produce concordant pain, a SIJ problem is likely; when all tests are negative, a SIJ problem can be ruled out [29].

The reference standard for identifying zygapophyseal joint (ZJ) pain is controlled comparative local anesthetic blocks (see Chapter 10). Single blocks are associated with at least 27% to 38% false-positive rate in the lumbar spine [11]. Studies traditionally have failed to link any clinical features of history or physical examination with ZJ problems [31–36] and have specifically ruled out certain features that initially had been suggested might be diagnostic [37–40]. At this point in time, compared with criterion standards, it does not seem possible to identify ZJ pain using physical examination findings with any accuracy. It appears most likely to be a diagnosis by exclusion rather than one by positive clinical identification.

LBP location is a possible diagnostic clue. Discogenic LBP is more likely to occupy the midline, whereas SIJ and ZJ pain is more likely to be located para-midline [22,32,18]. When combined with age and positive pain provocation during SHF, discogenic pain is more likely than SIJ or ZJ LBP [18]. Patients 55 years old or younger with complaints of midline LBP that is provoked by SHF have a 94% probability of their LBP being due to a painful intervertebral disc [41].

A void of evidence exists demonstrating that improved treatment outcomes are plausible using astute physical examination findings to diagnose the source of LBP. Whereas surgery has been identified as a useful treatment for lumbar radiculopathy, with better short-term outcomes than conservative management [42], surgery has not proven superior to conservative treatment in the management of chronic discogenic pain [43].

Dynamic Internal Disc Model

One model of pain generation for LBP is the dynamic internal disc model, with the disc the commonest cause of mechanical back pain [44–46]. Pain provocation studies have commonly demonstrated reproduction of patients' LBP with discography [46–51], which can include radiating symptoms [15,46,52–54]. The degree of radiation may reflect the degree of mechanical pressure that the ruptured and weakened annular fibers are subjected to, with outer annular tears being strongly associated with pain [45]. More mechanical pressure is associated with more distal referral of symptoms in anatomic studies in general [55–59]. The mobile disc has been demonstrated in cadaveric experiments [60–63], and in living subjects [64–68], with a posterior displacement of the nucleus on flexion, and an anterior displacement on extension. Increased displacement of the nucleus or pressure on the outer annulus or nerve root may produce more peripheral symptoms, whereas reduced pressure could relieve these symptoms.

The process might develop in a sequential manner, with the distortion, then failure of the annulus leading to the formation of radial fissures, which are a prerequisite of displacement. In its turn the displacement can be checked by the outer annular wall or this can be ruptured also and a complete herniation result. Once the annular wall has been completely breached and the hydrostatic mechanism of the disc is impaired, it is no longer possible to influence the displaced tissue [69].

Other experimental and clinical studies [70,71] support this dynamic internal disc model when in the presence of fissures and disc fragments the effects of normal loading can lead to the unphysiological displacement of discal material, protrusions, and extrusions. The development of radial fissures would seem to be the key factor in the pathology of disc problems. These entities can be painful in themselves, but in some patients these fissures may also act as conduits for intradiscal material to be displaced, to protrude, or to be extruded beyond the contours of the annulus.

Although the end result may be actual disc herniation with nerve root involvement, this only represents the extreme end of the continuum, and a minority of patients. The majority of patients present at an earlier stage in this continuum, with the outer annular wall still intact or if not intact, no nuclear herniation, when the displaced tissue can be influenced by movement and positioning, and when the symptom generating mechanism is reversible. This dynamic disc model may be the anatomical explanation for the clinical phenomena of peripheralization and centralization. For a fuller review on the use of repeated movements to identify symptomatic intervertebral discs, see [72].

MECHANICAL DIAGNOSIS AND THERAPY

In distinction from an anatomic diagnostic approach, the McKenzie method of Mechanical Diagnosis and Therapy (MDT) [73] uses nonspecific mechanical syndromes, which are determined by symptom response to repeated movements and sustained postures. As noted already, symptom response procedures have better levels of reliability than procedures using palpation or observation [4], with kappa values greater than 0.6 for identifying centralization and directional preference [74,75]. Furthermore, the method has prognostic validity; with the identification of centralization and directional preference, a good outcome is likely if treatment is guided by directional preference findings [76–79]. A portion of the remainder of this chapter will consider the algorithmic reasoning used in MDT to rule out red flags, to identify mechanical responders with mechanical syndrome classification, and to identify nonresponders who might fit one of a number of specific categories and who may benefit from alternative interventions, including medical ones. It will also present evidence, where available, that underpins the assessment process, classification system, and treatment efficacy.

THE McKENZIE INSTITUTE
LUMBAR SPINE ASSESSMENT

Date _____

Name _____ Sex M / F

Address _____

Telephone _____

Date of Birth _____ Age _____

Referral: *GP / Orth / Self / Other* _____

Work: Mechanical Stresses _____

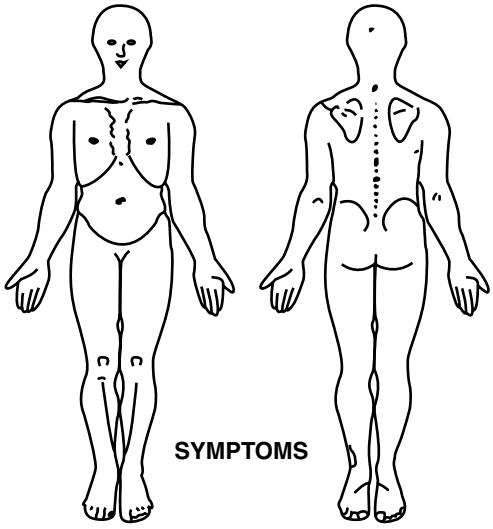
_____

Leisure: Mechanical Stresses _____

Functional Disability from present episode _____

Functional Disability score _____

VAS Score (0-10) _____



SYMPTOMS

HISTORY

Present Symptoms _____

Present since _____ *Improving / Unchanging / Worsening*

Commenced as a result of _____ *Or no apparent reason*

Symptoms at onset: *back / thigh / leg* _____

Constant symptoms: *back / thigh / leg* _____ Intermittent symptoms: *back / thigh / leg*

Worse *bending* *Sitting / rising* *standing* *walking* *lying*

am / as the day progresses / pm *when still / on the move*

other _____

Better *bending* *sitting* *standing* *walking* *lying*

am / as the day progresses / pm *when still / on the move*

other _____

Disturbed Sleep *Yes / No* Sleeping postures: *prone / sup / side R / L* Surface: *firm / soft / sag*

Previous Episodes 0 1-5 6-10 11+ Year of first episode _____

Previous History _____

_____

Previous Treatments _____

_____

SPECIFIC QUESTIONS

Cough / Sneeze / Strain / +ve / -ve Bladder: *normal / abnormal* Gait: *normal / abnormal*

Medications: *Nil / NSAIDS / Analg / Steroids / Anticoag / Other* _____

General Health: *Good / Fair / Poor* _____

Imaging: *Yes / No* _____

Recent or major surgery: *Yes / No* _____ Night Pain: *Yes / No* _____

Accidents: *Yes / No* _____ Unexplained weight loss: *Yes / No*

Other: _____

EXAMINATION

POSTURE

Sitting: *Good / Fair / Poor* Standing: *Good / Fair / Poor* Lordosis: *Red / Acc / Normal* Lateral Shift: *Right / Left / Nil*

Correction of Posture: *Better / Worse / No effect* _____ Relevant: *Yes / No*

Other Observations: _____

NEUROLOGICAL

Motor Deficit _____ Reflexes _____

Sensory Deficit _____ Dural Signs _____

MOVEMENT LOSS

	Maj	Mod	Min	Nil	Pain
Flexion					
Extension					
Side Gliding R					
Side Gliding L					

TEST MOVEMENTS **Describe effect on present pain – During:** produces, abolishes, increases, decreases, no effect, centralising, peripheralising. **After:** better, worse, no better, no worse, no effect, centralised , peripheralised.

	Symptoms During Testing	Symptoms After Testing	Mechanical Response		
			↑ Rom	↓ Rom	No Effect
Pretest symptoms standing:					
FIS					
Rep FIS					
EIS					
Rep EIS					
Pretest symptoms lying:					
FIL					
Rep FIL					
EIL					
Rep EIL					
If required pretest symptoms:					
SGIS – R					
Rep SGIS - R					
SGIS - L					
Rep SGIS- L					

STATIC TESTS

Sitting slouched _____ Sitting erect _____

Standing slouched _____ Standing erect _____

Lying prone in extension _____ Long sitting _____

OTHER TESTS _____

PROVISIONAL CLASSIFICATION

Derangement Dysfunction Posture Other

Derangement: Pain location _____

PRINCIPLE OF MANAGEMENT

Education _____ Equipment Provided _____

Mechanical Therapy: *Yes / No* _____

Extension Principle: _____ Lateral Principle: _____

Flexion Principle: _____ Other: _____

Treatment Goals: _____

McKenzie Institute International 2005©

Figure 3.1 McKenzie lumbar spine assessment sheet. From McKenzie Institute International.

MDT—Assessment

MDT uses standardized assessment sheets (see Figure 3.1) as well as standard questions about the patient's details: the onset, nature and history of the episode, the aggravating and relieving factors, and any history of previous episodes. The history also includes standard red-flag questions to rule out the presence of serious spinal pathology. Although rare [80], back pain can occasionally be caused by cancer, inflammatory disease, fractures, or be accompanied by cauda equina syndrome, which all require specialist referral and are contraindications for MDT. These are best detected by strategic questions during the history taking [2]. The history may also alert the clinician to the presence of "yellow flags": psychosocial barriers to recovery that can impact negatively on treatment. These might be detected by the patient's response to onset of symptoms, their work status, or their opinions about movement and back pain, but can be evaluated more thoroughly using one of a number of screening tools (for instance, Hill et al. [81]). Nonresponse to MDT and high levels of psychosocial barriers may indicate the need to adopt a "pain management" type of approach, but this is likely only in a small minority. Formal assessment of pain levels and disability can also be evaluated using tools such as the Numeric Pain Rating Scale [82] and the Roland-Morris Disability Questionnaire [83], respectively.

The history will also be used for hypothesis-generating about the possible presence of one of the mechanical syndromes, namely derangement, dysfunction, or postural syndrome. Operational definitions for these are given in Table 3.1. Note these are based entirely on symptom and mechanical responses to repeated movements or sustained postures. However, their presence can be suspected or ruled out from items in the history, such as the pain history of the episode (for instance, constant or intermittent symptoms, duration of symptoms, and back pain only or back and referred symptoms) and aggravating and relieving factors. Clinicians experienced in MDT will conclude the history with a clear sense of whether a mechanical syndrome might be present or absent, and whether a directional preference might be present or absent.

The physical examination is used to confirm or deny these hypotheses. The physical examination includes a neurologic examination in the presence of referred pain, consideration of the patient's posture, their symptom response to posture correction, their baseline range of movement, and then uses repeated movements to affect symptoms and range-of-motion, both of which are monitored. Not all the repeated movements on the assessment form (Figure 3.1) will be performed. That selection is based on the clinical reasoning of the clinician. The standard algorithm is to examine sagittal plane movements before frontal plane movements, except in the presence of a lateral shift. If sagittal plane movements exacerbate or peripheralize symptoms, frontal plane movements will be explored. A standard set of terms are used to describe the various types of symptom response during and after the repeated movements (Table 3.2).

During the physical examination, symptom response is monitored for the presence of centralization or directional preference. Operational definitions are provided in Table 3.3; again, these are based entirely on symptom or mechanical responses. Both centralization and directional preference identify the presence of a derangement, which is by the far largest classification amongst spinal mechanical syndromes. At the end of the physical examination, the clinician will make a provisional classification, to be confirmed at follow-up visits.

Table 3.1 Operational Definitions for Mechanical Syndromes

Reducible derangement
- Centralization in response to therapeutic loading strategies
- Each progressive abolition is retained over time, until all symptoms are abolished, and
- If back pain only is present, this moves from a widespread to a more central location and then is abolished, or
- Pain is decreased and then abolished during the application of therapeutic loading strategies
- The change in pain location, or decrease or abolition of pain remain better, and
- Should be accompanied or preceded by improvements in the mechanical presentation (range of movement and/or deformity)

Irreducible derangement
- Peripheralization of symptoms: increase or worsening of distal symptoms in response to therapeutic loading strategies, and/or
- No decrease, abolition, or centralization of pain

Dysfunction
- Spinal pain only, and
- Intermittent pain, and
- At least one movement is restricted, and the restricted movement consistently produces concordant pain at end-range, and
- There is no rapid reduction or abolition of symptoms, and
- No lasting production and no peripheralization of symptoms

Postural syndrome
- Spinal pain only, and
- Concordant pain only with static loading, and
- Abolition of pain with postural correction, and
- No pain with repeated movements, and
- No loss of range of movement, and
- No pain during movement

Table 3.2 Terms Used to Monitor Symptom Response

Standardized terms[a]	
During loading	
Increase	Symptoms already present are increased in intensity
Decrease	Symptoms already present are decreased in intensity
Produce	Movement or loading creates symptoms that were not present prior to the test
Abolish	Movement or loading abolishes symptoms that were present prior to the test
Better	Symptoms produced on movement, decrease on repetition
Centralizes	Movement or loading abolishes the most distal symptoms
Peripheralizes	Movement or loading produces more distal symptoms
After loading	
Worse	Symptoms produced or increased with movement or loading remain aggravated following the test
Not worse	Symptoms produced or increased with movement or loading return to baseline following the test
Better	Symptoms decreased or abolished with movement or loading remain improved after testing
Not better	Symptoms decreased or abolished with movement or loading return to baseline after testing
Centralized	Distal symptoms abolished by movement or loading remain abolished after testing
Peripheralized	Distal symptoms produced during movement or loading remain after testing
No effect	Movement or loading has no effect on symptoms during or after testing

[a] These are the words used to describe symptom response during the physical examination.

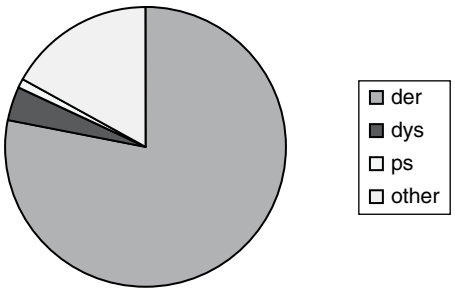
Table 3.3 Operational Definitions for Centralization and Directional Preference

Centralization
- Distal pain is abolished in response to repeated movements or sustained positions and remains better
- Remaining spinal pain is abolished in response to repeated movements

Directional preference
- Centralization, or
- Decrease in symptoms in response to repeated movements, or
- Increase in range of movement in response to repeated movements

Classification, Centralization, and Directional Preference

Evidence suggests that the majority of patients with back pain classified by MDT-trained clinicians are classified into one of the three mechanical syndromes, of which the majority are classified with derangement syndrome [4,78]. Derangement syndrome has been identified in about 70% to 80% of patients with spinal problems [4,78,84] (Figure 3.2). In a systematic review, centralization, which only occurs in the derangement syndrome, was identified in 70% of 731 patients with subacute and 52% of 325 patients with chronic LBP [76]. In a randomized controlled trial, directional preference was identified at the initial mechanical evaluation in 230 of 312 (74%) acute-to-chronic patients with LBP [78].



der, derangement; dys, dysfunction; ps, postural syndrome

Figure 3.2 Classification of 607 patients with spine pain. From Ref. [4].

The importance of these findings is that they have consistently been found to be associated with good prognosis [76–79,85,86]. Centralization is the only physical examination finding that has been shown to predict outcomes 1 year later [86]. Equally, noncentralization is associated with poor outcomes and nonorganic signs, overt pain behaviors, fear of work activities, and somatization [86,87]. In other words, noncentralization indicates patients with more marked psychosocial barriers to recovery who will likely need a cognitive behavioral approach of management. If no change has occurred by seven sessions, there is unlikely to be further improvement with this approach [85]. However, although the majority (97%) of those showing a centralization response on day one will continue to show this positive response, 60% of those not responding initially will come to show a centralization response at multiple visits [30]. The presence of centralization or directional preference not only indicates a good prognosis but also a clear guidance for management using the repeated movement exercises that produced this favorable response. The patient is instructed to continue these exercises as long as a similar response is forthcoming.

Thus, for each mechanical syndrome, a clear therapy management strategy is preordained:

- For derangement—exercises that centralize, abolish, or decrease symptoms and regain range of movement.
- For dysfunction—end-range exercises that reproduce symptoms, but which are not worse after, and change only slowly.
- For posture syndrome—education in posture correction to avoid end-range sustained loading.

Management is chiefly then an exercise, or end-range lumbar bending movement, which is repeated regularly every 2 to 3 hours by the patient, and is supplemented by education about posture, interrupting some loading strategies, temporarily avoiding some activities, and maintaining general activity. If the patient is out of work, a major emphasis is on returning them to work as soon as possible. As can be seen, the emphasis is on a patient-centered self-treatment approach to management, in which they are the

chief architects of their recovery. If recovery plateaus or slows, patient exercises can be supplemented with manual therapy, but this is only used to return the patient to the position where they can self-manage again. As the episode resolves, the emphasis shifts to considerations of prevention of future episodes. This is done through consideration of postural loads, by encouraging general exercise, and by use of the treating exercises proactively, as a preventative measure. The value of using extension-in-lying exercises to prevent recurrences of LBP has been demonstrated in a controlled trial [88].

Centralization has been demonstrated in patients with nonspecific back pain as well as in those with sciatica. In both cases, it was associated with a good prognosis. Patients with sciatica who are noncentralizers are six times more likely to need surgery [89]. This group of patients with disc herniations and lumbar radiculopathy are more likely to require a surgical intervention. In this subgroup, it has been demonstrated that the introduction of a McKenzie assessment and treatment clinic in one county in Denmark caused a decline in the rate of first-time lumbar disc surgery by about two thirds compared with the levels of disc surgery in the rest of Denmark [90].

Nonresponders and Nonclassified

The MDT algorithm identifies those who will respond and those who will not respond with the approach. Most nonresponders cannot be classified into one of the mechanical syndromes. The exception is the nonresponders classified with derangement, but further classified as being irreducible. Most with an irreducible derangement will have more severe symptoms with lumbar radiculopathy, and probably accompanying neurologic signs and symptoms. If their response does not change, they may become candidates for surgery. As noted earlier, many with sciatica symptoms will respond to this type of conservative management [89,90], but some will not. These will be rapidly identified by their failure to centralize and by all repeated movements producing peripheralization or worsening of peripheral symptoms. If these symptom responses to the MDT examination continue over several sessions, these patients would be referred on for further investigations.

Not all patients with back pain would be classified with a mechanical syndrome, but the MDT algorithm first excludes the presence of one of the mechanical syndromes before considering alternative or "other" classifications. Thus, repeated movement testing could be continued for up to five sessions to demonstrate the required response. More experienced MDT clinicians would classify a nonresponder that could not be classified into one of the three mechanical syndromes more quickly than this. If, after a number of clinic sessions there is a failure to classify a mechanical syndrome, one of the "other" classifications could be considered; the main ones being spinal stenosis, hip joint pathology, SIJ dysfunction, mechanical inconclusive, spondylolisthesis, and chronic pain state. Each of these is a very small classification group and each has clearly defined operational definitions [73] (Table 3.4).

Some patients classified into one of the "other" classifications may respond to specific exercises. For instance, in those with spinal stenosis, a trial of directional exercises would be trialed to see if these might improve symptoms. Those with hip symptoms can often be classified into a mechanical syndrome related to the hip joint and respond well to appropriate self-treatment. Some with SIJ problems might similarly respond mechanically and be classified as an SIJ derangement [91], and those classified as chronic pain state would be recommended to a pain management program.

Two surveys of MDT clinicians reported that 17% to 19% of 765 spine patients were classified as irreducible derangement or "other" [4,82], with 6% identified as irreducible derangement [82], 6% as mechanically inconclusive, 4% as chronic pain state, about 2% as postsurgery, and about 1% each as stenosis, spondylolisthesis, SIJ dysfunction, and posttrauma [4]. The remainder were classified with one of the mechanical syndromes, the majority with derangements.

Effectiveness

The emphasis in this chapter has been about describing the assessment, examination, classification, and management system of MDT, but it is obviously relevant to identify reviews that analyze the effectiveness of this approach. A number of recent reviews, with different inclusion/

Table 3.4 Operational Definitions for "Other" Classifications

Stenosis
• Leg symptoms when walking
• Eased when sitting/leaning forward
• Loss of extension
• Age more than 50
• Possible nerve root signs and symptoms
• Extensive degenerative changes on x-ray
• Extension provokes symptoms
• Confirmation with CT/MRI
Spondylolisthesis
• Adolescent
• Recent sports related onset back pain
• Sport involves regular flexion/extension
• Confirmation on investigation
Sacroiliac joint
• Exclude lumbar spine
• Exclude hip
• Buttock pain
• Three or more positive pain provocation tests
Hip
• Specific pain pattern
• Pain on walking, eased on sitting
• Positive hip provocation tests
Mechanically inconclusive
• Symptoms affected by spinal movements
• No loading strategy consistently decreases, abolishes, or centralizes symptoms, nor increases or peripheralizes symptoms
• Inconsistent response to loading strategies
Chronic pain state
• Persistent widespread symptoms
• All activity increases symptoms
• Exaggerated pain behavior
• Mistaken beliefs and attitudes about pain and movement

exclusion criteria, are relevant here. These reviews have consistently been positive about the MDT approach, some for acute back pain, and some for chronic back pain, and they have also highlighted the improved outcomes gained from classification-based treatment [92–99].

CONCLUSION

This chapter has primarily been concerned with a brief overview of the McKenzie method of MDT to the assessment and management of patients with LBP. The emphasis here has been about the clinical reasoning algorithm that an MDT clinician would go through, which does not use specific pathoanatomical diagnoses, but rather focuses on nonspecific mechanical syndromes based on symptomatic and mechanical responses. There are some clinical presentations that suggest pathoanatomical diagnoses, but no clear link between a structural diagnosis and specific and optimal treatment.

The history taking is used to try to exclude patients with serious spinal pathology, using questions about "red flags," and also to try to identify patients with "yellow flags," that is patients with psychosocial barriers to recovery. The history taking is also used to help identify or exclude one of the MDT syndromes. The physical examination uses assessment of posture, posture correction, range of movement, and a neurologic examination (if appropriate) to establish baseline measures, and then uses repeated movements (sometimes sustained positions) during which symptomatic and mechanical responses are monitored. At the conclusion of the total assessment process, the patient is given a provisional classification. This will be confirmed at the next session, depending on their response to the prescribed exercises.

Classification into one of the mechanical syndromes is based on symptom and mechanical responses to repeated movements. Management is derived from the classification, with different management strategies dependent on the established classification. A minority of patients do not fit the MDT classification operational definitions, and might be classified in another way, and need an alternative management approach.

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4 Diagnostic Imaging of Lumbosacral Internal Disc Disruption

Paul E. Verrills, Nikolai Bogduk, and David G. Vivian

This chapter will focus primarily on the pathophysiology of internal disc disruption (IDD) and the indications and evidence for imaging studies. The interpretation of morphological abnormalities and an evidence-based review of the predictive value of radiographic findings will be included.

INTERNAL DISC DISRUPTION

IDD accounts for 39% of patients with low back pain [1], and although it is the most comprehensively understood cause of low back pain (Figure 4.1), diagnosis and treatment remains a challenge. IDD is not disc degeneration. It is a specific condition characterized by degradation of the matrix of the nucleus pulposus with radial fissures that penetrate the annulus fibrosus, but do not breach the outer lamella (Figure 4.2). The fissures may be entirely radial, or a radial fissure may extend in a concentric manner around the outer annular layers. The extent of fissuring may be graded according to whether the radial fissure reaches the inner, middle, or outer third of the annulus [2], or whether it extends circumferentially [3] (Figure 4.3).

The morphological features of IDD cannot be demonstrated by plain radiography or CT. Even MRI is of limited value. The features can only be shown by postdiscography CT (Figure 4.3).

A large study, using multiple regression analysis, showed that age changes and degenerative changes did not correlate with the disc being painful [4]. Grade III fissures, however, correlated strongly with pain and were not related to age changes (Table 4.1) [4].

The etiology of IDD has been established. Biomechanics experiments have shown that the vertebral endplate is subject to fatigue failure [5]. Subject to loads of 37% to 50% ultimate tensile strength, endplates can fracture after 2,000 or 1,000 repetitions, whereas in the case of 50% to 80% ultimate tensile strength, they can fail after as few as 100 cycles [6,7]. Such loads and repetitions are well within the ranges encountered during moderately heavy work activities.

When subjected to repeated compression loading, discs exhibit mechanical failure. If examined morphologically, the failure coincides with the presence of an endplate fracture. Furthermore, upon fracture of the endplate, the

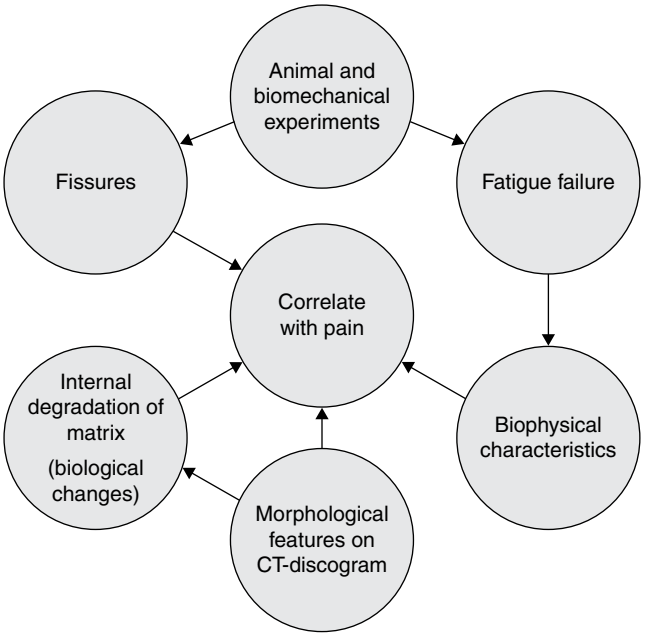

Figure 4.1 A synopsis of the correlates of internal disc disruption.

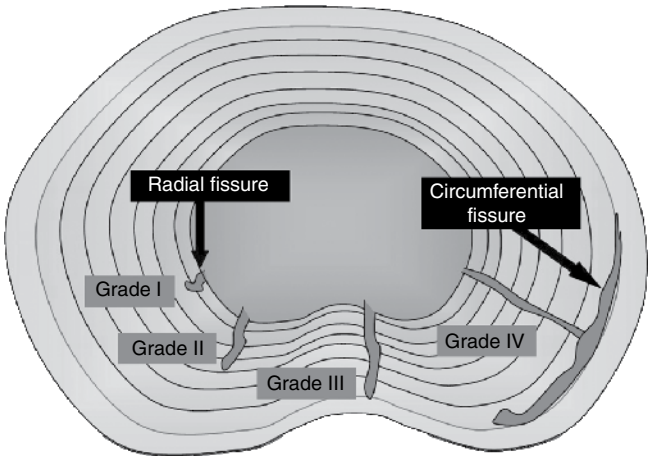

Figure 4.2 A sketch of a transverse section of a lumbar intervertebral disc, showing the grading of internal disc disruption according to the degree of fissuring of the annulus fibrosus.

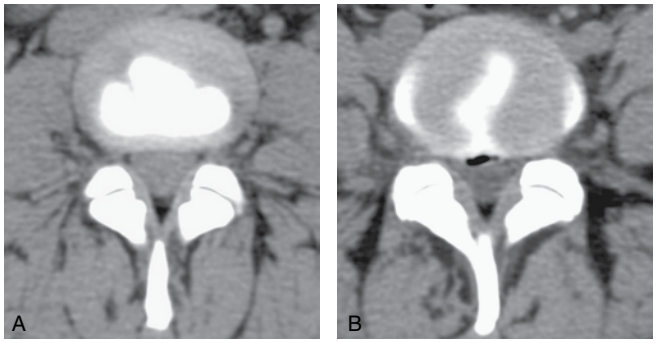

Figure 4.3 The appearance of discs on CT-discography. **(A)** Normal L3/4 disc. The nucleus is rounded and contained within an intact annulus. **(B)** Internal L4/5 disc disruption with a central posterior grade III fissure.

Table 4.1 The Correlation Between Anular Disruption and Reproduction of Pain from the Affected Disc

Pain Reproduction	Annular Disruption Grade			
	III	II	I	0
Exact	43	29	6	4
Similar	32	36	21	8
Dissimilar	9	11	6	2
None	16	24	67	86

Based on Moneta et al. [4].

disc exhibits the onset of the biophysical features of IDD; the nucleus is depressurized and posterior annulus stress abruptly increases (Figure 4.4).

The biochemical features of IDD have also been induced in live animal models [8]. Experimental fracture of an endplate causes deaggregation of proteoglycans in the nucleus, a reduction in water content, and depressurization of the nucleus, as well as delamination of the annulus.

IDD also exhibits highly specific biophysical features. Stress profilometry is a technique whereby the internal stresses within a disc, across its diameter, can be measured. Normal discs exhibit a uniform distribution of stress across the anterior annulus, the nucleus pulposus, and the posterior annulus [5] (Figure 4.5). In discs affected by IDD, two patterns emerge. The nucleus stresses are irregular and reduced, and may be zero in some discs (Figure 4.6). In the posterior annulus, the stresses are raised above normal (Figure 4.6). The depressurization of the nucleus reflects the degradation of the nuclear matrix, which can no longer retain water efficiently. This results in extra loading of the posterior annulus.

Each of these biophysical features correlates with the disc being painful [9] (Table 4.2). Discs with increased posterior annulus stress are likely to be painful; discs with normal annulus stress are uncommonly painful. Discs with a depressurized nucleus are highly likely to be painful; discs with normal nuclear pressure may or may not be painful. Painful discs are likely to exhibit increased annulus stress and a depressurized nucleus.

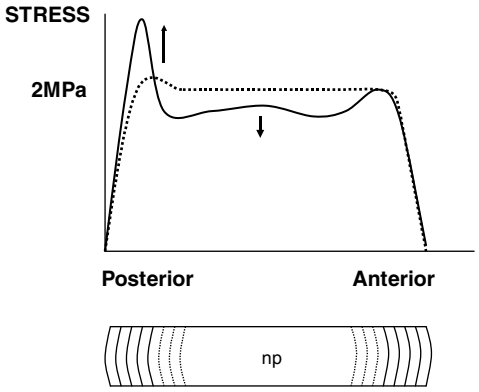

Figure 4.4 Stress profilometry of a disc immediately after the onset of a fatigue fracture of its vertebral endplate. The nucleus is depressurized and the posterior annulus stress increased markedly.

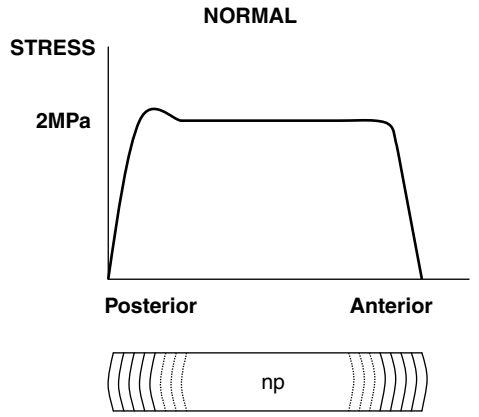

Figure 4.5 Stress profilometry of a normal disc. The stress is uniform across the anterior annulus, nucleus, and posterior annulus.

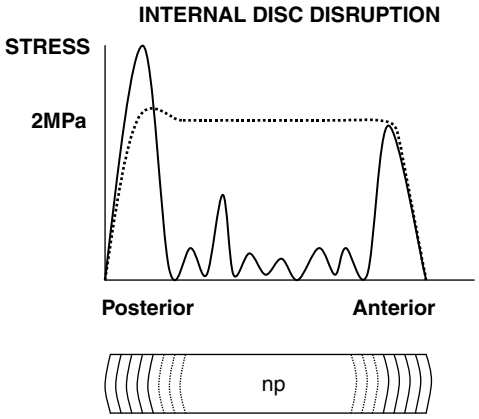

Figure 4.6 Stress profilometry of internal disc disruption. Nucleus stress is reduced and irregular. Posterior annulus stress is increased.

Table 4.2 The Correlation Between Pain and Each of Increased Annular Stress and Decreased Nuclear Stress

	Pain	No Pain	Fisher's Exact Test
Anular stress			
Stressed	17	2	
Normal	1	11	$P = .001$
Nuclear stress			
Depressurized	11	0	
Normal	7	13	$P = .017$

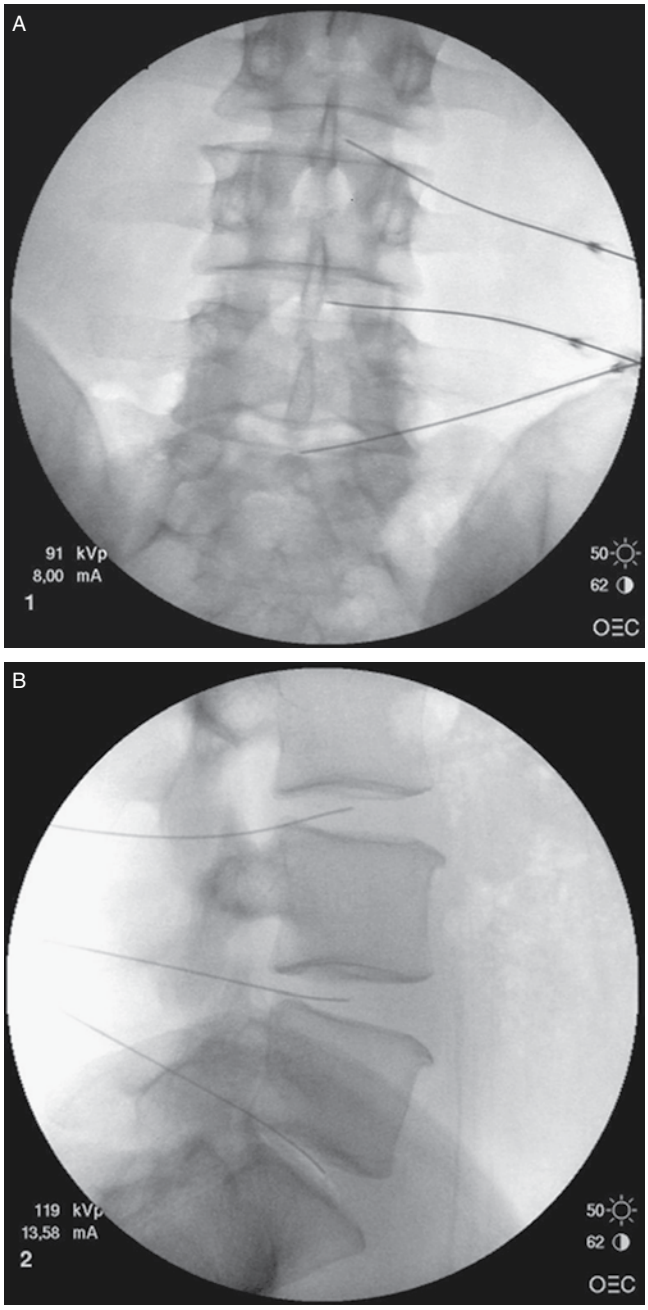

Figure 4.7 (A,B) The appearance of the three lower lumbar discs, after injection of contrast medium into the nucleus. L4/L5 central posterior fissure and L5/S1 posterolateral fissures.

Little is known about the complex forces acting on the deformable multilayered annulus at a microstructural level as the spine is compressed, flexed, and twisted. The recently described translamellar bridging network radially linking many lamellae at discrete locations around the disc wall could be expected to play a significant biomechanical role. The New Zealand research group found that typically encompassing a width of 300 to 600 μm, translamellar bridging fibers proceed radially in the interbundle space within an individual lamella. Upon traversing the lamella, the bulk of these radial fibers bend through 90° to merge with the fibers of the adjacent lamellae. The central fibers of this bridging system continue into the equivalent bridging structures in the adjacent lamellae. This work promises to open up new levels of understanding in how radial fissures and IDD evolve [10].

DIAGNOSIS

Clinical studies have determined that IDD is the basis for pain in as many as 40% of patients with chronic low back pain [11,12]. This estimate of prevalence is a worst-case estimate. It excluded two-level disease. The prevalence of IDD may be considerably higher than 40%; but 40% itself amounts to a considerable proportion of patients in whom a pathoanatomical diagnosis can be established.

The diagnostic criteria for IDD are reproduction of the patient's pain by stimulation of the affected disc (Figures 4.7 and 4.8), such that the evoked pain has an intensity of at least 7 on a 10-point scale, and pain is reproduced at a low pressure of stimulation, 15 psi (1 kg cm^{-2}), provided that adjacent discs does not reproduce pain, and postdiscography CT demonstrates a grade III or IV fissure (Figure 4.9).

CONTROVERSY

Some investigators have warned that disc stimulation may produce false-positive responses. They based this warning on the responses to disc stimulation of sets of patients who had no symptoms, who had chronic pain but not back pain, and who had been diagnosed as having a somatization disorder [13,14]. Explicitly, they imputed false-positive rates of 10%, 40%, and 75% in these groups, respectively. However, those percentages were based on sample sizes of only 10, 10, and 4 patients, respectively (Table 4.3) [13]. These small sample sizes result in wide confidence intervals of the estimated rates, which render them poorly representative. Other considerations modify the estimates as well.

The cited estimates did not adhere to the recommended criteria for disc stimulation. They were not subject to anatomical controls, which require that adjacent levels be not painful. They were not subject to manometric criteria. If the original data is reanalyzed with the criterion for anatomical controls applied, the imputed false-positive rate in asymptomatic subjects remains at 10%, but reduces to 20% for patients with chronic pain. The rate for subjects with somatization remains 75% (Table 4.4) [13]. The confidence intervals remain wide.

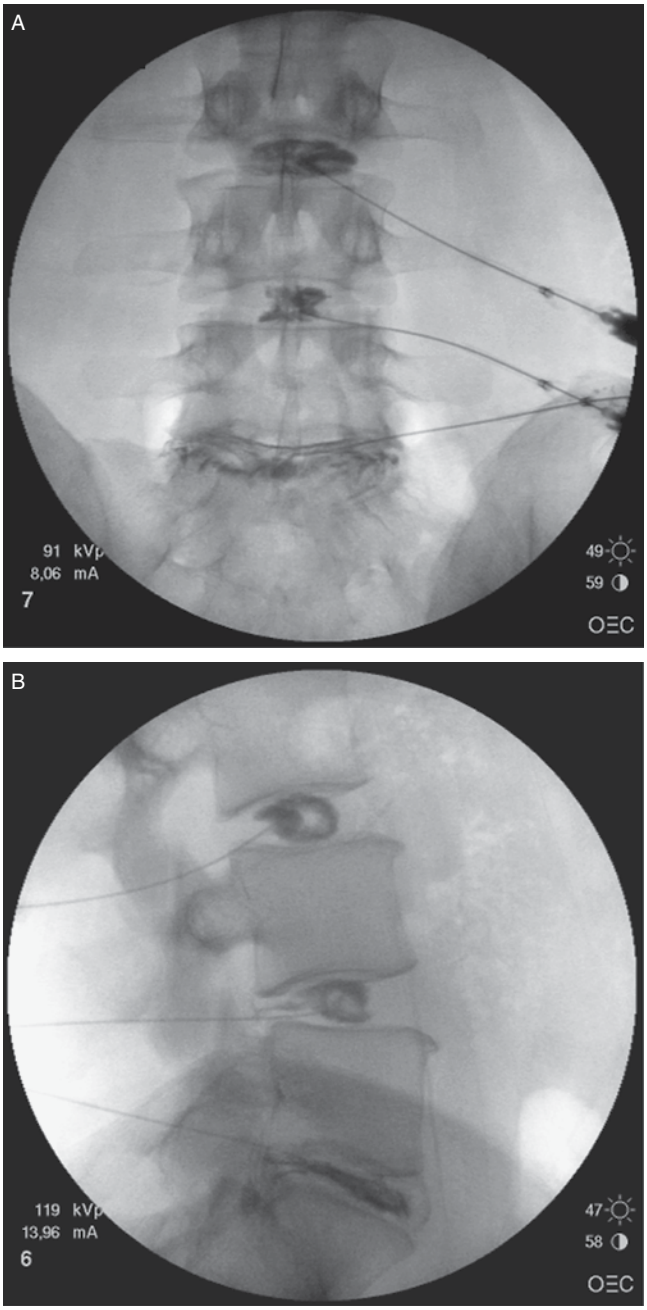

Figure 4.8 (A,B) Placement of needles into the three lower lumbar discs, before disc stimulation.

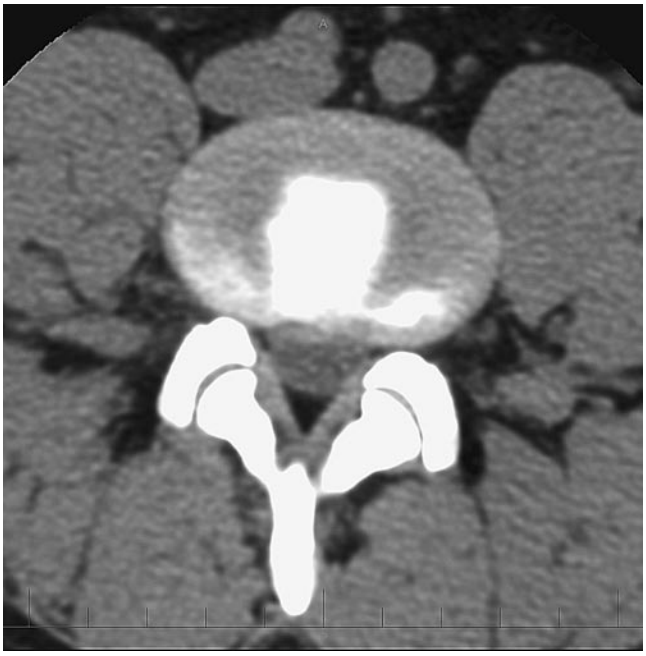

Figure 4.9 The diagnostic features of internal disc disruption on CT-discography. Posterolateral radial grade IV fissure with circumferential extension.

Table 4.3 The Imputed False-Positive Rate of Disc Stimulation in Three Categories of Subjects

Category of Subject	Imputed False-Positive Rate	95% Confidence Intervals
Asymptomatic	1/10 = 10%	0–29%
Chronic pain	4/10 = 40%	10–70%
Somatization	3/4 = 75%	33–100%

Based on Carragee et al. [13].

Table 4.4 The Imputed False-Positive Rate of Disc Stimulation in Three Categories of Subjects, if the Criterion for Anatomic Controls is Applied

Category of Subject	Imputed False-Positive Rate	95% Confidence Intervals
Asymptomatic	1/10 = 10%	0–29%
Chronic pain	2/10 = 20%	0–45%
Somatization	3/4 = 75%	33–100%

Based on Carragee et al. [13].

Manometric criteria are essential for disc stimulation, because it is a provocation test. In principle, any disc, even a totally normal one, might be painful if it is stressed strongly enough. The pressure limits beyond which a disc should not be stimulated can be derived from data available on normal volunteers [15].

If asymptomatic volunteers, or volunteers who have experienced back pain only occasionally, undergo disc stimulation, a pattern of responses emerges. In some subjects, some discs are not painful even if the disc is stressed to 100 psi (6 kg cm^{-2}). Otherwise, there is a twofold trend. The chance that a disc is painful increases as the pressure of the stimulation is increased, but if the disc is painful the intensity of pain tends to be low and the pain is unlikely to be severe (Table 4.5) [15].

Across such data, a boundary can be identified: at pressures below which pain does not occur in normal volunteers, or at which the intensity of pain does not exceed certain prescribed values (Table 4.5). For example, the chances are effectively zero that subjects with no history of back pain with normal psychometric parameters will perceive pain if their discs are stimulated up to a pressure of 20 psi. Alternatively, the chances are zero that they will

Table 4.5 The Responses to Disc Stimulation of Subjects with no History of Back Pain (No) and Subjects with a History of Occasional Back Pain Only (Occ), According to the Pressure of Stimulation and the Intensity of Pain Evoked

PRESSURE (psi)	VAS	0	1	2	3	4	5	6
100	Occ	0.30	0.40	0.25	0.25	0.25	0.10	0.00
	No	0.17	0.48	0.30	0.22	0.09	0.04	0.04
90	Occ	0.35	0.40	0.25	0.25	0.25	0.10	0.00
	No	0.22	0.43	0.30	0.22	0.09	0.04	0.04
80	Occ	0.55	0.30	0.25	0.25	0.25	0.10	0.00
	No	0.22	0.43	0.30	0.22	0.09	0.04	0.04
70	Occ	0.55	0.30	0.25	0.25	0.25	0.10	**0.00**
	No	0.52	0.30	0.17	0.13	0.04	0.00	**0.00**
60	Occ	0.65	0.30	0.25	0.25	0.25	0.10	**0.00**
	No	0.65	0.30	0.17	0.12	0.04	0.00	**0.00**
50	Occ	0.75	0.20	0.15	0.15	0.15	0.05	**0.00**
	No	0.83	0.17	0.09	0.06	0.04	0.00	**0.00**
40	Occ	0.80	0.15	0.10	0.10	0.10	**0.00**	**0.00**
	NO	0.96	0.04	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**
30	Occ	0.95	0.05	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**
	No	1.00	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**
20	Occ	1.00	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**
	No	1.00	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**	**0.00**

The tabulated figures are the cumulative frequency of responses that reflect the chances of pain of a particular intensity occurring at a particular pressure of injection. The light grey area indicates the boundary below which normal volunteers do not experience pain. Adopted from Derby et al. [15]

Table 4.6 The Imputed False-Positive Rate of Disc Stimulation in Three Categories of Subjects, if the Criterion for Anatomic Controls is Applied Together with the Manometric Criterion of 50 psi

Category of Subject	Imputed False-Positive Rate	95% Confidence Intervals
Asymptomatic	1/10 = 10%	0–29%
Chronic pain	1/10 = 10%	0–29%
Somatization	2/4 = 50%	1–99%

Table 4.7 The Imputed False-Positive Rate of Disc Stimulation in Three Categories of Subjects, if the Criterion for Anatomic Controls is Applied Together with the Manometric Criterion of 15 psi

Category of Subject	Imputed False-Positive Rate	95% Confidence Intervals
Asymptomatic	0/10 = 0%	0–28%
Chronic pain	0/10 = 0%	0–28%
Somatization	1/4 = 25%	0–69%

perceive pain of intensity 6/10 or more if their discs are stimulated up to a pressure of 70 psi.

These data vindicate previously invoked, ad hoc, operational criteria [16]. At injection pressures of up to 50 psi, it is highly unlikely that subjects with no history of back pain will experience pain with intensity levels exceeding 6/10. Similarly, up to 15 psi, none of these subjects should experience any pain. Applying these manometric criteria reduces the imputed false-positive rate of disc stimulation.

If the criterion of 50 psi is applied, the false-positive rates in asymptomatic subjects and in subjects with chronic pain fall to 10% (Table 4.6), which are clinically tolerable levels. If the criterion of 15 psi is applied, the false-positive rates become zero in asymptomatic subjects and in subjects with chronic pain. In patients with somatization, they fall to 25% (Table 4.7).

These considerations indicate that the threat of false-positive responses to disc stimulation have been exaggerated. In asymptomatic individuals and in patients with chronic pain, the imputed false-positive rate is effectively zero, provided that the stringent operational criteria for disc stimulation are satisfied. Only in patients with somatization, might a concern about false-positive responses be justified.

What the false-positive rate might be in such patients is not clearly evident because of the small sample size that has been studied; but it does appear to be greater than zero.

A systematic review and meta-analysis of 11 studies identified a false-positive rate of 5.6% per patient and 3.85% per disc among chronic pain patients, asymptomatic of low back pain. Postdiscectomy patients have false-positive rates of 15% and 9.1%, respectively.

Based on meta-analysis of the data, using the International Spine Intervention Society (ISIS) standard, discography has a specificity of 0.94 (95% CI, 0.88–0.98) and a false-positive rate of 0.06 [17].

IMAGING

The role of imaging in the diagnosis of lumbosacral internal disc disruption (LIDD) syndrome is evolving. Because the diagnosis of LIDD demands lumbar disc stimulation (LDS), other imaging techniques, especially MRI, are used to exclude rare and exotic causes of back pain, and to establish whether a clinical presentation should proceed to LDS. The clear challenge is for diagnostic imaging to provide equivalent information to LDS about the likelihood of a disc being not only painful but also treatable.

The traditional diagnostic algorithm for axial back pain has been plain x-ray, CT scan, and then MRI, with bone scan variably included. Diagnostic imaging has a major role in the exclusion of red-flag conditions affecting the lumbar spine. In contrast, its role in the diagnosis of non red-flag conditions is limited because common morphological changes are representative of genetic and age-related changes, which in turn are either unrelated or marginally related to low back pain. MRI has high sensitivity and is, thus, also the best screening test for red flag and unusual causes of back pain, such as tumors, infections, and metabolic disorders.

Plain x-rays are unhelpful; many studies have demonstrated that radiography of the lumbar spine in patients with simple acute low back pain is not associated with improved patient functioning or severity of pain. Indeed, the overall health status of those who had a lumbar spine x-ray appeared to be worse than those who did not [18]. Early use of imaging does not appear to affect overall treatment [19,20]. Further, in omitting x-rays no serious diagnoses were missed, and symptom resolution, functional improvement, and satisfaction were similar [21].

Lumbar imaging for low back pain without indications of serious underlying conditions does not improve clinical outcomes. Therefore, clinicians should refrain from routine, immediate lumbar imaging in patients with acute or subacute low back pain and without features suggesting a serious underlying condition [22,23]. Further, 380 patients presenting in primary care with low back pain were randomized to receive either plain radiographs or rapid MRI, demonstrated no significant difference in outcome measures at 12 months follow-up. The costs were higher in the MRI group and there was a trend toward having more surgery [24,25].

LIDD is suspected in a clinical presentation of axial low back pain with somatic referred pain. The gold standard for the diagnosis of LIDD is LDS. The final clinical decision that predicates LDS is determined by lumbar MRI. Discogenic pain, as diagnosed on LDS, is uncommon in pristine discs [26], and although an MRI can find high-intensity zones (HIZ) as an indirect indication of LIDD, a normal MRI does not exclude significant morphological change in the intervertebral disc [27]. Furthermore, the relatively low sensitivity (26.7% to 59%) and high false-positive (24%) and false-negative (38%) rates reduce the value of MRI in screening for the existence of painful IDD (reviewed in [1]). Clinicians need to determine whether or not it is, therefore, worthwhile subjecting patients to LDS if MRI is normal. Although the odd clinical scenario might present where such investigation may appear indicated, in general terms LDS should not be performed on patients with a normal lumbar MRI. Certain features—Modic lesions and HIZ—evident on MRI increase the likelihood that the affected disc has IDD and is painful.

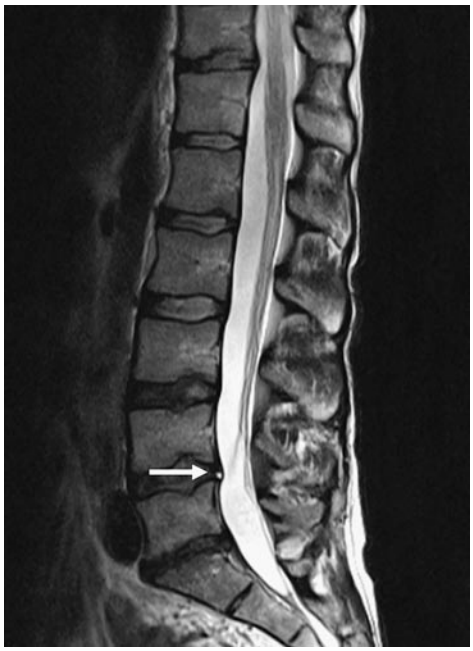

Figure 4.10 The anatomy of high-intensity zones (HIZ). The HIZ seen on sagittal MRI of an L4/5 disc (arrowhead).

HIZ are very bright signals contained within the posterior anulus fibrosus, as seen in sagittal sections on MRI. They are sagittal sections of circumferential fissures (Figure 4.10). They represent nucleus pulposus material migration along a radial fissure [28]. However, not all fissures or gray spots on an MRI constitute HIZ (Figure 4.11). To constitute HIZ, the zone must have a very bright signal on heavily T2-weighted scans; the brightness should rival or exceed that of the cerebrospinal fluid.

The original study of HIZ found that their presence in patients with low back pain correlated strongly with the affected disc being painful on disc stimulation [3]. HIZ does not prove that the disc is definitely the source of pain, but it increases the odds that the disc is the source of pain by a factor of 6.5.

Several studies have reinvestigated this association. Although the specific statistical variables differ, the same pattern recurs (Table 4.8) [3,29–33]. HIZ do not occur in all

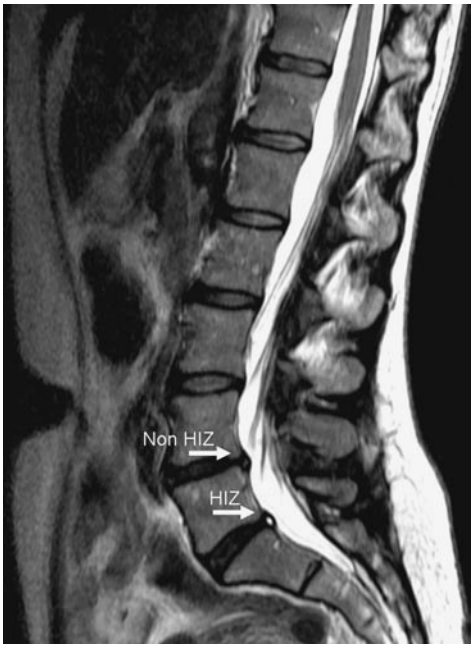

Figure 4.11 Not all spots in an annulus fibrosus constitute high-intensity zones (HIZ). Gray spots may represent a fissure in the annulus, but they are not HIZ. In HIZ, the signal intensity exceeds that of the cerebrospinal fluid.

Table 4.8 The Strength of Relationships Between a High-Intensity Zone and Discogenic Pain

Sensitivity	Specificity	Likelihood Ratio	Reference
0.71	0.89	6.5	[3]
0.52	0.90	5.2	[30]
0.27	0.95	5.4	[31]
0.78	0.74	3.0	[32]
0.31	0.90	3.1	[33]
0.09	0.93	1.3	[29]

patients. This is reflected by the low sensitivity of the sign as a predictor of pain. However, all studies, including the one detracting study [29], consistently show high specificity. That feature indicates a double negative: if present, it is very uncommon for HIZ to occur in a disc that is not painful. This results in a high positive likelihood ratio: the presence of a HIZ strongly implies that the affected disc is the source of pain. A likelihood ratio of 5 increases the likelihood that IDD is the cause of pain from a pretest probability of 0.4 to a posttest probability of 0.77. Even a likelihood ratio of 3 provides a posttest probability of 0.67.

A prospective blind study was conducted to evaluate the lumbar disc HIZ with the pain provocation response of lumbar discography. Consecutive patients with low back pain unresponsive to conservative treatment and being considered for spinal fusion were subjected to MRI followed by lumbar discography. Ninety-two HIZ were identified in 73 patients, mainly occurring at L4/5 (48%) and L5/S1 (35%). Significant correlation was found between abnormal disc morphology and the HIZ ($P < .001$). In

Table 4.9 The Prevalence of High-Intensity Zones in Samples of Asymptomatic and Symptomatic Subjects

	Asymptomatic	Symptomatic
HIZ Present	13	25
HIZ Absent	41	17
Prevalence	0.24	0.60
95% CI	0.13–0.35	0.45–0.75

Based on Carragee et al. [35].

Table 4.10 The Strength of Relationships Between High-Intensity Zone Lesions and Disc Pain

HIZ	Disc	
	Painful	Not Painful
Present	24	9
Absent	29	47

Sensitivity, 0.45; specificity, 0.84; likelihood ratio, 2.8
From the study of Carragee et al. [35].

morphologically abnormal discs graded III or IV, there was a significant correlation between the HIZ and exact or similar pain reproduction ($P < .001$). The sensitivity, specificity, and positive predictive value (PPV) for pain reproduction were 81%, 79%, and 87%, respectively [34].

Some investigators have ventured to discredit the HIZ [35]. They claimed that the sign was not diagnostic because HIZ occur in subjects without back pain. However, their data nevertheless indicate that HIZ significantly correlate with pain (Table 4.9) [35]. HIZ occur nearly three times more frequently in patients with pain than in patients with no pain. The 95% confidence intervals of the respective proportions do not overlap (Table 4.9). Furthermore, the criticism of HIZ is misdirected. The HIZ was never advocated as a sign of pain. It is a sign in patients with back pain that the affected disc is more likely to be the source of pain. In this regard, even the disparaging study provides data to this effect [35]. The sign has a high specificity and reasonable likelihood ratio (Table 4.10) [35].

Not withstanding these arguments concerning MRI, detecting HIZ does not provide for a final diagnosis. Its presence renders it more likely than not that the affected disc is the source of pain. For conservative purposes, this level of confidence may be enough. However, if target-specific therapy is to be undertaken, the putative diagnosis needs to be confirmed by disc stimulation [36].

Kang's group reported on 62 patients (aged 17–68 years) with axial low back pain who underwent lumbar discography (178 discs tested). Based on the combination of HIZ and disc contour abnormalities on MRI, four groups were differentiated: (a) normal or bulging disc without HIZ; (b) normal or bulging disc with HIZ; (c) disc protrusion without HIZ; and (d) disc protrusion with HIZ. Disc protrusion with HIZ (sensitivity, 45.5%; specificity, 97.8%; PPV, 87.0%) correlated significantly with concordant pain provocation ($P < .01$). A normal or bulging disc with HIZ

was not associated with reproduction of pain. Disc degeneration (sensitivity, 95.4%; specificity, 38.8%; PPV, 33.9%), disc protrusion (sensitivity, 68.2%; specificity, 80.6%; PPV, 53.6%), and HIZ (sensitivity, 56.8%; specificity, 83.6%; PPV, 53.2%) were not helpful in the identification of a disc with concordant pain. Thus, disc protrusion with HIZ on MRI is predictive for positive discography in patients with discogenic low back pain [37].

MRI-detected vertebral body endplate signal intensity (Modic) changes have provoked the most interest as to whether MRI can predict LDS outcome. Modic changes are reliably detected [38,39]. They are classified into subtypes: type 0, normal; type 1, nonfatty high signal intensity; type 2, fatty; and type 3, sclerosis.

Modic type I lesions occur in the spongiosa of the vertebral bodies adjacent to the affected disc. They appear dark on T1 and as a high-intensity signal on T2-weighted images. They indicated edema of the spongiosa. Type 1 changes appear to be inflammation on MRI, and although this is supported on histological and biochemical studies, ordinary fludeoxyglucose PET imaging does not reveal increased metabolism, either indicating that the process itself is low key, or that PET imaging needs refinement [40].

Modic type II lesions appear as a high-intensity signal in the spongiosa on T1- and T2-weighted images. They reflect fatty infiltration of the vertebrae. These lesions have a strong correlation with the disc being painful on stimulation (Table 4.11) [30,41].

Weishaupt reported on 50 patients with chronic low back pain who underwent MRI and provocation discography and determined that only moderate and severe type I and type II endplate abnormalities were predictive of pain generating discs with concordant pain on provocation (sensitivity, 38%; specificity, 100%; PPV, 100%) [42].

In Thompson's retrospective analysis, type 1 changes (n = 155) had a high PPV (0.81; 95% CI, 0.74–0.87) for positive LDS; type 2 changes (n = 126) had a lower PPV (0.64; 95% CI, 0.55–0.72), and type 3 changes (n = 21), with a PPV (0.57; 95% CI, 0.34–0.78) were not predictive of positive LDS. It was also noted that the PPV of a Modic type 1 endplate change for a tear in the annulus fibrosus of the disk was also insignificant (0.14; 95% CI, 0.09–0.20). A similar analysis between a type 1 endplate and the presence of a disk herniation (PPV, 0.26; 95% CI, 0.19–0.34), and between a type 1 endplate and vertebral body spondylolisthesis (PPV, 0.28; 95% CI, 0.20–0.35) were significant. It was concluded that type 1 signal intensity changes on MR images have a high PPV in the identification of a pain generator [43].

In summary, although the low sensitivity reflects the fact that not all patients with discogenic pain exhibit these features, the high specificity, however, indicates that when moderate to severe Modic type I or II are present, they are nearly always associated with a painful disc.

Table 4.11 The Strength of Relationships Between Modic I and II Changes and Discogenic Pain

Sensitivity	Specificity	Likelihood Ratio	Reference
0.23	0.97	7.7	[41]
0.22	0.95	4.4	[30]

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5 Provocation and Analgesic Discography Evaluation of Lumbosacral Internal Disc Disruption

Kevin Pauza

INTRODUCTION

Lumbar disc stimulation, even with its limitations and controversy, remains by far, the best indicator for confirming or denying discogenic symptoms.

The etiology of a person's low-back pain (LBP) is not discernible through any imaging modality, and therefore, treating a person's presumptive discogenic pain with surgery or any other means is not appropriate when the basis of treatment rests solely on the findings of magnetic resonance imaging (MRI), computed tomography (CT), or other imaging modalities. Investigations reveal that up to 85% of asymptomatic individuals possess bulging, degenerated, or herniated intervertebral discs [1].

Fortunately, lumbar disc stimulation, or provocation discography, allows physicians to better determine the etiology of lumbar symptoms, because no imaging study, including MRI, CT, or myelography, is able to accurately determine whether or not an intervertebral disc causes a person's low back or referred lower limb symptoms [2–6]. All investigations support this claim and stand without contest. The disc's appearance, no matter how pristine or degenerated, cannot allow determination as to whether or not a lumbar intervertebral disc is the source of symptoms. Indeed, intradiscal pathology and intervertebral disc disruptions without herniations or bulges play an important role in LBP generation [7]. Regarding the ability to diagnose discogenic pain, at best, limited diagnostic utility exists in the rare circumstances where MRI demonstrates morphologic endplate abnormalities in conjunction with decreased signal intensity on T1-weighted spin-echo images (Modic type 1) correlated with segmental hypermobility and LBP. However, it should be noted that these are trends, and not absolute correlates [8]. Two other investigations suggest a possible relationship between endplate abnormalities revealed by MRI and discogenic pain [9,10]. In these studies, moderate and severe type 1 and type 2 endplate abnormalities were considered abnormal, and all injected discs caused concordant pain on provocation [11], and no studies have correlated endplate pathology with MRI findings in patients with LBP.

Annular fissures are not necessarily a feature limited to aging or degenerative discs, and discogenic pain does not necessarily correlate with aging or degenerative changes. A radiographically imperceptible annular tear (annular fibrosus), in an otherwise seemingly normal intervertebral disc, may cause debilitating symptoms [12]. For this reason, lumbar disc stimulation, even with its limitations and controversy, remains by far the best indicator for confirming or denying discogenic symptoms. The value of another test, Functional Anesthetic Discography (FAD), is under investigation. This procedure involves injecting anesthetic directly into the disc through a catheter, thus selectively anesthetizing suspected disc(s) while the patient performs activities that typically generate pain. The FAD procedure allows for both functional and anesthetic assessment of these suspected discs in patients with LBP.

Consider this fact: the value of every diagnostic test relies on its ability to affect the patient's therapeutic outcome. When employing this doctrine, no invasive disc treatment is justified when the treatment is based solely on the results of radiographic studies and physical examination. Together, they do not possess sufficient sensitivity and specificity to merit a therapeutic intradiscal intervention. To date, no publication demonstrates a satisfactory disc treatment outcome correlating with radiographic observation. However, in stark contrast, several investigations performed in a randomized and blinded manner demonstrate treatment efficacy when the primary diagnostic tool of inclusion was lumbar disc stimulation [13–15].

NOMENCLATURE AND TERMINOLOGY

The correct terminology for this described procedure is "lumbar disc stimulation" because this name best connotes the fact that the most important component of this diagnostic test is the elicitation, or lack of elicitation, of concordant symptoms during the introduction of contrast medium into the intervertebral disc. The *discogram* component of the test describes the radiographic appearance of the disc's morphology as delineated by the injected contrast, as visualized through fluoroscopy and CT. The discogram also serves to confirm accurate placement of contrast within the targeted nucleus pulposus and not the outer annulus fibrosus. A postdiscogram CT is not necessary to make this determination; but instead, an anterior-posterior (AP) and lateral plain film radiograph obtained during the procedure adequately allows this determination. The term "provocation discography" defines the

composite procedure of disc stimulation and discography. "Provocative discography" is an inaccurate term mistakenly replacing the adjective *provocation* with the adjective *provocative.*

THE BIOLOGY OF INTERVERTEBRAL DISC PAIN

Lumbar intervertebral discs in humans of all ages are richly innervated. The tissue components of all motion segments, including the capsular surfaces of the zygapophysial joints and the outer aspects of intervertebral discs, are innervated with nociceptors. Specifically, within the disc, the greatest concentration of nociceptors exists within the posterior aspect of the annulus fibrosus. The second greatest concentration exists in the posterior-lateral annulus fibrosus, and least concentration of nociceptors exists in the anterior annulus fibrosus. Coincidentally, this directly correlates with the regions of the annulus fibrosus most frequently affected by annular tears or herniations.

These nerve endings originate as branches of the sinuvertebral nerves, the grey rami communicantes, and the lumbar ventral rami [16–19].

DePalma and his colleagues concluded that discogenic pain is, in varying degrees, caused by the sensitized nociceptors within annular tears [20]. Histochemical studies in human and animal material show that these nociceptors contain peptides such as calcitonin gene–related peptide, vasoactive intestinal peptide, and substance P, which are characteristic of nociceptive nerve fibers [21–23]. Inflammatory cytokines and nerve in-growth into the vertebral endplates may both play a role in mediating discogenic LBP. In one study, endplate abnormalities were shown to be related to inflammation and axon growth induced by tumor necrosis factor. Therefore, tumor necrosis factor expression and protein gene product 9.5-positive nerve in-growth in abnormal endplates may be a cause of LBP [7,8,24]. Early investigators postulated that the innervation of the disc occurs only as a result of in-growth of granulation tissue after disc injury; but this contention has been challenged and is incompatible with the observations that fetal and infant disc in humans are well innervated [18,19].

HISTORICAL BACKGROUND

The misuse of the procedure's name throughout the literature, past and present, may, in part, be related to the test's original purpose when it was introduced in the 1940s, because it was first introduced as a test to diagnose only the appearance of lumbar intervertebral disc herniations [25–27], by outlining the nucleus pulposus with contrast medium, thus demonstrating the shape of the nucleus and any disc displacement. Discography was believed to be superior to oil-contrast myelography [28–30], but other physicians believe that lumbar discography is indicated for evaluating unusual or atypical cases [31,32].

The paradigm of diagnosing LBP shifted as early as 1955 when physicians noted that their patients who demonstrated abnormal disc morphology possessed a higher likelihood of experiencing concordant symptoms when compared to their counterparts who demonstrated normal intervertebral disc morphology while injecting contrast into their discs [25,26,32–43].

For completeness sake, the commonly cited, yet notoriously erroneous investigation performed by Holt is mentioned here because it was the first investigation attempting to seriously discredit disc stimulation. That study's credibility was tarnished by concerns including: the realization that study's subjects were prisoners allowed to enroll and receive payment of cigarettes only after claiming that they had no history of LBP; the protocol's technique excluded fluoroscopy; and asymptomatic patients, by definition, are unable to claim concordant pain during the pressurization, because they possessed no chronic underlying pain on which to report [44].

CONTROVERSIES AND LIMITATIONS OF DISC STIMULATION

Pauza found the false-positive rate of disc stimulation is approximately 1.0%, after screening 4319 subjects with chronic lumbar pain. In that investigation, disc morphology was compared with concordant symptoms and the false-positive rate was defined as a morphologically normal discs which caused concordant symptoms during pressurization. Meticulously performed disc stimulation utilizing sham pressurizations was performed on each disc [45].

Some investigators, without utilizing accepted procedural guidelines, claim significantly higher false-positive rates with disc stimulation [46,47]. Critics note that these investigations neglect crucial aspects: First, an asymptomatic subject cannot report concordant pain because they normally do not suffer from LBP; Secondly, the investigator's technique failed to require sham pressurizations, as first recommended and described by the author [48]. Caragee's investigation claims disc stimulation resulted in accelerated disc degeneration, disc herniation, loss of disc height and signal and the development of reactive endplate changes compared to match-controls, and therefore careful consideration of risks and benefits should be used in recommending procedures involving disc injection [49–51]. Critics of this investigation note: a limited subject follow-up rate of 66% after 10 years; the low likelihood that completely asymptomatic individuals would volunteer to undergo disc stimulation; and the likelihood that the subject cohort following up after 10 years was skewed, because those subjects willing to follow-up after 10 years may have been the subjects suffering from LBP, thus minimizing the likelihood of matched cohort groups at 10 years [47,50–52]. Another study of asymptomatic subjects employing strict procedural techniques recommended by International Spine Intervention Society (ISIS) revealed no pain elicited with pressurization of asymptomatic individuals [53].

POST DISC STIMULATION RADIOGRAPHIC IMAGING

Although post disc stimulation CT discography is not routinely indicated, axial CT views of the disc reveal the radial

dispersal of contrast medium [54]. In abnormal discs, the contrast medium disperses outwards along radial fissures and circumferentially around the anulus fibrous. The irregular pattern seen in lateral radiographs results from the superimposition of regular, radial and circumferential tracts of contrast medium.

A unique capactiy of the discogram (referred to as the "post disc stimulation CT" or "post disc stimulation plain film radiograph") is its ability to reveal "unseen" annular tears, and may reveal the extravasation of contrast through these tears into the epidural space onto the descending spinal nerves. Although not necessarily producing symptoms during the disc stimulation portion of the test, this leakage of contrast may suggest symptoms associated with chemical radiculitis. More specifically, the contrast medium from the disc parallels the extravasation of nucleus pulposus through the annulus fibrosus onto the desceneding spinal nerves which has been shown to cause chemical radicutitis [55].

Dallas Discogram Scale

Axial CT views allow one to objectively quantify annular disruption. The degree of disruption dependes on the extent to which the annulus fibrosus is disrupted and not on the extent of disc degeneration. This led to the introduction of the Dallas discogram scale in which annular disruption is graded on a four-point scale. In the Dallas Discogram Scale, grade 0 describes contrast medium contained entirely within a normal nucleus pulposus. Grades I to III describe extension of contrast medium along radial fissures into the inner third, middle third and outer third of the anulus fibrosus respectively [56]. Later, a grade IV category was added defined by fissures extended circumferentially around the annulus fibrosus by at least 30° of arc [57] (Figure 5.1).

Subsequent investigation revealed that LBP reproduction during disc stimulation correlated with the extent of annular disruption. Grade 0 and grade I disruptions were rarely painful, but 75% of grade III disruptions were associated with exact or similar LBP reproduction; conversely, 77% of discs with exact or similar pain reproduction exhibited grade III annular disruptions. Grade II disruptions were less regularly associated with pain reproduction [11].

The distribution of disc nociceptors within the annulus fibrosus correlates with the Dallas Discogram Scale. Recall that the inner third of the disc is rarely innervated; the outer third is regularly innervated; the middle third's innervation is variable. Therefore, correlations exist between the innervation of the disc, pain reproduction during disc stimulation, and a demonstrable disc lesion.

Discogenic pain does not correlate with ageing or degenerative changes. However, LBP is strongly associated with the presence of outer annular fissures [10].

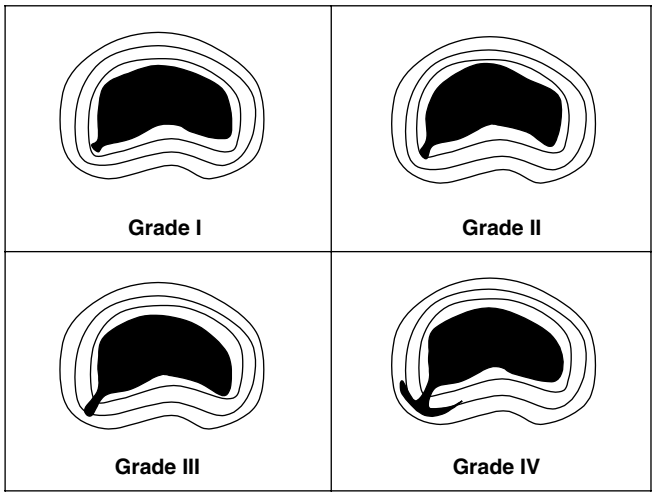

Figure 5.1 Axial views demonstrating the Dallas Discogram Scale.

INDICATIONS

This test, like any test, should only be performed if the results of this test will directly affect the direction of the therapeutic algorithm.

Lumbar disc stimulation assists in determining whether or not a lumbar intervertebral disc is a source of a pateint's LBP. Additionally, the mandatory sham pressurizations of noninjured discs allow this test to help discern whether or not a patient is feigning an illness or exaggerating symptomology.

Disc stimulation is normally not indicated in patients with acute or subacute LBP because those patients are likely to have their symptoms resolve spontaneously within 6 months. In comparison, those with chronic LBP confirmed by disc stimulation are all likely to experience some degree of pain approximately 5 years later [45]. This same investigation showed that 68% improved slightly and 24% worsened [58,59]. In another investigation, 38% of the placebo control group, all with positive provocation discography, demonstrated clinical improvement at 6 months after receiving the sham treatment [45]. Derby disputed the high rate of false positives by performing provocation discography on a small sample of historically asymptomatic subjects. Some experienced pressure sensation but none experienced concordant pain [60].

Patient Selection

Patients should have chronic LBP, defined as pain for greater than 6 months, with or without somatic referred pain in the lower limbs. More recently, because of the introduction of biologic therapies for painful discs, including fibrin sealant and other tissue restorative growth factors, one may want to evaluate the disc for its possibility of leaking nucleus pulposus.

Psychosocial Factors

Patient screening should incorporate the fact that highly abnormal psychosocial factors have been shown to

potentially increase the false-positive responses to provocation discography [48].

Patients should have been thoroughly assessed to confirm there is no other readily diagnosed cause of their pain. They should not have anatomical abnormalities that might preclude or interfere with the safe conduct of disc stimulation. They should be able to understand the requirements of disc stimulation, and be able to comply with those requirements, which include, but are not limited to, being able to tolerate the procedure, and being able to cooperate in providing responses.

When disc stimulation is undertaken as a primary investigation in patients with idiopathic LBP, the patients should be evaluated according to a disciplined algorithm. Such an algorithm ensures that disc stimulation is not undertaken arbitrarily when other possible sources of pain may be more likely than discogenic pain. Adherence to such strict operational criteria will minimize false positives and minimize psychological factors' effect on false-positive rate.

CONTRA-INDICATIONS

Absolute

- The patient is unable or unwilling to consent to the procedure.
- Inability to assess patient response to the procedure
- The patient has evidence of an untreated localized infection in the procedural field.
- Pregnancy

Relative

- Allergy to contrast medium, local anesthetic, or antibiotics
- Known bleeding diathesis
- Anticoagulants
- Anatomical derangements, congenital or surgical, that compromise the safe and successful conduct of the procedure
- The patient has known systemic infection.

FACILITIES, EQUIPMENT AND SUPPLIES

Radiological Equipment

The test area should be in a procedure room suitable for aseptic procedures. A sterile surgical suite is not necessary. The room must be equipped with fluoroscopy (C-arm or two-plane image intensifier), and a table that is x-ray compatible. The room should also be equipped with minimally invasive monitoring equipment including ECG, pulse oximeter, and blood pressure cuff. Supplemental O_2 and suction should be available.

Needles, Gowns, Drapes, etc.

- Solutions for skin preparation may be an iodine-based solution (e.g., povidone-iodine), or an alcohol-based antiseptic (e.g., chlorhexidine 0.5% in 70% alcohol).
- Drapes or sheets, to achieve a sterile field and an aseptic region. -For each disc to be stimulated, a single 90 to 150 mm (3.5–6.0 inch) spinal needle is required. 23- or 25-gauge needle is preferable for patient comfort. A slight bend on the tip allows for navigation into the L5-S1 disc around the iliac crest, thus negating the need for the more cumbersome two-needle technique.
- A 90 mm (3.5 inch), 23- or 25-gauge needle for anaesthetising the skin, but not a needle track.
- Sterile gloves, at least two pairs.
- Pressure manomenter, according to operator preference, for the injection of contrast medium, local anaesthetic, and antibiotics.
- One 20 mL syringe for mixing contrast medium and antibiotic.
- Minimal volume extension tubing.
- Intravenous cannula for the administration of antibiotics and optional sedative agents.

Medications

- Antibiotics for prophylaxis against discitis (cefazolin, clindamycin, or ciprofloxacin).
- Local anesthetic of choice for skin infiltration.
- Nonionic contrast medium with 10 mg per cc of antibiotic (cephazolin/clindamycin)
- Short acting sedatives or analgesics of choice (ie, midazolam/fentanyl) may or may not be used.

Personnel

- There should be at least one assistant available in the room to attend to the patient and to document the procedure, including the patient's response to the disc stimulation.
- It is recommended to have a second assistant, preferably a certified x-ray technologist to operate the fluoroscope.
- The staff should be in clean attire (eg, scrubs suits) and if in close contact to the sterile field, should wear surgical caps and masks.

PRELIMINARY PROCEDURES

History and Physical Examination

A history should be obtained and a physical examination performed in order to establish that the patient and their complaint are, indeed, suitable for investigation by disc stimulation, and to detect any contra-indications to disc stimulation.

Informed Consent

The patient should be informed of all the risks and benefits of the procedure. The patient should know why he is undergoing the procedure and should understand, agree to, and desire the potential future therapeutic options dictated by the results of the disc stimulation test.

The patient should be made familiar with the terms "concordant pain" and "dissimilar pain," and understand that they will asked to report their LBP during the test and which of these terms best describes any symptoms that are produced during the procedure. The patient must

also be made familiar with the use of a visual analog pain scale (VAS) and be able to report the intensity of their pain before and during the procedure.

Premedication

The patient must be given standard nothing-by-mouth (NPO) orders if IV sedative medications are given. These NPO standards are specific to the institution.

Typical prophylaxis against discitis including; cephazolin 1 g, clindamycin 900 mg, or ciprofloxacin 400 mg IV, should be administered within 15 to 60 minutes before commencing the procedure. Aminoglycosides are not endorsed for systemic prophylaxis. If the patient is allergic to penicillins, an alternative is clindamycin IV 900 mg [61].

Allergy

If the patient has a known allergy to contrast medium, they should be pretreated with H1 and H2 blockers and corticosteroids prior to the procedure. Another option is to utilize gadolinium in those patients with a known allergy. Patient's ability to tolerate anxiety associated with any invasive test, especially disc stimulation, is variable. Because of this, sedative agents may be administered as the procedure commences, and again during the procedure, if required. However, the patient's response to disc pressurization is critical to the validity of the test. Therefore, careful titration of sedative and opioid medication is essential. The patient should be awake during the pressurization portion of the test.

TECHNIQUE

Preparation

Positioning

The patient lies on a radiography table either in a prone position or in an oblique position with the target side up, depending on patient comfort and operator preference.

Sterility

The skin of the lumbar region and upper gluteal region is prepared as for an aseptic procedure. The operator and any personnel who may come towards close contact with the prepared area should wear clean attire (e.g., scrubs suits). Surgical caps and masks are suggested but not mandatory.

If the operator scrubs the skin, he should don fresh gloves after the back has been prepared, and before inserting any needles.

To help minimize the chance of bacterial contamination to the needle and/or the disc the needle tip should not be touched by the gloved hand nor should any needle be unnecessarily exposed to the atmosphere. Upon being withdrawn from its scabbard it should be used promptly. A sterile instrument should be utilized when manipulating the needle tip.

Selecting Appropriate Disc Levels to Test

Both the International Association for the Study of Pain (IASP) [62] and the ISIS [63] have recommend that in order to be valid, provocation discography must be subjected to anatomical controls. Specifically, the diagnostic criteria for discogenic pain are:

1. Provocation of the target disc reproduces the patient's LBP;
2. AND that provocation of adjacent discs does not reproduce pain.

The most likely disc and two most adjacent discs should be studied, if possible. Investigation should commence with the disc least likely to be symptomatic, and progress to the disc most likely to be symptomatic. The patient should at all times remain blind to the level being investigated, and to the onset of stimulation. It is appropriate and thorough to always attempt to identify an asymptomatic and normal disc adjacent to the symptomatic disc.

Target Identification

An AP image of the lumbar spine is obtained and the target disc is identified. The fluoroscope is angled to the head or feet so as to obtain a view of the target disc such that the x-ray beam passes parallel to the ring apophysis or subchondral plate of the inferior vertebral endplate of the disc.

To minimize the likelihood of attributing unintended "iatrogenic spinal nerve needle procedural pain" with disc stimulation pain, the target disc should be approached from the side opposite to the side on which the patient experiences their predominant pain. If the patient's pain is central or bilateral, the disc may be approached from either side according to operator preference.

Once the side from which the disc will be approached has been selected, the beam of the fluoroscope is rotated laterally to an oblique position, thus allowing visualization of the target disc from its posterolateral aspect on the side selected. The beam should be angled until the lateral aspect of the superior articular process of the target segment lies opposite the axial division of the anterior two thirds and posterior one third of the target disc. This view allows needles to be advanced parallel to the beam, directing the needle tip intentionally to the center of the nucleus pulposus as it passed across the superior articular process. This view positioning the S.A.P. at the 2/3–1/3 demarcation of the disc intentionally directs the tip to the center of the nucleus pulposus. The target point for puncture of the annulus fibrosus lies on the transverse midline ("equator") of the target disc, just lateral to the lateral margin of the superior articular process (Figure 5.2).

At the L5-S1 level, the iliac crest may overlie the target disc in oblique views. Care should be taken to obtain a view such that the target point lies between the superior articular process of S1 medially, and the iliac crest laterally (Figure 5.3). If the iliac crest lies too far medially and precludes such a view, the target disc will need to be approach using a curved, two-needle technique. In the oblique view, a puncture point on the skin is selected

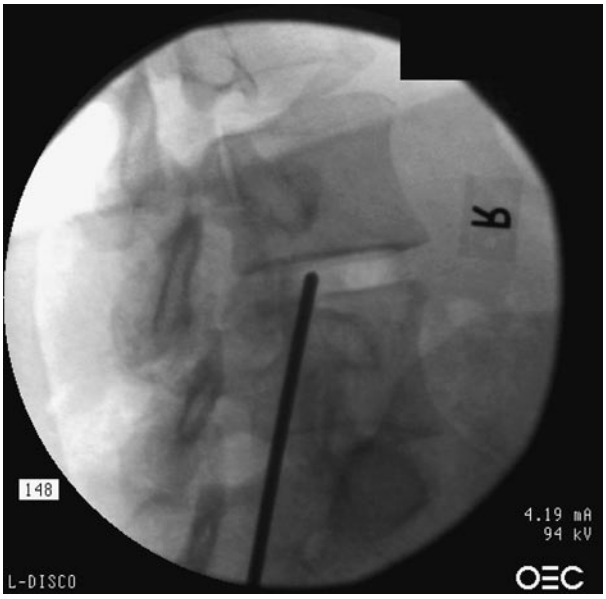

Figure 5.2 A right oblique view of an L4-5 intervertebral disc space, over which a metal probe has been placed to indicate the target point for discography. Courtesy of Kevin Pauza, MD.

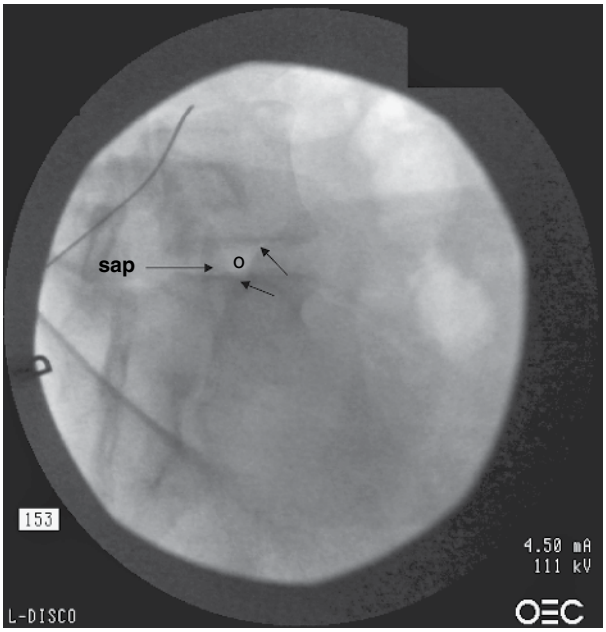

Figure 5.3 A right oblique view of an L5-S1 intervertebral disc space, showing the target point for discography (marked with a circle), between the S1 superior articular process (sap) and the iliac crest (arrows). Courtesy of Kevin Pauza, MD.

directly overlying the target point. A skin wheal is raised with local anesthesia (lidocaine 1 or 2%) using a 25- to 30-gauge skin needle.

Needle Placement

For each disc that is to be studied fresh needles must be used. The skin overlying the target disc is marked and anesthesized with the operator's choice of local anesthetic such as 1.5% lidocaine. The needle tract itself should not be anesthetized in order to avoid deposition of any local anesthetic along the vital descending spinal nerves. It is important for the patient to be able to warn the operator if the needle is mistakenly approaching or touching the spinal nerve, to avoid causing an iatrogenic radiculopathy. There are no nociceptors along the path from the skin to the disc and therefore the patient should not experience discomfort as the needle travels towards its target disc. For access to the disc, either a single needle or a two-needle technique may be used. (The two-needle technique was advocated in response to reports of discitis occurring as a result of discography, but single needle techniques have proved adequate and safe since the use of prophylactic antibiotics. There is no advantage to using a two-needle technique with respect to discitis.)

If the operator employs a two-needle technique to reach the target disc, a shorter, large gauge spinal needle provides preliminary access toward the disc, through which a smaller gauge, longer needle is used to enter the disc. In patients of average build, the introducer needle would typically be a 90 mm 18- or 22-gauge spinal needle, and the complementary needle would be a 150 mm 22- or 25-gauge spinal needle. Larger patients may require needles that are 150 mm and 200 mm in length respectively.

The introducer needle is advanced carefully through the puncture point, down the x-ray beam, towards the target point on the disc (Figure 5.4). Its passage is terminated opposite the depth of the inferior lateral border of the superior articular process, where it should aim at the target point on the disc. Its stylette is then removed and the longer, thinner needle is then advanced through the introducer needle as far as the depth of the superior articular process. It is then advanced slowly towards the surface of the target disc.

During this latter phase of introduction, care is taken not to impale the ventral ramus, which crosses the posterolateral quadrant of the disc. If the patient complains of paraesthesia or radicular pain, insertion of the needle should be stopped immediately, and the needle withdrawn slightly because it is likely approaching a descending spinal nerve from a more cephalad spinal segmental level. Its intended course should be adjusted, so as to avoid the point at which the ventral ramus was encountered, and the needle reinserted towards the disc very slowly while avoiding re-contact with the ventral ramus. If necessary to avoid contact with the nerve, if an introducer needle is being used, it may be redirected slightly.

Once the ventral ramus is avoided, the penetrating needle will encounter the annulus, which presents as a sensation of firm, almost "rubbery," resistance to the passage of the needle. Once the annulus is contacted, the needle is pushed through the annulus, and should be advanced to the center of the disc. Its progress into the disc should be monitored by alternating between both AP and lateral projections.

If a single needle technique is used, that needle is advanced towards the target disc in the same manner, as

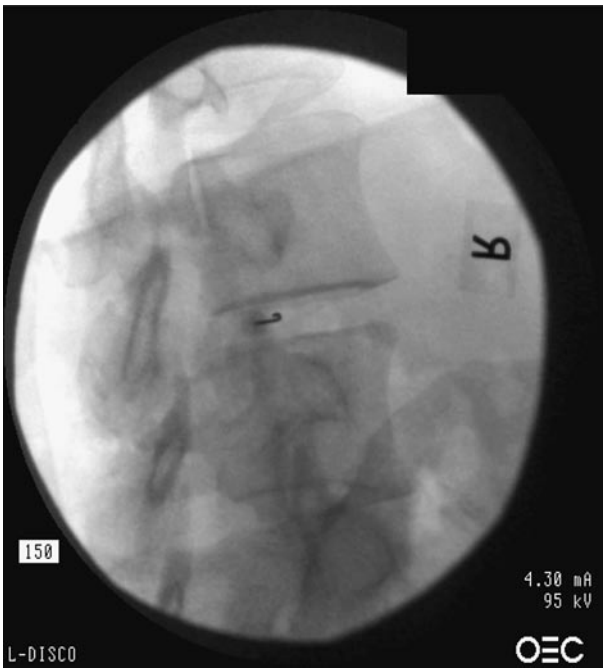

Figure 5.4 An oblique view of an L4-5 disc towards which a needle has been inserted. Courtesy of Kevin Pauza, MD.

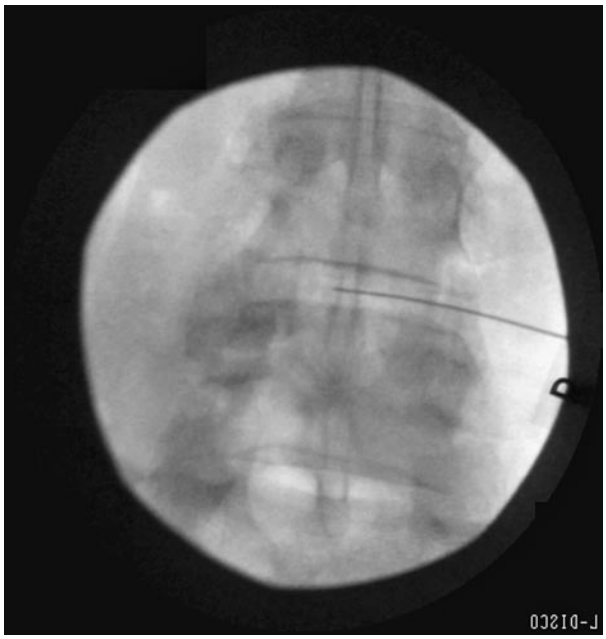

Figure 5.5 An AP view of a needle correctly placed at the center of an L4-5 disc. Courtesy of Kevin Pauza, MD.

the introducer needle when the two-needle technique was utilized. Upon reaching the depth of the superior articular process, the needle is advanced carefully across the intervertebral foramen, taking care not to pierce the ventral ramus. The same precautions are taken as when the penetrating needle is passed across this region, when the two-needle technique is used. Subsequently, the needle is advanced through the annulus fibrosus into the center of the disc. Its progress into the disc should be monitored first on AP projection, and checked with a lateral view (Figures 5.5 and 5.6)

If the needle tip is in the middle of the disc on the AP view but anterior on the lateral view, the needle entered the disc too laterally. If the needle tip is centered on the AP view but posterior on the lateral image, the needle entered the disc too medially. In either event, the location of the superior articular process should be checked on the oblique view, to ensure that the process did lie opposite the center of the disc. If upon checking this proves not to be the case, the correct view should be obtained, and the introducer needle adjusted or reinserted so that it correctly points to the center of the disc.

If the oblique view is correct but the penetrating needle has nevertheless strayed form the center of the disc, it may have deflected during its passage through the disc. In that event, the direction of deflection should be noted. The penetrating needle should be withdrawn completely. A slight bend should be made to the tip of the needle.

The penetrating needle can now be reinserted through the introducer needle and passed into the disc, using the bend to navigate it away from the previous direction of deflection, and accurately towards the center of the disc. Once the needle is correctly positioned in the center of the disc, its stylette is removed and the needle is connected to a closed system including a pressure transducer manometer, allowing the injection of nonionic contrast with antibiotic.

The contrast medium is injected slowly and the *opening pressure* is recorded. The opening pressure represents the pressure at which contrast first enters the disc and correlates with the disc's integrity. A low opening pressure reflects annular tears and an abnormally high opening

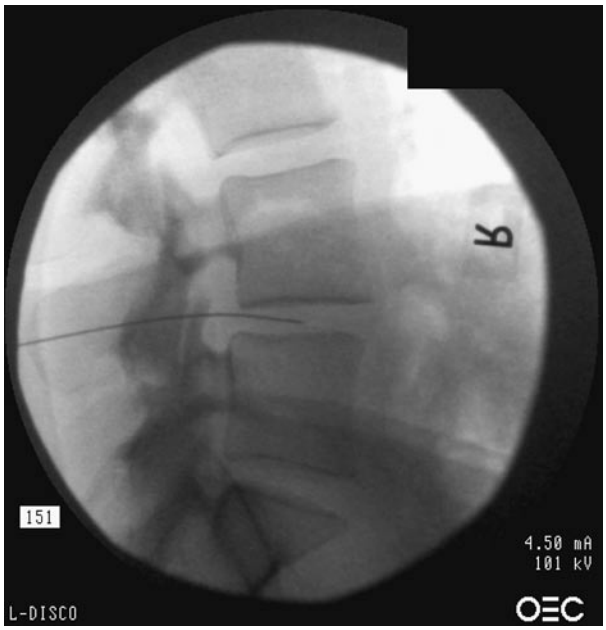

Figure 5.6 A lateral view of a needle correctly placed at the center of an L4-5 disc. Courtesy of Kevin Pauza, MD.

pressure suggests that the needle tip may be incorrectly positioned in the annulus fibrosus instead of a correct position within the nucleus fibrosus.

Injection should continue until:

- LBP is produced;
- contrast medium escapes from the disc; or
- pressure reaches 100 psi

If LBP is provoked, the pressure and volume of injection is documented. The patient should report if the pain produced is concordant with their accustomed pain, and rate its severity.

The injection can be repeated a short period of time later in order to reaffirm the previous response. Throughout the pressurizations the operator should perform sham pressurizations. During a sham pressurization, the operator tells the patient that the disc is being pressurized and asks the patient to report symptoms in a manner similar to the true pressurizations. Thus, the patient is unable to differentiate between a true pressurization and a sham pressurization. If a patient consistently claims symptom reproduction during the sham pressurizations than this suggests that the patient may be feigning symptoms, or exaggerating symptoms, or not understanding the operator's instructions.

A local anaesthetic agent can be injected into the disc, either or both as a measure to relieve any pain produced by the previous injection, or as an attempt temporarily to relieve the patient's accustomed pain. Intradiscal anaesthesia also putatively reduces the chance of pain provocation from a previously painful disc upon stimulation of an adjacent disc.

Records

Copies should be made of the discographic images, in AP and lateral views of all discs stimulated (Figures 5.7 and 5.8).

Postprocedural Care

After needles are removed, and puncture points sterilely dressed, the patient recovers with cardiopulmonary monitoring for a minimum of approximately 30 minutes. Short acting analgesics may be provided at this time. Patients are instructed not to drive on the day of their procedure, and to expect an increase in discomfort for a few days. Prescriptions for pain medication to cover this period of increased discomfort may be provided.

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, however, is extremely low (<0.1% per disc) since the introduction of prophylactic antibiotics.

DISC STIMULATION PAIN AND PRESSURE RECORDING

It is important to recognize a few misconceptions regarding the literature discussing disc pressure and accept the fact that these misconceptions are based on anecdote persisting as unsubstantiated dogma.

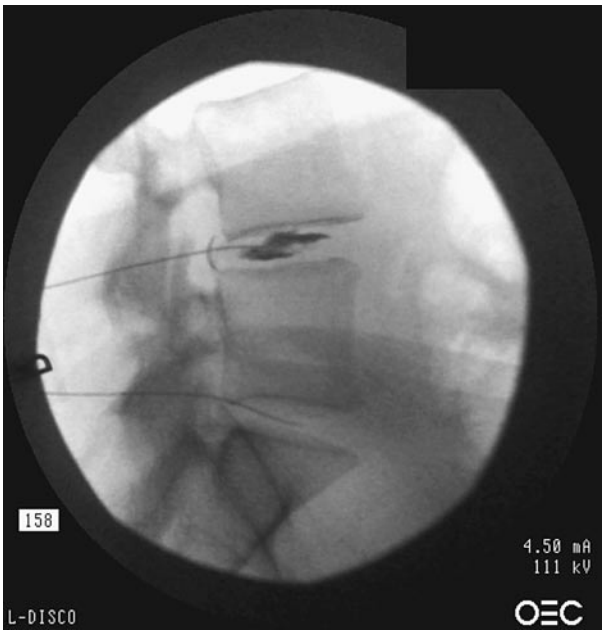

Figure 5.7 A lateral view of an L4-5 disc into which contrast medium has been injected. (A needle has also been placed into the L5-S1 disc, in preparation for injection of contrast medium.). Courtesy of Kevin Pauza, MD.

Historically, it has been claimed that discs eliciting pain upon stimulation with a low pressure were *chemically sensitive* discs. To date, no investigation exists allowing one to surmise that a "low-pressure positive" disc is a chemically sensitive disc: The chemical activity with respect to

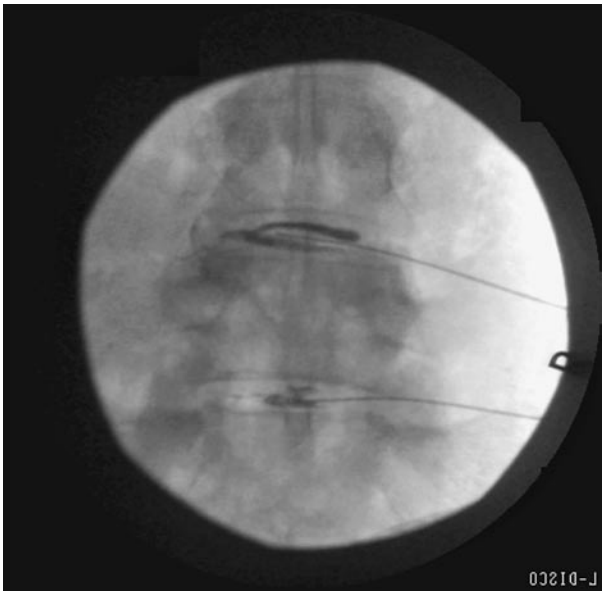

Figure 5.8 An AP view of an L4-5 disc into which contrast medium has been injected. (A small amount of contrast medium has also been injected into the L5-S1 disc.). Courtesy of Kevin Pauza, MD.

pressure and pain has not been correlated. It can only be claimed that a disc which elicits concordant pain at a low pressure is simply a "low-pressure positive" disc.

Additionally, obtaining pressure readings is recommended in an attempt to standardize the technique. The reader should be aware that guidelines stating that the pressure at which pain is produced by the disc allows the operator to define a result as; positive, negative, or indeterminate. The reader should also be aware that these pressure levels may serve as a helpful "starting point," but that they were arbitrarily chosen based on anecdote and not based on scientific merit, nor correlated with outcomes obtained following a therapeutic intervention [64].

It is espoused by some that a "highly positive" disc is one in which pain is elicited at a low pressure and a "mildly positive" disc is one in which pain is elicited at a higher pressure. One investigation demonstrated a correlation between pain produced during provocation discography with surgical and nonsurgical outcomes [65]. This claim has not been replicated by others. Some would contend that a "high pressure positive" disc is more likely to respond to a treatment intervention than is a "low-pressure positive" disc. This was evident in one randomized controlled trial [45]. Consider that maybe a "low-pressure positive" disc possesses such a low pain threshold among its nociceptors that nothing will help this disc. Likewise, consider the possibility that a "high pressure positive" disc may be *"so close to the threshold of no pain"* that it will more readily improve with a specific intervention, especially if that intervention is of a biological type.

The patient's response to disc stimulation should evaluated in a systematic manner and it is necessary to perform sham pressurizations to minimize the possibility of false-positive responses. All pressure, volume, and morphological data pertaining to the patient's responses should be recorded objectively at the time of the procedure, without bias and without coaching the patient. These data pertain to:

- if LBP was produced
- if that pain was concordant or not
- the VAS score of that pain
- the pressure at which the pain was produced

The Table 5.1 represents objective findings regarding disc pressure and volumes.

FUNCTIONAL ANESTHETIC DISCOGRAPHY

FAD involves injecting anaesthetic directly into the disc through an indwelling catheter, thus anesthetizing suspected disc(s) while the patient performs activities that typically generate pain. The FAD procedure allows for both functional and anesthetic assessment of discs in patients with suspected internal disc disruption causing symptoms. Several case studies suggest diagnostic utility, however none in peer reviewed journals. In one study, 32 patients with chronic LBP underwent standard provocation discography and FAD, and the results of lumbar fusion were compared in both groups. Fifty percent had confirmatory findings on the FAD test. Thirty-eight percent had positive provocation discograms that were negative on FAD testing. One patient had a negative provocation discogram, and yet pain relief on the FAD. Twelve patients from the study have undergone fusion and have been followed for at least 3 months (3–12 months). The mean preoperative Oswestry score was 58.5; mean post-op was 26.5. The mean pre-op VAS score for back pain was 7.2; mean post-op was 3.1 [66]. The diagnostic utility of FAD has yet to be proven. However, the role of intradiscal injection of local anesthetic in predicting surgical outcomes has gained some traction [67].

PROVOCATION DISCOGRAPHY AND FAD COMPLICATIONS

The complications of disc stimulation are categorized into three classes: (a) reactions;(b) infections; (c) and technique related.

Reactions

They include vasovagal reactions with vomiting and possible aspiration, paravertebral muscle pain, and contusion, allergic reactions to drugs (i.e., contrast medium, antibiotics, and local anesthetics); and cellulitis at the IV site.

Infectious Discitis

The overall rate of infectious discitis ranges from 0.1% to 2.3% per patient and 0.05% to 1.3% per disc. The

Table 5.1 Pressure and Contrast Volume Correlated with Disc Architecture and Pain Provocation for 309 Consecutive Patients Presenting to the Texas Spine and Joint Hospital 2004–05

Number of Discs		Disc Architecture	Pain Provocation	Mean Opening Pressure (psi)	Mean Maximum Pressure (psi)	Mean Contrast Volume (mL)
373	31.9%	Normal	None	26.0	111.9	1.44
23	2.0%	Normal	Disconcordant	22.7	90.2	1.32
21	1.8%	Normal	Concordant	19.8	85.5	1.43
289	24.7%	Abnormal	None	19.7	110.1	1.95
58	5.0%	Abnormal	Disconcordant	17.4	77.9	1.77
393	33.6%	Abnormal	Concordant	17.4	66.3	1.55
1170	100%					

From ref. [15].

common causative organisms include *Staphylococcus aureus*, *Staphylococcus epidermidis*, and *Escherichia coli* suggesting inoculation with surface organisms or misadventure through bowel perforation [68]. The incidence of discitis has been reported to be lower with double needle techniques and use of preprocedural antibiotics [64,69].

Technique

Complications related to needle misplacement include: penetration of the ventral ramus over the posterolateral quadrant of the target disc. This potential complication is avoided by careful attention to the patient's behavior as the needle is advanced towards the annulus fibrosus. Other reported injuries are anatomically impossible if correct techniques are employed, for the structures in question lie outside the procedural zone, and can only be encountered if grossly inappropriate techniques are used. The purported hazards include nerve root penetration, dural puncture with resultant postdural puncture headache, and bowel perforation.

SUMMARY

All of the information available supports the claim that Lumbar disc stimulation, even with its limitations and controversy, remains by far, the best indicator for confirming or refuting that an intervertebral disc(s) is the source of a patient's LBP.

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6 Epidural Steroids for Lumbosacral Internal Disc Disruption

Omar El Abd and Michael J. DePalma

INTRODUCTION

Extensive investigation since Dillane et al.'s [1] initial posit has demonstrated that the intervertebral disc is a common source of adult low back pain (LBP). Treatment options for adult LBP, therefore, should ideally address the pathophysiology of the injured and painful intervertebral disc. Similarly, the optimal anatomical level must be selected for a technique approach to properly address the most likely involved disc level(s) (see Chapters 1 and 3). Circumstantial evidence has well established an increased production of proinflammatory mediators and cytokines due to disc herniation [2–7] with more abundant inflammatory markers associated with extruded and sequestered discs. Painful degenerative lumbar discs in the absence of herniation produce higher levels of interleukin (IL)-6, IL-8, and prostaglandin E2 (PGE2) than their herniated counterparts causing primarily radicular leg pain [8]. Inflammatory cytokines are elevated in both annular and nuclear tissue of painfully degenerative discs [9]. Cyclical mechanical loading coupled with inflammatory stimuli increase PGE2 production by both nuclear and annular cells in vitro with the latter showing a stronger reactivity than the former [10]. Painful degenerative lumbar intervertebral discs have higher concentrations of sensory fibers, located in the endplate and nucleus, than nonpainful discs [11,12], and both IL-8 and PGE2 induce hyperalgesia [13]. The combination of the abundant innervation of the disc and increased production of proinflammatory mediators suggests that the mechanism for discogenic pain may involve hyperalgesia [8].

Hence, the instillation of corticosteroids into the anterior epidural space to maximally bathe the posterolateral and the posterior periphery of the annulus seems appropriate and logical. Such treatment may help curtail the biochemical stimulation of the intervertebral disc, thereby reducing pain and improving function; thus allowing the patient to tentatively participate in a comprehensive physical therapy program addressing biomechanical deficiencies following this reduction of hyperalgesia. Two basic tenets for this approach to be successful are that the target disc has been accurately deemed to be the source of pain and that the appropriate therapeutic medication deposited into the anterior epidural space must gain access to sensitized nerve endings. The only reason to perform lumbosacral epidural steroid injection (LESI) when treating LBP is to treat discogenic LBP as these injections are not intended to provide an effective treatment of other causes of axial LBP such as facet joint arthropathy and sacroiliac joint syndrome.

INJECTION ROUTES

Three routes may be used to introduce therapeutic agents into the lumbosacral epidural space: caudal epidural steroid injection (CESI); interlaminar epidural steroid injection (ILESI); and transforaminal epidural steroid injection (TFESI).

Interlaminar and Caudal Epidural Injections

Interlaminar and caudal epidural injections are the traditional and most frequently used spine therapeutic intervention for the management of axial discogenic pain and radicular pain. These injections can be administered using fluoroscopic guidance or blindly using anatomical landmarks.

The caudal approach was first described in 1901 by a French radiologist who injected diluted solutions of cocaine through the sacral hiatus to treat intractable LBP and/or sciatica [14]. It was not until 1957, when Cappio investigated the therapeutic benefit of injecting corticosteroids into the epidural space via the caudal approach [15].

The interlaminar technique was first described by Pages in Spain in 1921 [16], followed by Dogliotti in Italy in 1933 [17]. The therapeutic benefits of ILESI were reported about 20 years later [18,19]. In 1952, Robechhi and Capra [20] employed this approach to successfully treat lumbar and sciatic pain. Currently, there is debate among the interventionalists regarding the use of interlaminar and caudal methods versus the use of the transforaminal route. Generally, interlaminar and caudal epidural injections are considered nontarget specific when compared with transforaminal injections. If these injections are administered blindly, there is a significant rate of needle misplacement. The therapeutic agent has a high likelihood of not reaching the anterior epidural space [21–24], despite technical procedural experience [25].

The caudal injection is performed by placing a spinal needle into the sacral epidural space by way of the sacral

hiatus (this technique is described in Chapter 23). Relative ease of performance in thin individuals without requiring fluoroscopy is an advantage of this approach. The sacral epidural space must be filled before the injectated medication in order to reach the lumbar region, requiring large volumes that necessarily dilute the steroid mixture. Consequently, CESI rarely reach the ventral epidural space or pass cephalad to the L5-S1 segmental level [26]. In addition, our clinical experience demonstrated that during the caudal instillation of medicine there is considerable discomfort, which is not perceived with the other routes of administration.

The lumbar interlaminar method offers the potential advantage of delivering medication directly into the lumbar region, that is, closer to the putatively painful structure, but it is technically more demanding. Interlaminar epidural injections are performed blindly or with fluoroscopic guidance. The injectated agent is deposited into the posterior epidural space without a guarantee that it will flow anteriorly [27]. Traditionally, these procedures have been performed by practitioners skilled in using surface landmarks for needle placement. In a prospective study that included 316 patients undergoing blind epidural injections, needle positions were evaluated using fluoroscopy. Renfrew et al. report that, even in experienced hands, blind placement of the injection needle was optimal in only 60% of cases. They recommend fluoroscopic control and contrast administration to ensure correct needle placement and avoid inadvertent venous injections [25].

The addition of fluoroscopy and contrast enhancement allowed the visualization of whether or not the medication reached the potential pain generator, maximizing the chance of therapeutic benefit; however, this does not guarantee reaching the ventral epidural space. In fact, it was found that ILESIs achieve ventral epidural contrast spread in just 36% of attempts [27]. In a retrospective study of 75 patients, Manchikanti et al. [28] compared pain relief after blind interlaminar epidural injections, caudal epidural injections, and transforaminal epidural injections. The response was the most favorable to transforaminal injections, followed by the caudal injections, which surpassed the outcome of the blind interlaminar injections.

Overall, the literature shows better outcomes in acute rather than in chronic pain with longer duration of improvement of radicular pain. ILESI and CESI are falling in popularity amongst interventionalists while TFESI is gaining popularity due to more favorable research outcomes. The injections are used for treatment of lumbar discogenic axial pain and radicular pain that fails conservative management.

Transforaminal Epidural Injections

The transforaminal approach can only be performed using fluoroscopic guidance. This injection approach aims for the disc and spinal nerve interface. This is performed through the introduction of the needle into a triangular space within the anterior–superior third of the neural foramen bounded by the pedicle superiorly, the exiting nerve inferior medially, and the lateral margin of the neural foramen laterally "the safe triangle" (Figures 6.1, 6.2, 6.3). Once the

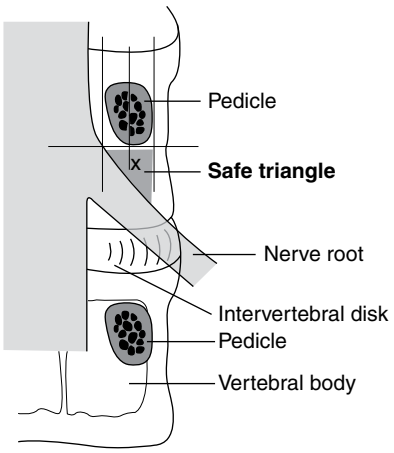

Figure 6.1 The safe triangle. From Omar El Abd. Spinal pathology: Nonsurgical intervention. Adapted from Magee et al., ed. Pathology and Intervention in Musculoskeletal Rehabilitation. Elsevier 2009.

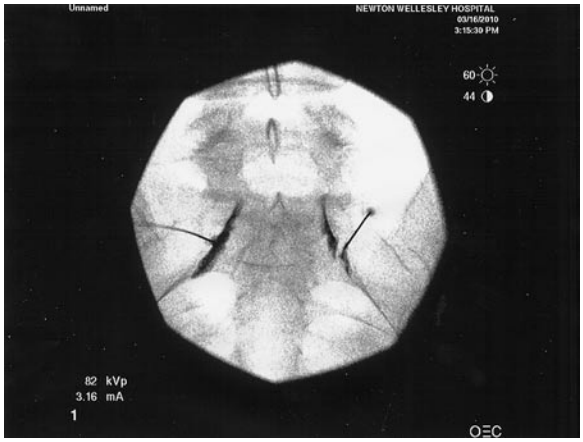

Figure 6.2 Fluoroscopic Guided Bilateral S1 Transforaminal Epidural Steroid Injections with cephalad epidural flow into the direction to the L5-S1 disc on both sides.

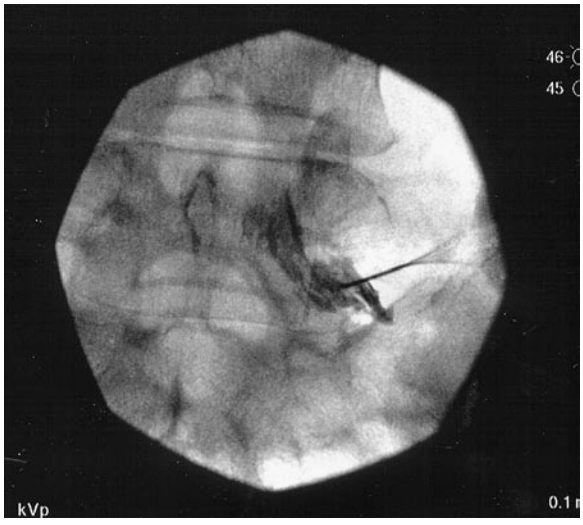

Figure 6.3 Fluoroscopic Guided Right L5 transforaminal Epidural Steroid Injection with cephalad epidural flow into the direction to the L4-5 disc on the right side.

needle is in position, efficient injection of the medicine into the lateral epidural space or around the emerging nerve root depending on needle position and bevel orientation becomes possible.

The administration of steroids at the level of the pathology is crucial to achieve effectiveness. With experienced hands, the transforaminal approach is safe and provides good outcomes. TESI is currently becoming more favorable than the interlaminar approach because it is more effective to administer the medicines at the spinal nerve/disc interface in the lateral epidural space rather than in the dorsal epidural space, which is separated from the lateral epidural space by the ligamentum flavum.

TFESI were found to achieve ventral flow in 100% of injections [29]. Furthermore, vascular evacuation of the therapeutic medication occurs in 11% of CESI and ILESI, and in 2% of TFESI [30,31] preventing the therapeutic agent from reaching its target. Therefore, the instillation of therapeutic doses of corticosteroid into the anterior epidural space, hence maximally reaching the targeted intervertebral disc, is best accomplished by the transforaminal approach rather than the interlaminar or caudal techniques. The effectiveness of TFESI has been the subject of multiple studies reviewed in this chapter.

Huston et al. [32] prospectively studied the side effects and complications of this approach. An analysis of 350 consecutive cervical and lumbar transforaminal injections identified no instance in which dural punctures occurred. Lutz et al. [33] found no epidural punctures or other major complications in 50 patients who underwent lumbar transforaminal epidural injections. Botwin et al. [34] reviewed complications in 322 transforaminal lumbar epidural injections done on 207 patients. They reported the complete absence of post-dural puncture headache. The most common complication found in their study was headaches occurring in 3.1% of patients. These headaches were transient and resolved after 24 hours. These patients epidurograms were reviewed and there was no intrathecal pattern noted.

TFESIs are not without associated risks. Spinal cord injury is reported after steroid injections using that route in the lumbar spine [35–38]. This is postulated to be secondary to the occlusion of the anterior spinal artery from an injury or an injection of particulate steroids involving an aberrant artery of Adamkiewicz or a feeder artery in the neural foramen resulting in a spinal cord infarction. This is largely due to the use of steroid preparations formed of large particulate granules that can occlude the anterior spinal artery especially if administered without a meticulous procedural technique. Therefore, these procedures ought to be performed by a medical doctor well trained in safe and competent performance of these procedures. Complications can arise requiring administration of rescue medications mandating that the treating physician be trained in life support. Fluoroscopically guided lumbosacral TFESIs are best taught and learned during a rigorous, comprehensive interventional spine or interventional pain, one-year fellowship. Weekend cadaver workshops may be useful for physicians in residency training to help them decide whether or not they wish to practice interventional spine care and thus pursue additional fellowship training. This is in contrast to CESI and ILESI that have traditionally been taught to physicians by fellow physicians trained to do them or at hands-on workshops.

These procedures can be performed in an adequately equipped office setting, ambulatory surgery center, or hospital-based surgery center. Ready access must be maintained to intravenous fluids, cardiac and pulse oximetry monitoring, and a code cart. ILESI and CESI can be performed without fluoroscopic guidance requiring fewer staff and support personnel. Hence, these blind injections can be readily offered in the office setting or either ambulatory or hospital surgery centers. Minimally invasive, percutaneous LESI categorically could be available in virtually any patient care setting. However, TFESI require specialized equipment somewhat restricting access to patients with access to such centers. Recently, it was recommended to use fluoroscopy equipped digital subtraction capability to properly identify vascular uptake while performing TFESI [39].

MECHANISM OF ACTION

The instillation of corticosteroid and anesthetic into the anterior epidural space introduces therapeutic agents with potent antiinflammatory properties adjacent to suspected painful intervertebral discs. Local anesthetics help curtail inflammation by inhibiting phagocytosis, decreasing phagocytic oxygen consumption, reducing polymorphonuclear leukocyte lysosomal enzyme release, and diminishing superoxide anion production [40–44]. Additionally, anesthetics improve neural blood flow and dysfunction [45,46]. Corticosteroids are well known for their antiinflammatory properties [47], and also stabilize neural membranes, suppress ectopic neural discharges [48], and may have direct anesthetic effect on small unmyelinated nociceptive C-fibers [49,50]. Painful lumbar intervertebral discs are innervated by substance-P containing nerve fibers [11,51], unmyelinated C-fibers, and thinly myelinated A delta fibers [12] that provide a substrate on which corticosteroids and local anesthetics exert therapeutic benefit. The nucleus pulposus of the lumbar intervertebral disc is biologically active responding to proinflammatory cytokines most sensitively after becoming degenerate [52], and once painful produces further proinflammatory mediators [8]. Hence, corticosteroids and local anesthetics may exert a therapeutic benefit by bathing the posterolateral annular fibers, which are most prone to injury [53–55], in antiinflammatory and neural stabilizing effects

DIAGNOSTIC WORK-UP

Deposition of corticosteroid into the epidural space would not be appropriate in the setting of spinal infection, malignancy, or acute fracture. Chronic spinal fractures may not represent a contraindication to LESI if the pain is likely discogenic in origin and the fracture is remote. Therefore, plain film radiography is the minimum diagnostic study required prior to epidural steroid injection. Advanced imaging studies such as magnetic resonance imaging or computed axial tomography will further define suspicious

abnormalities detected by plain films or suggested by the clinical presentation. Loss of disc height or decreased nuclear T2-weighted signal on magnetic resonance imaging is suggested to be predictive of outer annular tears; the majority of which are symptomatic [56]. High intensity zone lesions, a localized peripheral area of increased T2-weighted signal, may be a marker of symptomatic annular disruption [48,57,58]. Modic changes appear to be a relatively specific, but insensitive sign of painful lumbar discs [59,60]. Approximately 12% to 15% of normal appearing lumbar intervertebral discs may actually contain a painful annular tear [48]. Provocation discography, when performed adhering to strict operation criteria (see Chapter 5), possesses a low false-positive rate [61] and can be utilized to reveal painful annular fissures. Extension of dye into the outer annulus or beyond, and not the severity of disc degeneration, has demonstrated to be a strong predictor of concordant pain on discography [62]. An appropriate diagnostic evaluation of persistent lumbar pain would include plain films to assess alignment, disc height, and stability. MRI helps to guide the interventionalist target the appropriate disc(s) level(s). Persistent symptoms, lasting 6 months, recalcitrant to exhaustive conservative treatment measures, warrant lumbar provocative discography to better delineate the segmental level of pain generation and whether or not a corroborative outer annular tear exists accounting for persistent lumbar pain. Findings of concordantly painful outer annular disruption on discography suggest where to target TFESI to maximally affect the patient's symptomatology. However, such a strategy was not critically evaluated.

INDICATIONS

The primary indication for CESI and LESI is radicular pain. Despite minimal work having been completed investigating the efficacy of these interventions solely for axial lumbar spine pain [63], such injections are offered to patients presenting with chronic, nonradicular LBP. Therefore, the role of these injections to treat LBP has not been well-defined and is currently supported largely by conjecture and logic. Nonetheless, discogenic pain is a common source of LBP [64] and inclusion of LESI or CESI to target a discogenic source of pain is logical. Yet, the exact technique by which to do so has not been validated. The most direct way to deposit corticosteroid adjacent to a putatively painful disc is via the transforaminal approach. Therefore, a reasonable indication for lumbar TFESI is persistent LBP most consistent with a discogenic etiology. Deciding which level to inject is influenced by imaging findings and pain referral zones, but it is more commonly determined by considering the levels statistically most responsible for discogenic LBP [64]. The lowest two disc levels, L4-5 and L5-S1, are most commonly responsible for pain generation from one of the five lumbar discs [60,64]. The implication is that LESI should initially target these levels in the presence of MRI discogenic findings at these levels. Persistent symptoms, and subsequently more targeted lumbar LESIs at the disc level causing pain, would be evaluated by lumbar discography.

EFFICACY RESEARCH

Summary Statements

In 1994, a report was published by the Australian Working Party of the National Health and Medical Research Council (NHMRC) summarizing recommendations for epidural use of steroids in the management of lumbar pain. This summary statement referenced a body of evidence endorsing lumbar TLESIs and CESIs as viable treatment options for radicular pain. However, the report cited a minimal body of literature evaluating the use of epidural steroid injections for treatment of any form of nonradicular spinal pain [63].

A year later, Watts and Silagy [65] provided quantitative evidence for meta-analysis from 11 randomized studies involving a total of 907 patients that underwent TLESIs for radicular, but not lumbar pain that epidural administration of corticosteroids is effective in the management of lumbosacral radicular pain. They also reported no long-term adverse outcomes.

In 1999, Koes et al. [66] performed a systematic review of twelve randomized studies of ILESIs in patients with lumbar pain and/or sciatica. The benefits of epidural steroid injections, if any, were of short duration only. The efficacy of ILESIs was not established in this study. The most heavily weighted research study criteria identified by Koes et al. were large study groups, description of intervention, use of relevant outcome measures, and blinded outcome assessments. However, the investigators did not place a value on the techniques employed by each study. Consequently, technical flaws in each study, lack of fluoroscopic guidance and contrast confirmation of accurate needle placement, were not properly analyzed.

Similar critical appraisals [67,68] of these studies have similarly failed to adequately assess the technical shortcomings of nontarget specific injections utilized to treat inadequately diagnosed lumbar pain. DePalma et al. [69] assessed the efficacy and safety of lumbar TFESI and selective nerve root blocks treating specifically lumbar radiculopathy not axial lumbar pain (Table 6.1).

Parr et al. [70] conducted a systematic review of lumbar ILESI in management of chronic LBP studies. They reviewed in total 8 systematic reviews, 20 randomized trials and 30 observational studies. They identified that in the literature body, the available studies included only blind epidural injections without fluoroscopic guidance. Thus in their description lacks applicable evidence in contemporary interventional pain management practices. They found limited evidence for blind ILESIs in managing all types of pain except for short-term relief of pain due to disc herniation and radiculitis.

Buenaventura et al. [71] in their systematic review of LTFESI in management of chronic LBP and lower extremity pain evaluated 11 randomized trials and 31 observational studies. The studies reviewed were all addressing lower extremity pain rather than axial LBP. They only identified four randomized studies [72–75] that met their criteria as they mentioned paucity of literature as a limitation. However, they concluded that TFESIs when appropriately performed should result in reduction of patient's pain by

Table 6.1 Results of Published Investigations of Lumbar TFESIs in Treating Radiculopathy

Author (year)	Control Group	No. of Subjects	No.# of Injections	Outcome Tools	Relief at F/U Compared to C	Results	Comments	Study Quality
Breivik (1976)	Bupiv/saline	35 (T = 16, C = 19)	1-3 CESI	Satisfactory pain relief; return to work	56%/26%; 3–17 months f/u	Positive	Cross-over design with 14% compared to 73% of control improving after cross-over	Good/Fair: No fluoroscopic guidance; no definitive diagnostics
Yates (1978)	Saline or lidocaine	20	1 CESI	Symptomatic improvement; SLR; lumbar ROM	Improvement with steroid 30 minutes and 1 week	Positive	4 tx groups (Saline, lidocaine, saline + steroid, lidocaine + steroid)	Fair: Only 1 injection offered; short follow-up interval; no fluoroscopic guidance; no definitive diagnostics
Mathews (1987)	Local lidocaine	57 (T = 23, C = 34)	3 CESI	Pain level	67%/56% at 1–3 months	Negative	16 of CESI and 23 controls required additional tx	Fair: Short follow-up interval; no fluoroscopic guidance; no definitive diagnostics
Serrao (2003)	Intrathecal midazolam	28	1 TLESI	Pain level/analgesic use	No difference at 2 months	Negative	Prospective, double blind	Fair: Short follow-up; only 1 injection performed; no fluoroscopic guidance
Helliwel (2002	Inters-spinous saline	39 (T = 20, C = 19)	1 TLESI	Pain level	Statistically sign reduction over control at 1 and 3 months	Positive	Single blind	Fair: 1 injection performed at undisclosed level; no fluoroscopic guidance; no definitive diagnostics
Karppinen (2001)	SP	160 (T = 80, C = 80)	1 TFESI	VAS, OLBDQ; NHP	Greater improvement at 3 and 6 months in control group	Negative	Power analysis calculated; primary symptom radicular	Fair: 1 injection performed/patient; no definitive LBP diagnostics
Ng (2006)	Bupiv	86 (T = 80, C = 80)	1 TFESI	VAS, OLBDQ	No difference at 12 weeks	Negative	HNP and foraminal stenosis-related radicular pain primary complaint	Fair: 1 injection performed/patient; no definitive LBP diagnostics; stenosis patients included
Jeong (2007)	Ganglionic injection	239	1 preganglionic vs 1 ganglionic	VAS, 4-grade scale improvement scale	Mean interval 373 days. Better treatment effect in preganglionic group	Positive	No significant difference in mid-term follow up. No long-term follow up.	Prospective randomized controlled study addressing what is the better location to inject

64% to 81%, disability by 60% to 63%, and depression by 56%. While considering the low risk and less expensive nature of the procedure compared to surgical interventions epidural injections seem cost effective.

Another recent review by Roberts et al. [76] concluded that there is fair evidence that TFESI are superior to placebo, and that there is good evidence that they should be used as surgery sparing interventions. They also suggested that TFESI are superior to ILESI and CESI for radicular pain.

Prospective, Randomized, Controlled Trials

Caudal Epidural Steroid Injections

Six prospective, randomized, controlled trials have been published evaluating the efficacy of CESI [77–82] and only three of which evaluated lumbar pain [80–82]. Critical features of each study that must be assessed are route of injection (fluroscopic control), number of injections, clinical presentation (LBP vs radicular pain), diagnostic evaluation (provocative discography), length of follow up, and outcome measures.

Breivik et al. [77] in a prospective, double-blind, crossover study assessed improvement in chronic LBP and sciatic pain in 35 patients treated with up to three blind, caudal epidural injections of either bupivicaine and methylprednisolone or bupivicaine and normal saline. The study followed a parallel, cohort design allowing patients not benefiting from their randomized treatment to then undergo treatment in the reciprocal arm. Initially, 56% of patients receiving methylprednisolone experienced significant relief compared to 26% treated with bupivicaine and saline. In the crossover, 14% of the methylprednisolone group obtained relief from subsequent bupivicaine and saline injections, while 73% of the bupivicaine/saline group reported satisfactory relief after the methylprednisolone injection. Fifty percent of the steroid group and 20% of the bupivicaine group returned to work at a range of 3 to 17 months after treatment. Up to three injections were performed in each arm. Thirty-two patients had undergone radiculography demonstrating disc prolapse, arachnoiditis, or inconclusive findings. However, the CESIs were without fluoroscopic guidance, and no further diagnostic testing had been performed.

In a subsequent study, Yates [78] performed caudal injections of saline, lidocaine; saline, triamcinolone; and lidocaine, triamcinolone in random order in 20 consecutive LBP patients. Each patient was assessed at 30 minutes and again 1 week after each injection. Outcome measures were improvement in straight leg raising and lumbar range of motion, both improved more so after the injections of steroid. Patients reporting more than 50% improvement demonstrating significant improvement in lumbar range of motion and straight leg raising. Yet, no specific diagnostic criteria were utilized in selecting patients for the trial, the follow-up interval was short, and limited injections were completed.

In 1987, Mathews et al. [79] randomized 57 patients suffering from chronic LBP and sciatica to CESI [8] or local infiltration of lidocaine over the sacral hiatus [20]. Three injections were completed reducing painful symptoms in 67% of treated and 56% of control patients 1 month after intervention. This difference did reach statistical significance at 3 months. Improvement remained greater in the treatment group through 1-year follow-up period, but the most profound difference was observed at 3 months. However, 16 CESI and 23 control patients required additional treatment, and the diagnostic evaluation was limited to blood work and lumbar plain films, the details of which were not reported.

The remaining three randomized, controlled CESI trials [83–85] enrolled patients solely complaining of unilateral radicular pain and did not report on changes in axial symptoms. The investigations that assessed axial LBP demonstrated short-term efficacy of CESIs in treating lumbar pain with long-term benefits waning over time. However, these injections are not target specific, utilized large volumes of injectate diluting the corticosteroid, and were performed without fluoroscopic guidance. Furthermore, due to unavailability, precision diagnostic evaluations were not completed confirming the presence of discogenic lumbar pain in the study subjects.

Only one study has prospectively evaluated the efficacy of CESIs in patients diagnosed with discogenic LBP by provocative discography [83]. More than 50% reduction in pain was achieved at 6 months after completing 1 to 3 CESIs in 60% and 64% of discogram negative and positive patients, respectively. Although each patient had undergone negative diagnostic facet joint and sacroiliac joint blocks with local comparative anesthetic, the investigators did not assess for concordantly painful outer annular disruption. A small number of patients comprised the discogram positive group. Unfortunately, neither the positive discogram levels nor the immediate postinjection improvement in LBP reported. Publishing whether or not patients experienced immediate improvement might help confirm if the CESI adequately reached the putatively painful disc. The results of this study, despite utilizing discography, do not confirm that CESIs are effective for discogenic lumbar pain.

Lumbar Interlaminar Epidural Steroid Injections

Ten controlled studies have been performed evaluating the efficacy of lumbar ILESIs [84–94]. Three studies [95–96] have investigated these interventions for axial lumbar pain, and seven evaluated ILESIs for radicular pain [87,94].

In a prospective, double-blind, randomized fashion, Serrao et al. [93] studied the therapeutic effects of single injections of 80 mg methylprednisolone epidurally compared to 2 mg of intrathecal midazolam in 28 patients with chronic lumbar pain. No statistically significant difference in pain or analgesic use was observed between the two groups at 2 months. The authors did not report their diagnostic evaluation of these patients, the segmental level at which each injection was performed, and fluoroscopic guidance was not utilized.

Years previously, Helliwel et al. [94] studied 39 patients with low back and radicular leg pain in a single-blind investigation. Twenty subjects underwent a single

extradural injection of 80 mg methylprednisolone in 10 mL normal saline. These patients reported statistically significant reduction in pain levels at 1 and 3 months compared to 19 control patients that underwent an interspinous injection of 5 mL normal saline. However, the authors did not report at what level each injection was performed and did not clarify if the pain scores were recorded for lumbar pain or radicular pain primarily.

In another study Carette et al. [84] performed a randomized, double-blind trial, administering up to three interlaminar epidural injections of methylprednisolone acetate or isotonic saline to 158 patients with sciatica caused by a herniated nucleus pulposus (HNP). There were no significant differences in outcomes in the short term or in the long term (1 year). They concluded that interlaminar epidural injections offered no significant functional benefit, nor do they reduce the need for surgery. The major flaw in this study was that fluoroscopic guidance was not used. The needle positioning was not confirmed either with fluoroscopy or by adding local anesthetic, and transient sensory and motor deficits were not monitored after the epidural injection.

Results of these trials weakly clarify the efficacy of lumbar TLESIs for axial LBP. These injections may afford the patient short-term improvement in lumbar pain. However, TLESIs are not target specific, especially in the absence of fluoroscopy, and definitive diagnostic measures were not taken in these studies.

Butterman [95] performed ILESIs in 93 patients with DDD and inflammatory end-plate changes and in 139 patients without inflammatory end-plate changes. Seventy-eight patients with inflammatory end-plate changes and 93 without inflammatory end-plate changes were considered fusion candidates, who underwent discography with or without intradiscal steroid in a randomized fashion. Outcome measures were VAS pain, Oswestry Disability index, pain diagram, and opinion of success before and after the patients injection for a 2-year follow-up period. MRI and discography results were correlated with patient outcome scores. Buttermann concluded that ESIs were effective in improving pain and function, as assessed by outcome scores at short-term follow-up. However, at 2-year follow-up, less than one-third of his patients had no additional invasive treatment. Patients with inflammatory end-plate changes had greater improvement in ODI and PD scores in the first 6 months than did those patients without the end-plate changes. Intradiscal steroid injections into discs with concordant pain at the time of discography led to significant improvement in patients with inflammatory end-plate changes in all outcome scales, but only minimal temporary improvement in patients without the end-plate changes.

The drop out in his study over 2 years was 60% and 35% of the noninflammatory group underwent fusion.

Transforaminal Epidural Steroid Injections

TFESIs are well studied in treating lumbar radicular pain [69,97–102] and one systematic review has been produced [69]. However, no well-designed study has evaluated the utility of lumbar TFESIs in treating axial lumbar pain. Two studies [72,100] included assessment of LBP in addition to lower limb radicular pain. One recent study [101] evaluated specifically the effectiveness of bilateral TFESI in comparison to ILESI in management of axial back pain, which we believe is the first serious effort to evaluate the bilateral transforaminal route in treatment of predominantly axial back pain.

Lutz et al. [33] in a prospective study evaluated the outcomes of therapeutic transforaminal epidural steroids in 69 patients for a mean period of 80 weeks. About 75% of their patients reported improvement of pain intensity of at least 50% and near return to their functional activities after 1.8 injections.

In a prospectively randomized study, Riew et al. [73] evaluated 55 patients with lumbar radicular pain with radiographic confirmation of nerve root compression. All of their patients had requested operative intervention and were considered surgical candidates. Instead the patients were randomized and underwent a selective nerve root injection with either bupivacaine alone or bupivacaine with betamethasone. The treating physicians and the patients were blinded to the medication. Twenty-nine patients did not have surgery during a follow-up period of 13 to 28 months. Of the 27 patients who had received bupivacaine alone, 9 did not have surgery. Of the 28 patients who had received bupivacaine and betamethasone, 20 decided not to have surgery.

Vad et al. [75] prospectively included 50 patients with lumbar radicular pain longer than 6 weeks. All patients had MRI evidence of HNP with less than 50% narrowing of the neural foramen along with radicular pain corresponding to the MRI positive level of pathology. Patients were randomized into two groups. The treatment group underwent fluoroscopically guided transforaminal epidural injections while the control group received trigger point injections. Forty-eight patients completed the study. Sixteen months was the average follow up. Outcome measures were Roland Morris Questionnaire, Visual Analogue scale, and finger-to-floor test. There was an 84% improvement in the study group and only a 24% improvement in the control group.

In a prospective study, Botwin et al. [102] included 34 patients with unilateral radicular pain caused by degenerative spinal stenosis, which was not responding to conservative management. Their patients underwent an average of 1.9 fluoroscopically guided transforaminal epidural injections at the symptomatic side. Visual analog scale score, Roland 5-point pain scale, standing and walking tolerance, and patient satisfaction scale were assessed 2 months and 12 months after the injections. Seventy-five percent of patients reported more than 50% pain score reduction between pre- and postinjection. Sixty-four percent of patients had improved walking tolerance, and 57% had improved standing tolerance at 12 months.

Karppinen et al. [72] completed a double-blind, randomized, and controlled trial of patients with lumbar radiculopathy due to a corroborative disc herniation who underwent a single TFESI at the indicated level. Although the study was designed to assess the efficacy of a TFESI for nerve root pain, the authors did evaluate lumbar pain. Each injection was performed under fluoroscopy employing

2 to 3 mL of injectate at the level of clinical involvement. Only one injection was performed in each patient, with the treatment arm undergoing injection of 1 mL of 40 mg/mL methylprednisolone and 1 mL of 5 mg/mL of bupivicaine, and the control group underwent injection of 2 mL of isotonic saline. Eighty patients were enrolled into each group after a power analysis revealed a need for 68 patients in each study arm. No difference in immediate improvement in lumbar pain occurred between the groups. At 2 and 4 weeks, and 12 months follow up, lumbar pain intensity was subtly less in the steroid group, which did not reach statistical significance. In contrast, at 3 and 6 months statistically greater improvement in lumbar pain occurred in the saline group.

Although the Karppinen study utilized an appropriate number of subjects, the investigation did not ideally address the question of whether or not TFESIs are effective for discogenic lumbar pain. Each patient's lumbar pain was presumably due to the disc herniation, whose posterolateral annular fibers were reached by the injectate [27], affecting the treated nerve root. However, further diagnostic interventions such as provocative discography were not performed, the side of the annular disruption may not correspond to the side of symptom manifestation [101], and the axial pain symptoms may have actually represented proximal nerve root pain. Only one injection was completed in each patient. Frequently, more than one injection may be necessary to adequately treat radicular pain [33,73], but similar evidence is lacking suggesting how many injections may be necessary to adequately treat discogenic lumbar pain. Epidural saline may have a greater therapeutic benefit [103,104] than placebo thus underestimating a treatment effect in Karppinen's study. Long-term benefits may not have been achieved due to a subtherapeutic number of TFESIs performed in the treatment arm, lack of true placebo control (sham injection), and improperly targeting the correct level of axial pain generation.

Ng et al. [105] randomized 86 lumbar radicular pain patients in double-blind manner to either periradicular infiltration of 2 mL of 0.25% bupivicaine or 2 mL of bupivicaine and 40 mg of methylprednisolone. Inclusion criteria included patients with lower limb pain equal to or greater than back pain and MRI evidence of a corroborative disc herniation or foraminal stenosis. Each patient underwent only one injection, and VAS rating and Oswestry Disability Index were measured at weekly intervals up to 12 weeks with 100% follow-up rate. No difference was observed for lumbar pain or disability at 12 weeks after one injection. It is difficult to derive conclusions from this study regarding the efficacy of one TFESI to treat axial lumbar pain. Corroborative diagnostic evaluation such as discography was not performed. Patients with foraminal stenosis were included. These two factors may have diminished the likelihood of discogenic lumbar pain in a portion of enrolled patients. Patients requiring repeat injections were deemed treatment failure and removed from the study follow up. Therefore, a subtherapeutic number of TFESIs may have been undertaken.

Ackerman and Ahmad [100] compared TFESIs versus ILESIs and CESIs in management of S1 radiculopathy secondary to L5-S1 disc herniation. Ninety patients were uniformly randomized to receive the injections every 2 weeks with a maximum of three injections. Pain relief, disability, and activity levels were evaluated for a period of 24 weeks. TFESI subjects obtained better pain relief with more of the subjects obtaining complete relief. The TFESIs required fewer injections to obtain pain relief of 1.5 compared to 2.2 in ILESIs and 2.5 in CESI groups. Additionally, epidurograms of the three injection types were evaluated by a blinded observer trained in epidurograms interpretation, and it was found that anterior epidural spread of contrast was associated with more complete relief.

Lee et al. [101] evaluated bilateral TFESI versus fluoroscopic-guided ILESI in 93 patients with herniated lumbar discs and 99 patients with spinal stenosis that reported axial back pain without radiation for more than 3 months. The injections were performed at the level that best matched the patient's clinical presentation. Their patients received one injection. Both injection routes accomplished significant pain reduction for 2 weeks to 4 months in both groups. The spinal stenosis group showed a more significant reduction in the Roland 5-point pain score and obtained more successful Numerical Rating Scale results using the TFESI as compared with the ILESI. Patients with herniated discs did not show any differences with either technique. They concluded that the ILESI can be more affected by tissue fibrosis, scarring, or hypertrophy, which is more prominently featured in spinal stenosis than in herniated discs; these prevent the injectate delivered via the posterior route from spreading to the ventral epidural space. This study is important as it only evaluated patients with axial back pain and compared the bilateral TFESI, which is currently largely used by interventionalists for management for axial LBP.

Synthesis of Best Evidence

One to three CESIs appear to be effective in reducing nonspecific LBP in the short term. More definitive statements regarding efficacy of CESIs for discogenic LBP and the duration of benefit cannot be made as such data has not been generated. A single ILESI may provide short-term relief from nonspecific LBP; however, long-term improvement in nonspecific LBP with ILESI has not been founded. TFESI do not appear to be effective in reducing nonspecific LBP after performing just one injection in the absence of definitive diagnostic evaluation establishing discogenic LBP. However, conclusive statements regarding the appropriate role of CESI, ILESI, and TFESI cannot be made until better research protocols are developed.

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7 Intradiscal Heating Procedures for Lumbosacral Internal Disc Disruption

Leonardo Kapural

INTRODUCTION

The diagnosis of discogenic pain often remains imprecise secondary to its nonspecific clinical features. More typical features include persistent, nociceptive low back, groin, and/or leg pain that worsens with axial loading and improves with recumbency. These features alone, however, are frequently insufficient to establish an accurate diagnosis and comprehensive treatment plan for the patients with such complaints. This has led many practitioners to employ provocation lumbar discography (chapter 5) in conjunction with magnetic resonance imaging (MRI; chapter 4) studies as a means of validating their clinical diagnosis of discogenic low back pain (LBP). Although MRI images are helpful in visualizing such pathology as disk degeneration and desiccation, high-intensity zones, and loss of disk height, the results commonly correlate poorly with clinical findings, leaving open the critical question of causality. To date, provocation discography is the only available method of linking the morphologic abnormalities such as annular fissures that may or may not be seen on MRI with clinically observed pain, and its predictive value has been repeatedly questioned mainly as a result of reported false-positive rates [1–3].

Once the provisional diagnosis of discogenic pain has been suitably established, the next challenge involves instituting an effective therapy. Several of the most common current therapies involve careful heating of the annulus fibrosus (annuloplasty procedures) in which the morphologic substrate for LBP resides. Historically, these modalities have been used despite a somewhat poorly understood relationship between the therapeutic effects and the histologic changes observed [4–8]. A presently held opinion is that denervation of the tissue or destruction of the nociceptors, and less likely alteration of the collagen fibers in the annulus producing denaturation and coalescence, may be the predominant therapeutic mechanism [5–8]. Major advantages of these procedures generally include their minimally invasive approach, low cost, and relative simplicity compared to surgical procedures such as lumbar fusion or disc replacement. Intradiscal Electrothermal Therapy (IDET; Smith and Nephews, London, UK), discTRODE (Radionics Inc., Burlington, MA) and intradiscal biacuplasty (Baylis Medical Inc., Montreal, Canada; Figures 7.1 and 7.2) are several examples of the annuloplasty approaches using heat to treat discogenic pain.

MECHANISMS OF PAIN RELIEF

Delamination or tearing of the lamellar layer of the annulus, dehydration, and loss of nuclear material with increasing age are associated with disc degeneration. Physical changes observed in the degenerative disc are also closely associated with biochemical and cellular changes. The degenerating disc spontaneously produces inflammatory cytokines including tumor necrosis factor-α (TNF-α), nitric oxide, and matrix metalloprotineases (MMPs) [9,10]. Increased vascularization in the degenerated disc further facilitates the introduction of these inflammatory cytokines [9]. Nerves that are restricted to the outer third of the annulus in a normal disc penetrate into the degenerated disc within newly vascularized annular fissures [11–14]. Immunohistochemical studies have shown that these ingrowing nerves are nociceptive in origin

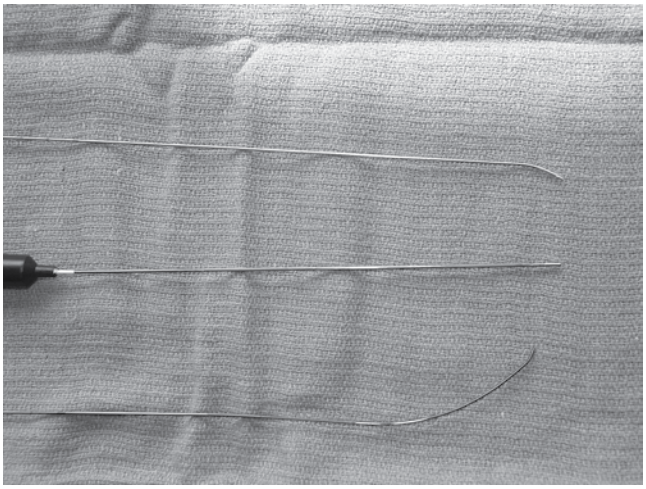

Figure 7.1 Compared are three currently available electrodes used for the lumbar annuloplasty. On top is discTRODE radiofrequency electrode with the flexible upper part to facilitate the electrode placement via the introducer and within the posterior annulus. Middle electrode is one of two bipolar electrodes used for the radiofrequency biacuplasty. Note the rigid electrode tip that cannot be shaped. Lowest is the IDET resistive coil with a flexible, slightly curved tip allowing relatively smooth placement of such coil in a circular fashion at the interphase between the annulus and nucleus.

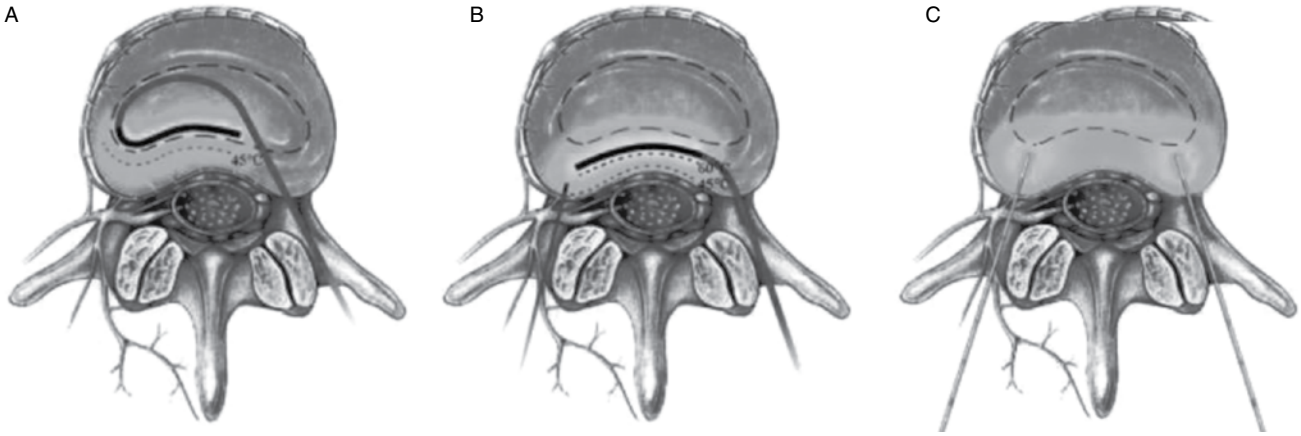

Figure 7.2 Schematic of the appropriate intradiscal electrode positioning during various currently available annuloplasty approaches. **(A)** An IDET coil proper positioning at the interface between annulus and nucleus. **(B)** Final position of the RF DiscTrode electrode and temperature probe within the posterior annulus. **(C)** Shown are two cooled electrodes for biacuplasty and their spatial relationship within posterior annulus of the lumbar disc. The heat distribution is achieved across posterior annulus without proximal dissipation of the heat to posterior neural elements.

(C- and Aδ-fibers), and thus responsible for transmitting pain responses [10,13]. It is believed that the inflammatory cytokines increase the sensitization of these ingrown nerves and may be the primary source of discogenic pain. Buffering these nociceptive fibers may disrupt the transmission of pain signals.

Tissue modulation including shrinkage, denaturation, and structural changes to collagen fibres in the annulus to increase annular stability, is one hypothesis proposed to explain the mechanism of action for the thermal treatment of discogenic pain. In fact, the rationale of heating the disc as a treatment for discogenic pain was largely influenced by animal and clinical studies that heat can stabilize the shoulder capsule by modifying and shrinking collagen [15]. As the annulus is composed of collagen, heating may cause collagen shrinkage, create a seal to limit infiltration of inflammatory molecules and leakage of disc materials, and cause neural coagulation of nociceptive fibers. Collagen shrinkage and subsequent denaturation has been demonstrated to occur at 60 to 65°C in animal models [16]. However, there is limited evidence to support this mechanism of action at least for IDET type of annuloplasty. A study by Kleinstueck et al. [17] in cadaveric lumbar specimens found that temperatures generated during IDET are insufficient to alter collagen architecture.

Perhaps, a more likely mechanism of action for thermal disc treatments is denervation of ingrown nerves by neuroablation. Temperatures reached in the disc using the TransDiscal System or IDET are sufficient to cause nerve destruction, which occurs at 42 to 45°C [7,18–20]. It is believed that the pain pathway is disrupted by ablating ingrown nociceptive fibers by the delivery of radiofrequency (RF) energy to the disc. Ultimately, scientific evidence to demonstrate neuroablation as the mechanisms of action for discogenic pain relief remains unavailable and continues to be the impetus for continued research in this area.

The temperature profiles of the latest intradiscal heating procedure, intradiscal biacuplasty, had been investigated in both porcine and human cadaveric lumbar discs. Histological examination showed no evidence of injury to the surrounding neural structures and no evidence of tissue charring in sections adjacent to where the probes were positioned. Histological analysis also showed no sign of tissue degradation due to heating or changes in the collagen structure in both degenerated and nondegenerated disc samples [19,20]. Overall, these studies suggest that the disc biacuplasty procedure can be used to ablate ingrown nerves in an internally deranged disc, but not to alter collagen.

INTRADISCAL ELECTROTHERMAL THERAPY

Efficacy

When considering indications for interventional approaches such as IDET annuloplasty, the most common criteria include discogenic LBP, persistently present for more than 6 months. Further, this pain must remain despite comprehensive conservative treatment including physical therapy, a directed exercise program and at least one fluoroscopically guided epidural corticosteroid injection. Saal and Saal initially prescribed additional study criteria for IDET that included: normal neurological examination, negative straight leg raise, absence of any inflammatory arthritides, or nonspinal conditions that may mimic lumbar pain, and absence of prior surgery at the symptomatic intervertebral disk level [21–23]. Further, no neural compressive lesions should be seen on MRI and provocation discography should reproduce concordant pain at low pressurization at one or more —but no more than three— intervertebral disk levels. This criteria set was one of several variations used in subsequent studies evaluating the efficacy of IDET (for review, see ref. [24]). When comparing the studies, variation in patient selection, as well as heating techniques, are thought to account for differences seen in clinical results [24–34].

Overall, the average pain score improvements in 13 studies analyzed were between 1.5 and 5 visual analogue

scale (VAS) points. SF-36 physical function (PF) scores for evaluation of functional capacity improved from approximately 15 to 30 in four separate studies [24]. Overall results of IDET appear to improve with several additional patient-selection criteria [27,33]. Such criteria were evaluated in Pauza and colleagues' sham-controlled, prospective IDET study. Specifically, they restricted patients to: (a) Beck depression scale score of less than 20; (b) less than 20% disk height narrowing on lateral x-ray; (c) no surgical interventions within previous 3 months of study enrollment [27]. Although improvement was seen in both groups, greater improvements in mean pain and functionality scores were reported in patients who underwent IDET. Pauza and colleagues' [27] use of provocation discography rather than MRI/discography combined criteria for the enrollment, may have contributed to high number of patients needed to treat—five to achieve more than 75% improvement in one patient.

There was significantly less improvement in functional capacity and pain relief following the IDET in patients with any signs of disk degeneration on MRI at more than two lumbar levels during the separate prospective study where multilevel disc degeneration patient group improvements were compared to those with only one or two degenerated disks as shown on the MRI. In this particular study, patients were matched for the number of lumbar disk levels positive on discography [33]. As single-level disease is less commonly present, it is reasonable to believe that Pauza and colleagues' patient selection realistically illustrates the expected results of the IDET procedure in the majority of patients presenting with discogenic pain [27,33]. Overweight patients [34] and patients receiving workers' compensation benefits [32,35] represent additional patient subsets that are less likely to benefit from the IDET.

Using significantly different selection criteria than Pauza et al., recently published randomized, double-blinded, controlled IDET study by Freeman and colleagues reported no significant improvement between treatment and placebo in patients with discogenic pain [36]. Importantly, selection criteria for this study did not include data regarding body mass index and depression scores, nor the number of disk levels that appeared degenerated on MRI. Further, more than half of the enrolled patients exhibited "marked functional disability" and were receiving workers compensation benefits at the time of their participation in the study. Freeman and colleagues also used patients belonging to the groups previously referred as likely IDET failures [32–35].

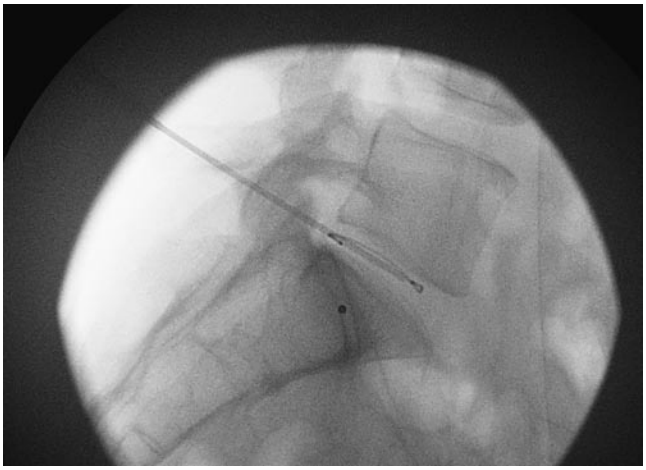

Figure 7.3 Lateral view of an IDET resistive coil appropriately positioned within the L5-S1 intervertebral disc. Note that the resistive coil formed a circle within the intervertebral disc and away from the anterior epidural space.

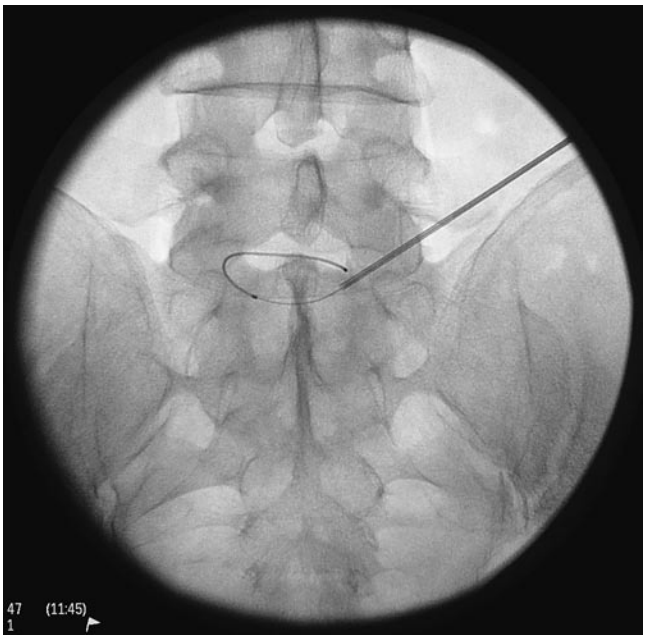

Figure 7.4 Craniocaudal view of the final position of an IDET resistive coil. The coil appears to be within the disc and covers most of the posterior interface between intervertebral annulus and nucleus.

Technique

Briefly, IDET is performed under local anesthesia and mild intravenous sedation under sterile conditions. Intravenous antibiotics, 1 g of cefazolin or 1 g of vancomycin, should be given 30 to 60 minutes before the procedure. Patients are positioned prone using midabdomen support to correct for the lumbar lordosis. Using local anesthesia, a 17-gauge needle is inserted under fluoroscopic guidance into the targeted disc. Through that same needle, a catheter with thermal resistive coil is navigated until positioned appropriately within the disc (Figure 7.3). The key is to position such a catheter across all of the semicircumference delineated by the interface of the posterior annulus and nucleus (Figure 7.4). The thermal resistive coil generates gradual rising temperature inside the disc up to 90°C in 0.5°C increments. The temperature is then maintained at 90°C for 4 minutes according to manufacturer protocol (Smith and Nephews, London, UK).

OTHER ANNULOPASTY PROCEDURES

IDET is not the only minimally invasive annuloplasty procedure. However, no differences in pain scores or improvement in functional capacity between the sham and the treated patient groups were seen in the randomized controlled trial using the original Sluijter RF technique in which the nucleus (and not the annulus) was heated to 70°C for 90 seconds [37]. A disappointing performance was turned in by the novel annular probe termed "discTRODE" as well, after treatment of patients with discogenic pain reported only modest or no improvements in pain scores and functional capacity [38,39]. This technology proved to be ineffective in improving functional capacity and VAS scores versus IDET during study where strict patient-selection criteria were employed [38].

INTRADISCAL BIACUPLASTY

Efficacy

Intradiscal biacuplasty is the latest minimally invasive posterior annulus heating technique. This technology employs bipolar RF electrodes and—based on improvement in pain scores and functional capacity in patients with discogenic pain—is likely the most promising of all currently available, minimally invasive intradiscal heating methods [40–43]. This method works specifically by concentrating RF current between the ends of two straight probes. Relatively even heating over the larger area of the posterior annulus is achieved by internally cooling the electrodes [19,20,44] (Figures 7.2 and 7.3). The procedure is completed under fluoroscopy with the patient lying in the prone position. Two TransDiscal electrodes via introducers are placed bilaterally in the posterior annulus of the intervertebral disc. The generator controls the delivery of RF energy by monitoring the temperature measured by a thermocouple at the tip of the probe. The temperature increases gradually over a period of 7 to 8 minutes to 45°C with final heating for another 7 minutes. During this time, the patient should be awake and can communicate with the physician.

The first disc biacuplasty procedure was performed on a young male with severe chronic back pain for 2.5 years. The procedure was performed in the L5/S1 intervertebral disc following elicitation of concordant pain by provocation discography with two control discs. The cooled RF probes were positioned inside the disc as described earlier. The patient received significant improvement in pain scores and functional capacity at 1 and 6 months following the procedure with no perioperative complications. The VAS pain score changed from 5 cm to 1 cm, reduction from 14 to 6 points was observed on the Oswestry scale, improvement in SF-36 scores, and absolute patient satisfaction [40]. Later, two case series involving 8 and 15 patients also demonstrated significant pain relief following the disc biacuplasty procedure at 3, 6, and 12 months [41,42,45]. In the European case series involving eight patients, there was an average of about 50% pain reduction at 3 months with overall good patient satisfaction. No patients reported any postprocedural pain, often associated with other therapies, and needing additional analgesia [45]. In the prospective pilot study involving 15 patients, Kapural et al. [41,42] reported patient improvements in several pain assessment measures after undergoing disc biacuplasty procedure for discogenic pain, again with no procedure-related complications. Results from these pain assessment measures included a reduction in the median VAS pain score from 7 cm to 4 cm at 1 month and remained at 3 cm at 6 and 12 months follow-up, improvement in Oswestry index from 23.3 to 16.5 points at 1 month and remained similar after 12 months, and increase in the SF-36 bodily pain score from 38 to 54 points [41,42]. Pilot studies and case series, even when designed as a prospective trial, tend to exaggerate the positive outcomes. Therefore, we are awaiting results of the sham, prospective randomized study in order to accept or refute such approach in treating discogenic pain.

In addition to primary discogenic pain, residual LBP or back pain replacing leg pain is commonly seen in as many as 75% of the patients following surgical discectomy. It is likely that discectomy produces permanent anatomical, biochemical, and functional changes within the treated intervertebral disc, potentially leading to the development of LBP [46]. A recent case report described successful treatment of discogenic pain using the disc biacuplasty procedure in previously discectomized disc [43].

Intradiscal biacuplasty seems to present several advantages over previous techniques. There is minimal disruption to the native tissue architecture, and thus the biomechanics of the spine likely remain unchanged. The relative ease of electrode placement eliminates the need to thread a long heating catheter (e.g., IDET). Lower peak heating temperature in the disc compared to other thermal annular procedure seems to allow better patient tolerance. In addition, internal cooling of the probes prevents excessively high temperatures in the disc that may cause tissue adherence [19,20,44]. Randomized controlled trials are currently underway to evaluate the efficacy of the disc biacuplasty procedure for discogenic pain.

Technique

The biacuplasty procedure is completed under fluoroscopy with the patient lying in the prone position. Anxyolysis and analgesia can be provided for relaxation and pain control before and during the procedure. Two 17G TransDiscal introducers are placed bilaterally into the posterior annulus of the intervertebral disc. Target for entry into the disc is identified by rotating the C-arm of the fluoroscope 20° to 30° from the sagittal plane such that 1/3 to 1/2 of the disc appears lateral to the Superior Articular Process (SAP). The introducer should be directed along this line, and should enter the disc at approximately the center of the disc height. This will ensure the electrodes are sufficiently far away from the endplates to increase the safety of the procedure. The introducers are advanced into the disc until the tips appear to be aligned with the medial edge of the pedicles in an anterior-posterior (AP) image. Two 18G TransDiscal probes are then placed inside the disc through the two introducers. Probe placement should be checked in the AP and lateral views to ensure

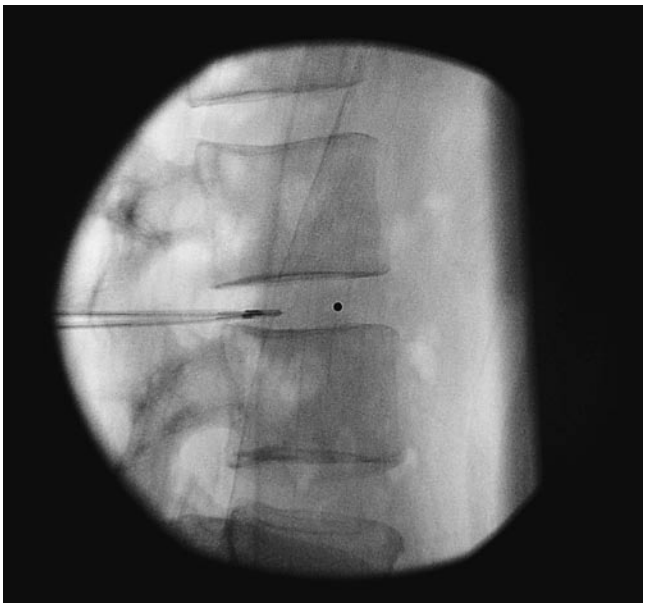

Figure 7.5 Transdiscal electrodes shown in a lateral fluoroscopic view. The radiopaque band denotes the beginning of the active electrode tip and it must be positioned within the disc space.

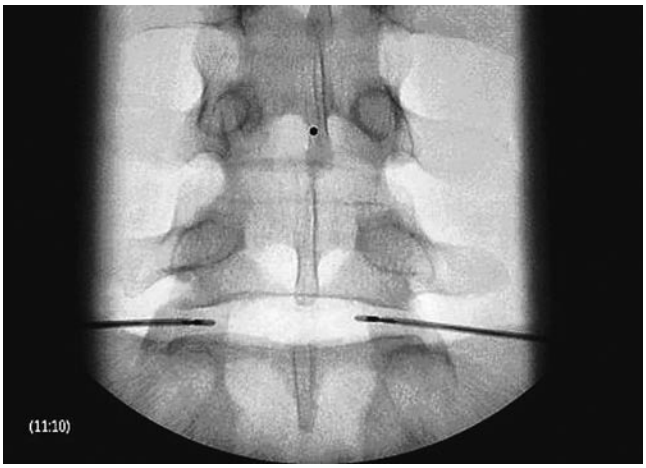

Figure 7.6 Anterior-posterior fluoroscopic view of the intradiscal biacuplasty electrodes within the annulus of L5-S1 intervertebral disc.

appropriate disc entry points and depth of probe placement. Figures 7.5 and 7.6 show the lateral and AP views of the lumbar spine after probe placement. The generator controls the delivery of RF energy by monitoring the temperature measured by a thermocouple at the tip of the probe. The temperature increases gradually over a period of 7 to 8 minutes to 45°C with final heating at 45°C for another 7 minutes. It should be noted that although the temperature is set to 45°C on the RF generator, tissue temperature reaches 65°C because of ionic heating. During this time, the patient should be awake and can communicate with the physician. Following completion of the procedure, the patient is required to wear a brace and follow physical therapy instructions over a rehabilitation period.

CONCLUSIONS

Several new minimally invasive intradiscal techniques for discogenic LBP control have been introduced recently, but sufficient clinical evidence of their efficacy and extent of application is still lacking overall. DiscTRODE annuloplasty and conventional nuclear RF seem to be ineffective in reducing pain and improving functional capacity in patients with discogenic LBP. IDET and intradiscal biacuplasty types of annuloplasty may produce positive therapeutic effect in highly selected patient group.

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8 Intradiscal Biologic Agents for Lumbosacral Internal Disc Disruption

Rajeev K. Patel

INTRODUCTION

Development of a cell-based biological replacement to restore, maintain, and improve the function of injured annular tissue represents a strategic approach that warrants investigation. Relating recent findings about the molecular mechanisms involved in the initiation and/or propagation of degenerative and pathologic structural alterations of the intervertebral disc (IVD) will be crucial to achieving success in developing biological instruments to cure discogenic low back pain (LBP).

ANATOMY AND PATHOPHYSIOLOGY OVERVIEW

The IVD transmits load resulting from body weight and muscle activity, and provides flexibility to the spine. Three highly specialized structures constitute the IVD: the endplates, the annulus fibrosus, and the nucleus pulposus. The two cartilaginous endplates form the cephalad and caudad interface between the disc and the adjacent vertebrae, acting to enclose the disc axially. The annulus fibrosus is composed of several lamellae formed by parallel collagen fibers interspersed with elastin fibers. The nucleus pulposus—a gelatinous core containing randomly organized collagen fibers, radially arranged elastin fibers, and a highly hydrated aggrecan-containing gel—surrounds the annulus fibrosus. The hydrated proteoglycans within the nucleus pulposus are essential to maintain the osmotic pressure and, therefore, play a vital role in the load-bearing properties of the disc.

The IVD is the largest avascular structure in the human body. As we age, and as growth and skeletal maturation proceed, degenerative processes begin to change the morphology and function of the disc. The most widely accepted conceptual model of spinal segmental degeneration was proposed by Kirkaldy-Willis. In this model, the nucleus pulposus of degenerated discs is characterized by reduced water and proteoglycan content leading to the loss of its gel-like radiographic appearance and hydrostatic properties. Degenerative changes of the annulus fibrosus are less obvious by imaging findings but result in irregular lamellae with disorganization of the collagen and elastin networks. The replacement of the gel-like structure of the nucleus pulposus with fibrocartilaginous tissue decreases the IVD's flexibility, leading to cleft formation and annular fissures. Up to 50% of the IVD cells show signs of necrosis and some reveal signs of apoptosis, resulting in cell loss from the disc [1]. Although there is a broad consensus about these hallmarks of degeneration, the extent of revascularization and/or reinnervation of the inner portions of the disc that can occur during degeneration has been debated [2]. Although studies have described revascularization, possibly accompanied by reinnervation, of the inner parts of the IVD, it is not completely clear at which stage of degeneration and/or injury these events occur [2]. Clarification of this scenario is meritorious because the interplay between neovascularization and neoinnervation might be of crucial importance for the morphological substrate for LBP emanating from degenerated and injured discs. Answers to these questions may ultimately determine the rate-limiting step to the potential of biological cures of symptomatic degenerative discs.

Degeneration of the IVD involves a complex interplay of nutritional effects, mechanical load, and genetics. Of these, nutrition and waste product removal likely play a special role in realizing the potential intradiscal biologics. Insufficient nutrient supply to the disc cells is thought to be a contributing abnormality to disc degeneration. Cells of the IVD face the precarious situation of maintaining a huge extracellular matrix with a "fragile" supply of nutrients that can be easily disturbed because of the avasularity of the IVD whose nutrition is dependent upon diffusion. Because of the size of the IVD, the nutrients need to diffuse from a capillary network within the vertebral bodies through the endplates and the disc matrix to the cells in the nucleus of the disc. The blood supplied for IVD nutrition becomes more restricted over time as the originally cartilaginous endplates become calcified with progression of the degenerative process. As IVD access for glucose and oxygen are restricted due to diffusion distances, the removal of metabolic waste such as lactic acid becomes critically impaired. Measurements have demonstrated low oxygen concentrations in the nucleus, which increased toward the disc surface, whereas the lactic acid concentration showed the reverse trend. The accumulation of lactic acid results in an intradiscal environment with a reduced pH. Low oxygen concentrations and acidic pH adversely affect the synthetic activity and proteoglycan

synthesis rates of disc cells. This toxic environment may lead to a fall in proteoglycan content and, consequently, to disc degeneration. This suboptimal environment may lead to increased cell death, and reduced cell numbers in the disc [3]. Ultimately, the result of poor nutritional supply to the IVD is a burden placed on the few remaining cells struggling to maintain an extensive matrix. Unfortunately, it is likely true that the progression of matrix degeneration becomes irreversible once the cell density falls below a minimal threshold.

THERAPEUTIC BIOLIOGIC STRATEGIES

Biological treatments for the degenerated IVD have been used sparsely to regenerate or cure the painful, deteriorated disc and restore biological function. Currently, intradiscal biological agents can be classified into four categories:

1. Direct injection of a biologically active factor(s) (Figure 8.1)
2. Modification of the gene expression of resident disc cells in vivo (direct gene therapy) (Figure 8.2)
3. Supplementation with autologous implantation of in vitro cultivated and modified cells (Figure 8.3)
4. Stem cell–based gene therapies (Figure 8.4)

These applications aim for sustained delivery of biologically active substances to the disc that should stimulate regeneration or conserve the status quo. The nature of the respective active factor is defined by our knowledge of the molecular mechanisms active in the disc during the various stages of degeneration. The applicability of the various approaches depends largely on our current knowledge of disc cell biology, the state of degeneration of the IVD, and potential safety concerns. The percutaneous delivery technique is similar to discogragphy (see chapter 5) by which the active agent is injected within the nucleus under fluoroscopic guidance via a posterolateral, parapedicular approach.

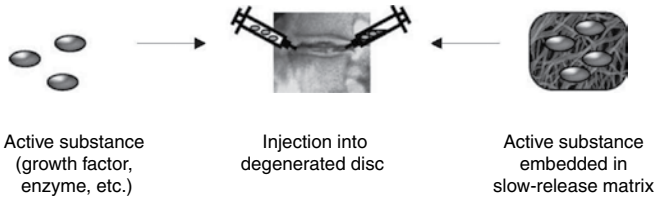

Figure 8.1 Intradiscal injection of a "naked" biologically active factor done to facilitate the sustained release of an agent into the cellular matrix.

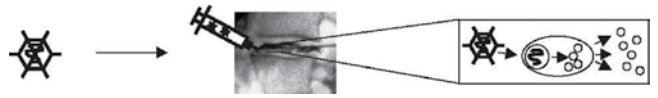

Figure 8.2 Modification of the gene expression of resident disc cells in vivo (direct gene therapy) using a viral vector carrier resulting in transformation and sustained expression of the active protein Z.

INTRADISCAL INJECTION OF A "NAKED" BIOLOGICALLY ACTIVE FACTOR

Percutaneous intradiscal injection into the disc provides the most straightforward delivery to the disc cells of an active biologic factor. Although direct application of potentially beneficial factors (growth factors, cytokines, or anabolic enzymes) has frequently been explored in vitro, few studies have been published attempting this approach in vivo. Promising findings have been observed after injecting rabbit lumbar IVD in vivo with osteogenic protein-1 (OP-1), a growth factor belonging to the TGF-β superfamily of growth factors. Direct intradiscal injection of OP-1 resulted in significantly increased proteoglycan synthesis and restoration of disc height, which persisted up to 24 weeks after injection [4,5]. Additional studies demonstrated that IVD OP-1 injection reduced pain-related behavior in a rat disc degeneration model [6,7]. Subsequently, Chubinskaya et al. [8] documented an anticatabolic effect of OP-1 after intradiscal injection in a rat model by demonstrating reduced immunostaining for aggrecanase, MMP-13, substance P, TNF-α, and IL-1β. Because substance P is a neuropeptide linked with inflammation and pain, the aforementioned reduction of this noxious protein supports the observed reduction in pain-related behavior in the rat disc degeneration model [6–8]. Furthermore, Miyamoto et al. [9] were able to demonstrate that intradiscal injection of OP-1 restored the biomechanical properties of IVDs in the rabbit model of degenerative discs. These investigators reported that a single injection of OP-1 not only significantly restored IVD height, but that the treated discs demonstrated a higher viscous and elastic modulus because of increased proteoglycan content in the nucleus as well as increased collagen content in the nucleus and annulus. Work by Kawakami et al. [10] addressed concern about the potential of ectopic bone formation within the epidural space with OP-1 therapies. These authors demonstrated that there was no macroscopic evidence of ectopic bone formation, no motor paresis, and no behavioral differences to motor stimuli with epidural administration of OP-1 in a rat model. Collectively, these findings demonstrate the feasibility of direct intradiscal injection of OP-1. Yet, this technique may be limited to the presence of disc cells that are still healthy, numerous, and able to respond to a biologically active agent. Considering the decreasing viability and synthetic activity of human disc cells during progressive degeneration, future directions for this technique may best be suited for success in the younger discogenic LBP patient population whose painful IVD are modestly degenerated or in which synergistic combinations with other biologically active agents are pursued.

In contrast to injecting a biologically active enzyme or growth factor, Klein et al. [11] published a clinical pilot study using direct injection of a mixture of matrix components and aiding components known to induce

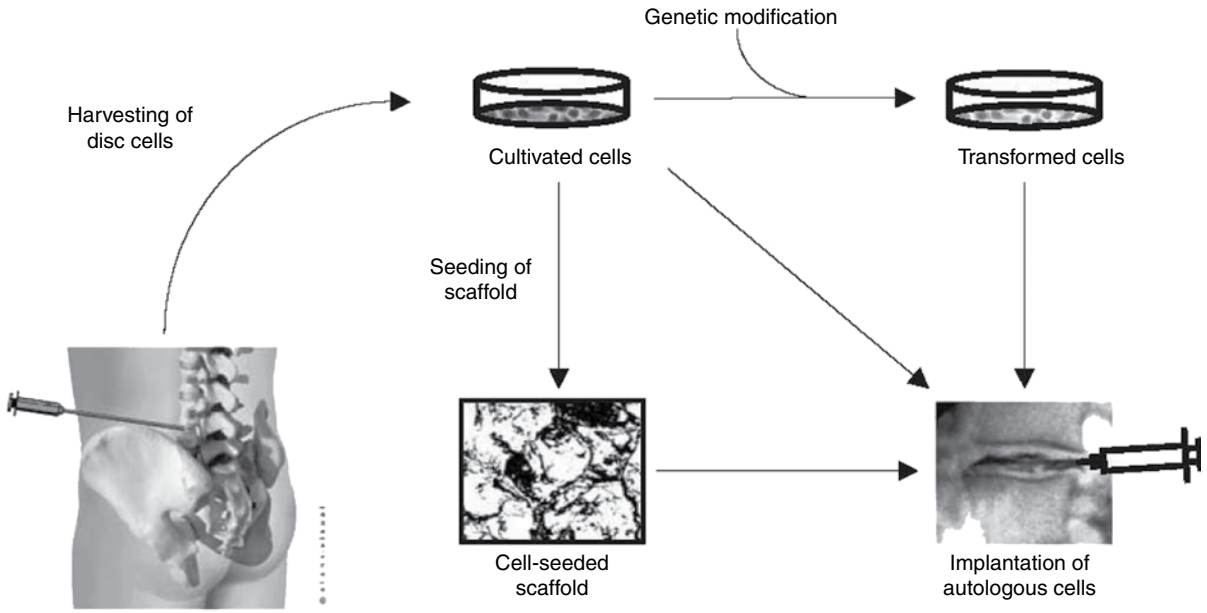

Figure 8.3 Supplementation with autologous implantation of in vitro cultivated and modified cells. Cultivated cells can be genetically modified in vivo before implantation (indirect gene therapy), seeded into a scaffold, or, simply implanted directly.

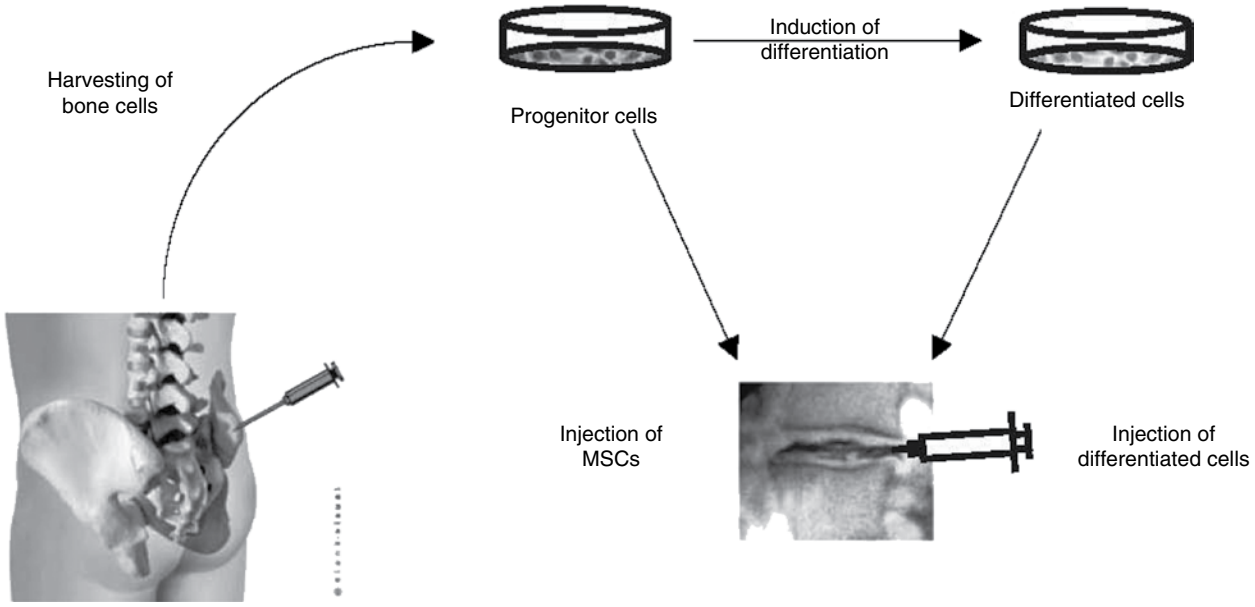

Figure 8.4 Stem cell–based gene therapy. Mesenchymal stem cells can be cultivated as progenitor cells and either injected directly into the disc matrix or be differentiated in vitro into a disc cell and then injected.

proteoglycan synthesis. This solution of glucosamine and chondroitin sulfate combined with hypertonic dextrose and dimethylsulfoxide was injected into 30 patients who exhibited concordant pain on provocation during lumbar discography (PLD). These patients reported reduction in disability and LBP at an average follow-up of 13 months. The authors suggested that the good outcome might be due to the combination of several components resulting in a parallel replenishment of the matrix by increased proteoglycan synthesis and induction of disc repair by simultaneous induction of multiple growth factors. This strategy might prove superior to the injection of a single bioactive factor. Perhaps, the injected matrix components modulate and improve the intradiscal environment enabling native disc cells, even in a degenerated disc, to react to the resulting secretion of growth factors and continue to maintain the cellular environment. However, from this pilot study it is not clear whether the injected

components will be contained inside the disc in heavily degenerated discs and be able to ensure a prolonged beneficial effect on the disc cells. Further, controlled comparative studies are required before any therapeutic conclusions can be drawn.

Common to these "injection techniques" is the concern that the demonstrated short-term effects are not durable over time because the originally injected material is consumed or is lost from the disc cells by diffusion. To provide the disc with a continuing supply of biologically active factors, it would be desirable to continuously produce the biologic agent of choice or include the substance in a pharmacological slow-release system as suggested for the use of growth factors [12]. Considering these data, it is conceivable that combination therapy of growth factor(s) and matrix replenishment might be the way to obtain a more sustained improvement in the altered parameters of degenerated discs. This approach may also allow for expansion of the previously stated bounding limitations with application of this technique to various grades of degeneration because of the requirement of a certain density of healthy disc cells.

GENE THERAPY APPROACHES

Deliberate modification of the genetic make-up of disc cells to produce a desired gene product may provide prolonged supply of a beneficial agent to the discal matrix. Recent advances in molecular genetics have cultivated techniques to readily insert genetic elements (DNA) into almost any type of target cell. These genetic elements typically consist of the gene encoding the desired product and a control element that modulates the expression of the respective gene. Two strategies can be used to achieve expression of the desired gene at the target site. Direct, or in vivo gene therapy is the direct introduction of the gene of interest into resident cells in situ (Figure 8.1), whereas indirect, or ex vivo gene therapy requires the removal of target cells, introduction of the gene of interest in vitro followed by implantation of the transformed cells (Figure 8.2) [13]. A carrier or vector can be used to optimize uptake of the desired genetic material, which usually is in the form of pure "naked" DNA. Viral vectors are attractive carrier options because they are very efficient transporters of genetic material, are able to enter mammalian cells, and take over DNA replication and the protein expression machinery. Several engineered viruses are available for the purposes of gene therapy whose original viral genome has been removed or inactivated and, in addition, are modified to not replicate nor exhibit their pathogenicity. These viruses vary in their ability to integrate the transferred DNA into the host cell genome, to invade dividing or nondividing cells, and in their infection efficiency. The properties of the IVD and its cells mandate that the virus of choice efficiently infects nondividing, quiescent cells. Furthermore, the reduced cell density within the disc might hamper adequate infection of a sufficient proportion of the disc cells. On the other hand, the avascular and contained IVD might provide an advantageous environment to achieve high concentrations of the injected viral vector, leading to improved efficiency of the infection process while also lessening the danger of an immune response against viral proteins.

Studies have demonstrated that adenoviral vectors have been able to efficiently transform cells of various species' IVDs. The main disadvantage of adenoviral vector's mechanism is the activation of innate and adaptive parts of the patient's immune system when the vector is applied in vivo. Lattermann et al. [14] recently tested an adeno-associated viral vector (AAV) for its applicability to degenerative discs as a potential strategy to overcome this potentially lethal complication. The authors found that AAV was able to efficiently transduce human disc cells in vitro and rabbit disc cells in vivo. Although AAV induces a humoral immune response, no significant cellular immune response, as is seen with adenoviral vectors, was observed. Interestingly, despite the observed humoral immune response, significant transgene expression was observed in the pre-exposed animals [14]. These findings suggest that AAV may offer a valuable and safer alternative to adenoviral vectors in the future. Although these studies support the feasibility of direct gene therapy using viral vectors to target disc cells, the question of the delivered gene and therefore expressed gene product remains open. Nishida et al. [13] initially studied the delivery of an exogenous therapeutic gene in vivo using an adenoviral vector carrying the gene for TGF-β1 to modify cells of rabbit IVD. The authors observed significant increases of TGF-β1 and proteoglycan production in the injected disc, suggesting the feasibility of direct gene therapy to treat IVD degeneration [13]. LMP-1 is another potentially beneficial gene factor that has been shown to increase the disc cell production of bone morphogenetic proteins (BMPs) and proteoglycans in vitro [15]. In vivo studies involving the intradiscal injection of rabbit discs showed increased expression of the anabolic cytokines BMP-2 and BMP-7 mRNA and also led to increased production of aggrecan mRNA [15]. These data suggest that LMP-1 is also a beneficial factor that could be applied to intradiscal gene therapeutic agents for disc degeneration. Sox9, on the other hand, does not affect the proteoglycan content of the IVD cellular matrix. Sox9 is a gene transcription factor responsible for the synthesis of type II collagen and its transfer into cells from degenerated human discs resulted in increased production of type II collagen [16]. Injection of a Sox9 carrying adenoviral vector into traumatized rabbit discs resulted in preservation of the histological appearance as seen in healthy discs, whereas the injured control discs displayed degenerative changes [16]. Increased production of both proteoglycan and collagen type II seems to be able to prevent disc degeneration in vivo thereby offering multiple potentially therapeutic options. Although the increased production of a single gene product seems to result in the transformation of disc cells, a combination of related gene-producing factors may prove synergistic and more physiologic. Indeed, Moon et al. [17] have already reported that the combined transfer of TGF-β1, IGF-1, and BMP-2 amplified protein synthesis.

An opposite approach as a potential alternative to the use of anabolic factors to induce disc cell production of matrix components is the application of anticatabolic

factors. Inhibition of catabolic activity would maintain or increase the content of the respective matrix component by slowing down its degradation without the need to force the disc cells to increase synthesis rates. Wallach et al. [18] recently published an in vitro study in which an adenoviral vector introduced the gene encoding for tissue inhibitor of metalloproteinase-1 (TIMP-1) into disc cells isolated from degenerated human IVD. Gene delivery of TIMP-1 increased the proteoglycan content in the disc cell cultures, suggesting that the anticatabolic approach may be a potentially promising strategy for gene therapy of degenerative discs.

Despite the obvious potential, gene therapeutic approaches to the human IVD will be challenged by the suboptimal and eventually toxic microenvironment inside the severely degenerated disc. It is questionable if the remaining compromised IVD cells in the degenerated disc will be able to produce reasonable amounts of gene-induced growth factors over extended periods of time. Furthermore, one can argue that it is unlikely that the existing starving cells are able to properly respond and produce an improved matrix even if the production of the respective gene product is achieved.

AUTOLOGOUS IMPLANTATION OF CULTIVATED, MODIFIED CELLS

Degenerated discs could be treated by supplementing the deserted matrix with in vitro cells that have been removed, cultivated, and modified. Autologous cells are ideal because their utilization makes the potential immunological complications a moot issue. Autologous cells compatible with disc tissue have to be harvested, expanded in vitro, and subsequently implanted into the symptomatic disc. Once the cells have been removed and cultivated in vitro, indirect gene therapy can be performed via genetic modification of the withdrawn cells and/or tissue engineering via seeding of the cells in supporting biomaterials before implantation into the symptomatic degenerated IVD (Figure 8.2). The combination of these techniques may improve efficiency by improving cell survival or enhancing the biosynthetic activity of the implanted cells. Genetic modification of cultivated cells in vitro is technically similar to the previously described approaches.

For several reasons, it is extremely challenging to obtain suitable and sufficient numbers of target cells from IVD tissue. Removal of nucleus pulposus cells requires opening of the annulus fibrosus to gain access, almost certainly causing annular damage. In addition to the very restricted accessibility, the very low cell density in degenerated discs will further complicate the acquisition of ample usable cells for successful in vitro cultivation. Therefore, only a limited number of scenarios are conceivable that would allow the withdrawal of sufficient cells from the disc to perform a disc cell–based approach without accelerating the disc's degeneration. Withdrawal of herniated disc material represents one scenario that would facilitate the removal of sufficient disc cells for in vitro cultivation. The direct insult as well as the likely accelerated degenerative process of the surgical level post microdiscectomy predisposes that segment to discogenic LBP postoperatively. This increased LBP risk may justify cell implantation to prevent postoperative acute and/or chronic discogenic LBP. In addition, cell-based approaches could be used to prevent the accelerated degeneration of discs adjacent to an interbody fusion level (Figure 8.5). The disc material removed during the fusion surgery could be used as source of cells to treat the adjacent disc. However, this would imply an intervention at an asymptomatic nondegenerated disc that only has the potential to degenerate in the future and is therefore questionable. Currently, autologous disc cell transplantation is limited to few clinical scenarios but has the potential to become a useful approach within these limitations.

The most direct approach to support a degenerated disc by autologous cells would be injecting a suspension of ex vivo proliferated disc cells into the degenerated disc [19]. Autologous disc cells can be prepared for transplantation using three-dimensional cultivation systems. Maldonado et al.'s initial work and subsequent investigations demonstrated that cultivation of disc cells in three-dimensional constructs conserved the native phenotype, as demonstrated by the synthesis of matrix components similar to those observed in native discs [20,21]. The feasibility of experimental strategies have been evaluated in vivo. Gruber et al. [22] in a study using a sand rat-based model, implanted autologous disc cells, expanded in routine monolayer cultures and seeded into a three-dimensional scaffold, to a hollowed cavity created in an IVD. At 33 weeks post implant, no giant cell response was observed and the cells showed an appropriate morphology. In addition, no abnormality in the cell-surrounding matrix was observed, suggesting appropriate survival of the implanted cells during the analyzed time period. It appears that autologous

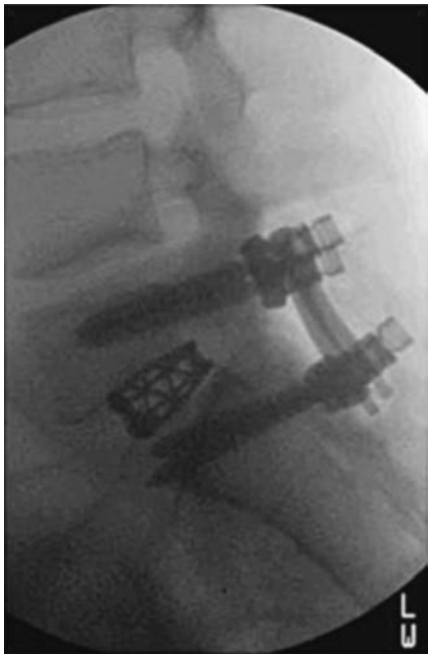

Figure 8.5 Lateral plain x-ray demonstrating adjacent level loss of disc height and degenerative changes at L4–5 status post-instrumented posterior lumbar interbody fusion at L5-S1.

disc cell implantation can be successful, although technically challenging. Yet, because of the immediate implantation after the seeding of the scaffold, the disc cells do not have the time to synthesize appropriate amounts of matrix before encountering the adverse environment within the disc. Cultivation of the disc cells in a three-dimensional system before implantation may improve the chances for survival in the hostile environment of the degenerated disc. Sato et al. evaluated this strategy by using a nonimmunogenic atelocollagen scaffold to seed and cultivate annulus fibrosus cells. Compared to cells grown in monolayers, this process demonstrated an increased ability to express type II collagen mRNA and deposited more type II collagen and proteoglycan. Atelocollagen scaffolds seeded with annulus fibrosus cells have been allografted into the lacunae of recipient rabbits after laser discectomy of the nucleus pulposus [23]. Implantation resulted in a significant prevention of the narrowing of the IVD space at 12 weeks post implantation compared to the nucleotomized control animals. Histological analysis also showed that the allografted cells were viable, proliferated, and produced a hyaline-like matrix in the disc tissue of the recipients. Although the rabbit model does not appropriately simulate the mechanical forces experienced by implanted disc cell in human IVDs, it is conceivable that cells surrounded by their own matrix might withstand mechanical forces with greater resiliency. A shortcoming of autologous implantation strategies is the inability to address acute shortage of nutrients experienced by the cells after implantation into the degenerated disc. Considering that the nutrient supply is hardly sufficient for the original disc cells, it is questionable whether the additional cells will survive for a prolonged time span likely required in order to provide a sustained structural improvement of the disc.

IMPLANTATION OF MESENCHYMAL STEM CELLS

Adult mesenchymal stem cells (MSCs) are uncommitted pluripotent stem cells that are found in several tissues such as skeletal muscle, bone marrow, synovial membranes, and the dermis [24,25]. MSCs are of high plasticity and have a high capacity for multilineage differentiation. Members of the BMP family of growth factors have been used so far to induce differentiation of MSCs into chondrocytes [26]. However, because BMPs are not exclusively inducing cartilage differentiation, their expression needs to be carefully timed and modulated to avoid the sequent generation of osseous structures. To overcome this problem, signal transduction and transcription factors, such as members of the Sox family and the Brachyury factor, that exclusively induce cartilage differentiation, have been tested and demonstrated with encouraging results [27,28]. It has been found that cocultivation of MSCs with disc cells might be sufficient to induce a disc cell–like phenotype in MSCs [29,30]. Although these data originate from in vitro experiments, it might be conceivable that MSCs would also differentiate in vivo after injection into the disc. Besides the cocultivation with disc cells, using three-dimensional cultivating systems to cultivate MSCs appears to be sufficient to induce a nucleus pulposus–like phenotype [31]. Implantation of collagen-gel-embedded MSCs into artificially degenerated rabbit discs resulted in preserved nuclear and annular structures, prevention of proteoglycan decrease from the nucleus pulposus, and increased disc height [32,33]. The implanted cells were able to survive and express genetic markers typical for nucleus pulposus cells. Similar findings were also observed after injection of a bone-derived MSC suspension into rabbit discs and injection of gel-embedded MSCs into rat coccygeal discs [34,35].

The use of MSCs provides a new and exciting approach to biologically treat disc degeneration supported by encouraging initial results. The comparably easy access to autologous MSCs overcomes one of the major downsides of conventional approaches. The high expand-ability of MSCs together with the relative easiness to harvest the cells makes this approach attractive. However, continued and extended studies are required to assess the structure of the newly synthesized matrix and its biomechanical properties and prove its value under the mechanical loads the functioning spine must bear.

CONCLUSIONS

The mechanics of human gait confer constant and multiplanar loads to the IVD. The progressive structural alterations to the IVD that occur in continuum with spine segmental degeneration can be associated with clinical sequelae. The functions of the disc require a mechanically stable structure with a highly specialized cellular matrix to afford the needed flexibility and physical strength required by the spine. The known avascularity of the adult IVD restricts nutrient supply to diffusion and therefore poses a major challenge for the prolonged maintenance of the discal matrix by its cellular components. The previously mentioned mechanical stressors combined with the known inadequate nutrition eventually creates a toxic environment, resulting in progressive destruction of the matrix cellular structure and simultaneous extensive decay of the matrix. These properties and its alterations during degeneration define and limit the techniques applicable to biologically repair degenerated IVD. This might indicate that the clinical application of intradiscal biologic agents to regenerate the structurally compromised IVD is in the distant future. Recent studies evaluating various biological approaches to maintain and improve the structurally compromised IVD provide real leads into the exciting potentials of these novel new treatments. Further, basic science and clinical experiments both in vivo and in vitro are required to bridge the gap between scientific potential and clinical realities.

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9 Diagnostic Imaging of Painful Lumbar Facet Joints

Matthew Smuck and Divya Agrawal

INTRODUCTION

In 1911, an American orthopedic surgeon, Joel E. Goldthwait, first suggested lumbar facet (zygapophyseal) joint involvement in low back pain [1]. In the century that followed, facet joint anatomy and biomechanics were defined. Meanwhile, skepticism about their capacity to cause pain continued, quelled eventually by the discovery of facet joint nociceptors [2] and the creation of de novo back pain by intra-articular injection of irritants [3]. Despite these advances, identifying facet joint pain proved to be a clinical challenge. Thus, every advance in spine imaging has spurred investigation into the value of newly recognized radiographic abnormalities, only to discover a high prevalence of abnormalities in asymptomatic volunteers. As a result, diagnostic injections (see Chapter 10) are currently the preferred method to confirm a facet joint as the source of lumbar pain. Also, these injections have provided a tool for the investigation of the role of new imaging techniques in the evaluation and treatment of lumbar facet joint pain.

FACET JOINT ANATOMY AND BIOMECHANICS

The facet joints are diarthrodial synovial joints composed of a fluid-filled joint space bordered by hyaline cartilage over subchondral bone, surrounded by a synovial membrane and a fibrous joint capsule. Invaginations of the joint capsule form menisci that fill voids and aid load distribution [4]. Articular entrapment of these menisci was once considered a potential source of pain; however, this condition is rarely seen in people with axial pain [5,6].

Microscopically, the facet joint capsule has a dense outer layer composed of parallel bundles of collagen and an inner layer of elastic fibers [7]. The hyaline joint cartilage extracellular matrix is made primarily of two macromolecules—collagen and aggrecan. The most abundant of the two, collagen, forms a highly organized structure that provides tensile and shear strength. Aggrecan is a large proteoglycan containing numerous charged glycosaminoglycan (GAG) molecules and provides compressive strength [8]. Because of the importance of these two macromolecules to the function of cartilage, emerging methods of cartilage imaging have focused on their interrogation [9].

Between two vertebrae, load transmission is shared by the intervertebral disc anteriorly and by the two segmental facet joints posteriorly [10]. The amount of load transmitted to the facet joints is dependent on posture, segmental alignment, and the degree of degeneration in the surrounding tissues [11]. Between 5% and 25% of segmental load is typically borne by the facet joints, increasing in extension [12]. Degenerative states can further increase facet load. Segmental disc degeneration with loss of disc height causes up to a fivefold increase in facet load in standing postures [13], to nearly 50% of the total segmental load [12,14].

The lumbar facet joints limit axial rotation and they stabilize the motion segment to prevent translation and dislocation during flexion and extension [15]. The forward shear forces acting upon the facet joints increase during flexion and are greater in the lower spine segments because of the higher weight and longer leverage of the body above. The increased sagittal orientation of the lower lumbar facet joints may be an adaptation to these forces [16].

Sometimes, there is a difference between right and left facet joint orientation at a single segment. This is called facet joint *tropism*. Initially linked to degenerative disk changes [17], facet joint tropism is known to play a role in facet degeneration and degenerative spondylolisthesis [18,19]. Patients with degenerative spondylolisthesis have statistically significant increases in sagittal facet joint orientation and in facet joint tropism relative to controls [18,19]. Also, segments with facet joint tropism demonstrate more prominent osteoarthritis in the joint with greater sagittal orientation [19] (Figure 9.1). It remains unknown if the increased sagittal joint orientation observed in degenerative joints is a factor predisposing to the development of osteoarthritis, or if it is a result of remodeling that occurs in osteoarthritis. A longitudinal study of facet joint morphology with aging may determine which comes first, but such findings have not yet been produced. Attempting to find answers, researchers have looked for a correlation between increased age and sagittal facet joint orientation finding mixed results [20,21].

FACET JOINT PAIN

In patients with chronic low back pain, lumbar facet joint pain is uncovered with greater frequency than other potential sources of pain [22,23]. Facet joint pain has

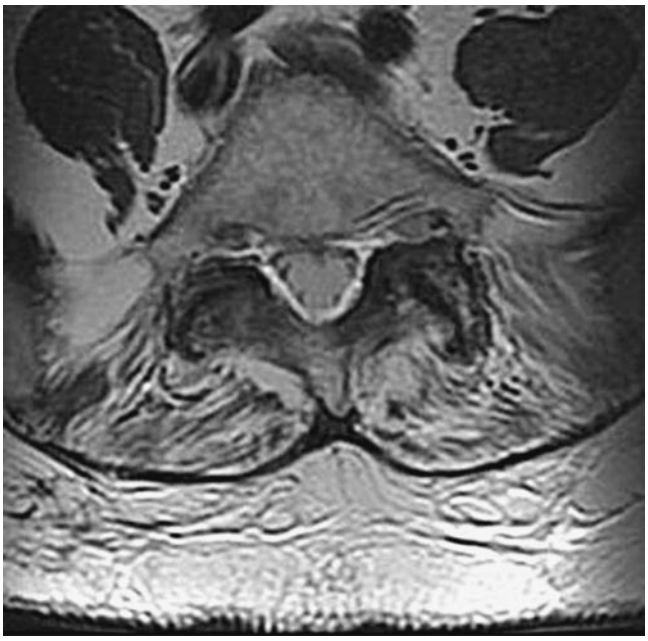

Figure 9.1 Axial T2 MRI at L5-S1. Notice the facet joint tropism with the left L5-S1 facet joint aligned closer to the sagittal plane than the right. As expected, the joint with greater sagittal orientation demonstrates more advanced degeneration including joint-space irregularity, subchondral cysts, osteophytes, and capsular thickening.

many potential causes. Macrotrauma, infection, pseudogout, synovial impingement, villonodular synovitis, inflammatory arthritides, and osteoarthritis to name a few [24–27]. Although some of these occur more often than others, osteoarthritis is by far the most common.

The mechanisms of pain associated with synovial joint degeneration are complex. Several factors linked to pain in other joints have been uncovered in the facet joints including: capsular and synovial nociceptors [2,28], cartilage erosion channels along with substance P innervation of the subchondral bone [29], and inflammatory cytokines in the cartilage and synovium [30]. Still, many questions regarding the mechanisms behind synovial joint pain remain unanswered. Osteoarthritis affects joints of the appendicular and axial skeleton, and causes pain that is not directly related to the degree of degeneration [31]. On the other hand, not all patients with joint pain will have abnormalities on standard imaging. This was demonstrated in a study of surgical specimens from patients who underwent segmental fusion for low back pain. All these patients experienced temporary pain relief with facet joint injections but had no x-ray evidence of facet joint abnormalities. Histological evaluation of the removed facet joints revealed focal cartilage necrosis without advanced alterations of osteoarthritis [32].

Theories on the mechanisms of pain caused by joint degeneration range from mechanical alterations to vascular changes and molecular signaling. Only recently have imaging technologies emerged to allow investigation of the latter two. This chapter will discuss some of these emerging tools, and will detail the role of existing imaging techniques when considering facet joint interventions.

PREVALENCE

The reported prevalence of facet joint abnormalities differ depending on the age of the population studied, their symptoms, the method used to view the joints, and the definition of abnormal. According to one large cadaver study, facet joint osteoarthritis begins early with nearly 60% of adults showing early signs of cartilage degeneration by the time they reach age 30 [33]. Rates steadily increase with age until 60 years when signs of degeneration become ubiquitous (Figure 9.2) [33]. Since early signs of facet joint degeneration are not evident on routine imaging, radiographic studies find a lower prevalence of facet degeneration than anatomic dissections. For instance, a cross-sectional study using computed tomography (CT) found evidence of degeneration in 25% in subjects under 40 years old, increasing up to 89% in subjects 60 years and older (Table 9.1) [34]. Studies agree that degenerative facet joint changes are most common at L4–5, followed by either L3–4 [33], or L5-S1 [34] (Table 9.2).

Initial studies suggested that facet degeneration is less common in asymptomatic populations, observed in approximately 10% of CT and magnetic resonance imaging (MRI) studies [35–37]. One MRI study of 60 asymptomatic volunteers between ages 20 and 50 years demonstrated no evidence of severe facet joint osteoarthritis leading the authors to suggest that severe osteoarthritis may play a role in low back pain since it was not observed in asymptomatic volunteers [37]. However, a more recent cross-sectional study showed no difference in the prevalence or severity of facet joint arthropathy observed in CT scans of age-matched symptomatic and asymptomatic participants [34].

MRI has proven useful to detect certain changes associated with more active facet joint disease. For instance, facet synovial cysts are observed in 9.5% of the MRI studies in a symptomatic population [38]. Depending on the age and symptoms of the study cohort, facet joint effusions are observed in 14% to 55% of those studied, most often at L4–5, followed by L3–4, then L5-S1 (Figure 9.3) [38–41]. Facet joint effusions are associated with higher grades of facet joint degeneration [38] and signify a substantially greater risk of dynamic instability at the involved level [40,42]. MRI using fat-saturation techniques has improved detection of edema in the bone and tissues surrounding facet joints. This is seen in 14% to 41% of MRIs of patients with low back pain, most often at L4–5, followed by L5-S1, then L3–4 (Figure 9.4) [43,44].

Some factors are known to increase the risk of facet joint degeneration. Facet joint tropism increases the risk of degeneration on the side with more sagittal facet joint orientation (Figure 9.1). Facet joint tropism has an incidence of between 20% and 30% among the general population [45]. Participation in some sports appears to increase this risk [46]. Other risk factors for facet joint degeneration include advanced age, genetic predisposition, segmental intervertebral disk degeneration, and malalignments such as hyperlordosis and scoliosis [15].

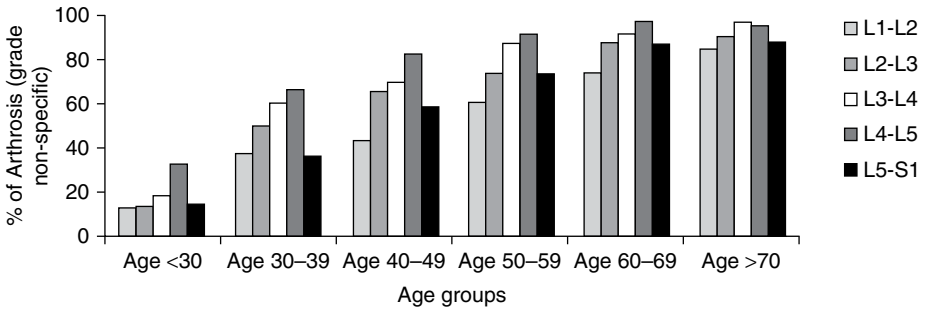

Figure 9.2 A study of the spines of 647 cadavers obtained between 1893 and 1938 showing the prevalence of facet arthropathy observed at each lumbar level based on the age of the cadavers at the time of death. With permission from [33].

Table 9.1 A Cross-Sectional, Community-Based Study Using Lumbar CT Demonstrates a Steady Increase in the Prevalence of Facet Joint Arthropathy to the Seventh Decade of Life

Age Group	Males		Females		Total Sample		X-Test (Males vs. Females by Age Group)
	N	%	N	%	N	%	
<40	5	31.3	1	12.5	6	24.0	$P = 0.6214$
40–49	15	50.0	6	35.3	21	44.7	$P = 0.3299$
50–59	22	66.7	27	84.4	49	74.2	$P = 0.1401$
60–69	16	88.9	17	89.5	33	89.2	$P = 1.0000$
≥70	4	57.1	5	83.3	9	69.2	$P = 0.2045$
X-test (age groups)	$P = 0.0070$		$P = 0.0001$		$P = 0.0001$		

Statistically significant at level $P < 0.05$.
Adapted from Kalichman et al. [34].

Table 9.2 A Cross-Sectional, Community-Based Study Using Lumbar CT Demonstrates the Prevalence of Facet Joint Arthropathy in Males and Females at Different Lumbar Levels

Spinal Level	Males		Females		Total Sample		X-Test (Males vs. Females by Spinal Level)
	N	%	N	%	N	%	
L2-L3	17	16.50	11	13.75	28	15.05	$P = 0.6076$
L3-L4	27	26.21	29	36.25	56	30.60	$P = 0.1439$
L4-L5	39	38.24	43	53.75	82	45.05	$P = 0.0368$
L5-S1	32	32.32	36	45.57	68	38.2	$P = 1.0707$
X-test (spinal levels)	$P = 0.0045$		$P < 0.0001$		$P < 0.0001$		

Statistically significant at level $P < 0.05$.
Adapted from Kalichman et al. [34].

RADIOGRAPHIC FINDINGS

Degenerative changes in the facet joints mimic that of other synovial joints. The process typically begins with focal, then diffuse articular cartilage damage leading to more advanced degenerative changes, joint-space narrowing, subchondral bone erosions and cysts, osteophyte formation, joint subluxation, capsule degradation, ligament thickening, joint effusion, and possibly synovial cyst formation. These later stages of degeneration can be observed by traditional radiography, however, many of the earlier changes cannot be.

Radiography

Plain radiographs are often used as a preliminary tool to screen for facet arthropathy but have limited diagnostic utility. Classic findings on plain films indicating facet arthropathy include joint-space narrowing, subchondral sclerosis, intra-articular gas, and hyperostosis with osteophyte formation. With advanced degenerative changes, the joint space may be widened because of facet joint subluxation leading to degenerative spondylolisthesis [47]. Disc-space narrowing can also be visualized with plain radiographs. This is an important finding because, the

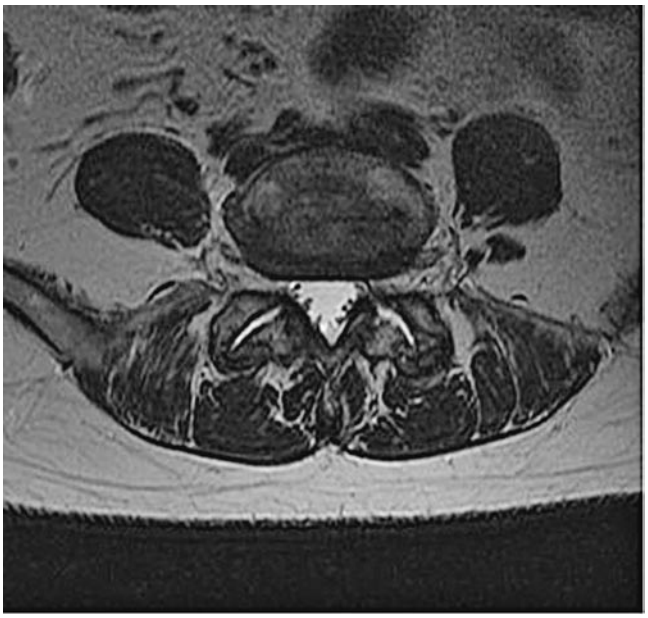

Figure 9.3 Axial T2 MRI at L5-S1 demonstrating bilateral facet joint effusions. The high signal intensity along the joint lines is an indication of increased fluid. This finding is less common than other markers of joint degeneration and appears in significantly greater numbers on the MRIs of symptomatic subjects compared to asymptomatic subjects [41]. In addition, finding a facet joint effusion on MRI signifies a 50% risk of dynamic instability at the involved intervertebral segment [40].

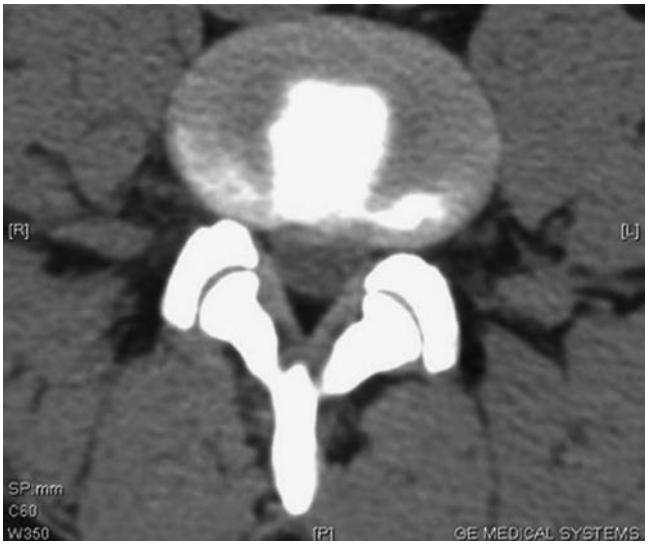

Figure 9.4 Sagittal STIR MRI demonstrating L5-S1 facet joint edema. The bright signal displayed on these fat-saturated images exposes the inflammation present within the joint, the bone and the adjacent soft tissues (white arrow), all findings that were not evident on the standard T2 images.

degree of disc degeneration correlated significantly with the degree of facet arthropathy observed on the images of a group of patients with radicular and nonradicular low back pain [48]. Pathria et al. defined a grading system for facet arthropathy based on the assessment of oblique radiographs (Table 9.3) [49]. Despite the limitations in interobserver agreement ($\kappa = 0.26$), it remains a widely used method of grading facet arthropathy on plain radiographs.

Table 9.3 Grading System for Facet Osteoarthritis Based on Assessment of Oblique Radiographs

Grade	X-Ray Findings
Grade 0	Normal
Grade 1	Narrowing of facet joint space (mild osteoarthritis)
Grade 2	Narrowing plus sclerosis or hypertrophy (moderate osteoarthritis)
Grade 3	Narrowing, sclerosis, and osteophytes (severe osteoarthritis)

Adapted from Pathria et al. [49].

Because of the curved configuration of the lumbar facet articulations, conventional radiography can only visualize the small portion of each joint that is parallel to the x-ray beam. The oblique view is optimal to visualize the lumbar facets because of their generally oblique orientation [49]. Studies have found that oblique views are most accurate in distinguishing the presence from the absence of facet joint arthrosis. Still sensitivity and specificity remain poor, 55% and 69%, respectively, mostly because of the inability to uncover mild pathology [49]. Plain radiographs perform better when evaluating more advanced degenerative changes. For instance, when distinguishing absent or mild disease from moderate or severe disease, specificity improves to 94% whereas sensitivity remains low [48]. Therefore, plain radiographs are most useful in demonstrating late changes associated with severe disease. In addition, plain radiographs have been found to underestimate the degree of involvement when compared to CT [48]. Although x-ray has no demonstrated value in predicting response to facet joint interventions, in the form of mobile C-arm fluoroscopy it is the tool of choice for accurate image guidance during these interventions.

Computed Tomography

In comparison to plain radiographs, CT scanning is significantly more sensitive in detecting both the presence and degree of facet arthropathy [49]. CT is capable of imaging the entire joint and provides high contrast between bony structures and the surrounding soft tissue [50]. Locations of osteophytes are better visualized including those causing adjacent nerve root compression. Synovial and ligamentum flavum hypertrophy can also be seen and may appear thickened, buckled, or calcified. Intra-articular gas is observed as an area of very low absorption within the facet joint [50]. Intraspinal juxtafacet cysts typically appear as a rounded cystic mass isointense to cerebrospinal fluid with possible calcification or gas formation within the cyst [51]. Hemorrhage into the cyst has also been reported, resulting in increased attenuation on CT [52].

Pathria et al. described a grading system for facet arthropathy similar to their plain radiograph-based

Table 9.4 Grading System for Facet Osteoarthritis Based on Axial CT and MRI Imaging

Grade	CT or MRI Findings
Grade 0	Normal facet joint space (2–4 mm width)
Grade 1	Narrowing of facet joint space (<2 mm) and/or small osteophytes and/or mild hypertrophy of the articular process
Grade 2	Narrowing and/or moderate osteophytes and/or moderate articular process hypertrophy and/or mild subarticular bone erosions
Grade 3	Narrowing and/or large osteophytes and/or severe articular process hypertrophy and/or severe subarticular bone erosions and/or subchondral cysts

Adapted from Weishaupt et al. [53].

Table 9.5 Grading System for Facet Synovitis as Seen on MRI Using Fat-Saturation Techniques

Grade	Fat-Saturated MRI Findings
Grade 0	No signal abnormality
Grade 1	Signal abnormality confined to joint capsule
Grade 2	Periarticular signal abnormality involving less than 50% of joint perimeter
Grade 3	Periarticular signal abnormality involving more than 50% of joint perimeter
Grade 4	Grade 3 with extension of signal abnormality into the intervertebral foramen, ligamentum flavum, pedicle, transverse process, or vertebral body

Adapted from Czervionke and Fenton [44].

system (Table 9.3), with improved interobserver agreement ($\kappa = 0.46$) and 95% agreement within one severity grade [49]. Weishaupt et al. described a similar grading system (Table 9.4) with additional improvement in the interobserver agreement ($\kappa = 0.60$) [53].

Magnetic Resonance Imaging

MRI underestimates the severity of facet arthropathy when compared to CT [47]. MRI is less sensitive in depicting the bony cortex margin, and cartilage thinning cannot be measured accurately because of signal artifact inherent to MRI [54]. Still, studies in patients with chronic low back pain have shown that the sensitivity and specificity of MRI is nearly as good as CT in detecting facet degeneration [48,53].

Because of its better spatial resolution and contrast with fat, T1-weighted sequencing best depicts the anatomy of the facet joints, ligamentum flavum, neural foramina, and nerve roots, particularly in the axial view [55]. Joint-space narrowing, or widening with synovial hyperplasia can also be visualized. Articular cartilage is seen as intermediate signal, although the separation of the two surfaces is rarely seen unless an effusion is present. Subchondral bone irregularities are not as clearly seen on MRI as on CT.

Soft tissue abnormalities are more easily observed on MRI. The ligamentum flavum can be displaced by osteophytes and show reactive thickening, seen as low signal intensity on T1 and T2. Facet cysts are best seen on axial T2-weighted images and appear with a hypointense wall with hyperintense contents. The majority are contiguous to the joint and extend anteromedially. T2 is also useful to detect facet joint effusions because of increased fluid in the facet joint (Figure 9.3). Weishaupt et al. described a grading system for facet arthropathy based on MRI (Table 9.4), with good interobserver agreement ($\kappa = 0.41$) and 95% agreement within one severity grade [53].

MRI with contrast enhancement is both sensitive and specific in diagnosing facet joint septic arthritis in its earliest stages [56]. Fat-saturation techniques can increase the visibility of certain signal changes and have been evaluated in the diagnosis of noninfectious facet synovitis using both T2-weighted (Figure 9.3) and postcontrast T1-weighted (Figure 9.4) MRIs [44]. A retrospective study of all lumbar MRIs completed during 1 month at the Mayo Clinic found a 41% prevalence of facet synovitis on fat-saturated images [44]. In patients with unilateral, single-level facet synovitis they found a 100% correlation between the side of the facet synovitis and the side of symptoms. Czervionke and Fenton described a grading system for facet synovitis observed using fat-saturated MR techniques (Table 9.5) [44].

Single Photon Emission Computerized Tomography

Bone scans have long been used to uncover occult skeletal lesions, and remain a good screening tool. However, anatomic precision is limited by poor spatial resolution. The development of single photon emission computerized tomography (SPECT) improved spatial resolution and is thus preferred over traditional bone scans for the localization of spine lesions. Although CT and MRI provide better anatomic details, SPECT has two substantial advantages. First, SPECT provides a whole body survey and can uncover unsuspected causes of a patient's complaints [57]. Second, SPECT provides a look at tissue function and enables a distinction between degenerative joints with active disease and those without. This has unique significance in the spine where it is often difficult to distinguish the source of a patient's low back pain because of overlapping pain patterns from nearby structures, poor specificity of the physical examination, and a high prevalence of common degenerative abnormalities. In fact, facet joints with increased activity on SPECT are often not the joints with the greatest amount of degeneration [58].

Increased SPECT activity has also been shown to correlate with MRI findings associated with more active facet joint disease [59]. A poor correlation was observed between facet hypertrophy on MRI and positive uptake on SPECT, suggesting a protective component to joint hypertrophy. On the other hand, increased facet joint fluid signal on MRI was highly predictive of a positive facet joint on SPECT [59].

Emerging Concepts in Spine Imaging

Despite the wide use of plain radiographs, CT, MRI, and to a lesser extent SPECT, studies so far have demonstrated

limitations in the reliability of these methods to determine a specific source of back pain [11]. The current gold standard for diagnosing lumbar facet pain is by positive response to local anesthetic blocks. Still, the search continues for a less invasive and less costly method to accurately diagnose facet joint pain.

One recent development involves the digital fusion of images from CT and SPECT, combining the specificity and anatomic detail of CT with the sensitivity and functional tissue interrogation of SPECT. Technical advances allow for both the CT and nuclear medicine components to be performed simultaneously by the same scanner. Case reports have demonstrated the localization of axial pain to the left C1-C2 joint and to bilateral L5-S1 facet joints, leading to successful interventions [60]. The authors suggest that this technology has the potential to improve the accuracy and efficacy of spine injections and to decrease the number of diagnostic blocks. Still, further prospective studies are needed to assess its clinical utility, sensitivity, and specificity, as well as the cost and risks involved.

There are several emerging MRI techniques designed to improve the evaluation of early joint degeneration. These include T1rho (T1r), Sodium MR, and delayed gadolinium-enhanced magnetic resonance imaging of cartilage (dGEMRIC). T1r signal changes correlate strongly with proteoglycan depletion and may be a useful tool for assessing early cartilage degeneration [61,62]. Sodium MRI and dGEMRIC reveal differences in concentrations of charged ions in the interstitial fluid and are sensitive to changes in the GAG content of the cartilage extracellular matrix. These techniques may provide better insights into early osteoarthritis and lead to better methods of early intervention and prevention.

UTILITY OF IMAGING FOR FACET JOINT INTERVENTIONS

Radiography and CT

The bulk of research investigating a link between imaging and response to facet joint interventions was published in the 1980s using x-ray or CT (Table 9.6) [50,63–70]. On the basis of current standards, these studies contained several methodological limitations. They used various clinical criteria to select patients for facet joint injections. Some indiscriminately injected the lowest two lumbar segments. Most were small studies with poorly defined radiographic criteria and lacked independent observers to measure outcomes. Despite these flaws, more than half of these studies demonstrated a link between severity of imaging findings and outcomes [63–65,71]. On the other hand, the largest and only prospective study of the 1980s group did not find a correlation between x-ray abnormalities and response to lumbar intra-articular facet joint injections [66].

Debate about the value of imaging continued until the late 1990s when it became widely accepted that the only valid method to diagnose lumbar facet joint pain was by diagnostic blocks of the facet joints or nerves subserving the joints [72]. Later, few studies addressed the role of imaging prior to facet joint interventions. The main criticism of the 1980s studies became their lack of diagnostic facet joint blocks prior to intervention. This shortcoming was addressed by Schwarzer et al. using placebo controlled intra-articular blocks and by Cohen et al. using one-time medial branch nerve blocks [67,68]. The former study treated patients with intra-articular injections of anesthetic plus corticosteroid and found no relationship between response and CT findings [67]. The later study treated patients with medial branch radiofrequency neurotomies and similarly found no relationship between response and imaging with MRI [68].

The conclusion drawn from these studies is that imaging has no utility in guiding interventional treatment of the facet joints [72,73]. Combining these studies with the dearth of clinical findings that predict successful facet interventions [64,74], one can understand the reliance on diagnostic facet joint blocks. Still, current interventional methods of diagnosing facet joint pain, discussed in this chapter, are not without controversy [75], and the desire for a less invasive and more cost effective means of diagnosis has renewed interest into radiographic predictors of successful facet joint interventions.

Consider again the findings from the studies listed in Table 9.6. A correlation between imaging and outcome was observed in four of the six studies that selected patients based on a clinical suspicion of facet joint pain. Thus, it appears feasible that subgroups of patients exist that respond better to facet joint interventions, especially among those who combine a suspicious clinical presentation with objective radiographic evidence of facet joint disease. Interestingly, recent investigations using advanced imaging techniques lend support to this reinterpretation.

Magnetic Resonance Imaging

It is well established that common abnormalities on MRI are not specific to people with low back pain. Even in the setting of a first episode of back pain, MRI has failed to demonstrate new pathology compared to a presymptomatic baseline study [76]. In addition, to date MRI findings have not proven to be a good predictor of response to facet joint nerve blocks [68]. Advances in MRI interpretation and technologies may change this. For instance, facet joint effusions (Figure 9.3) are observed in a fraction of degenerative facet joints and appear in significantly greater numbers on the MRIs of subjects with nonradicular low back pain compared to asymptomatic subjects [41]. Fat-saturation techniques have allowed for better detection of skeletal inflammatory conditions. Freidrich et al. defined facet joint edema as increased signal intensity in the bone marrow and soft tissues surrounding the facet joints, observed on sagittal short tau inversion recovery (STIR) images (Figure 9.4) [43]. They studied scans of 145 symptomatic subjects and only 21 (14%) were found with the described edema. All 21 were contacted for a follow-up study and 9 consented. On the follow-up MRI, between 6 and 12 months after the initial scans, increased edema was observed in two, no change was observed in three, and reduced edema was observed in four. Interestingly, there was perfect correlation between an interval increase or decrease in facet joint edema and an increase or decrease in pain—a finding that was statistically significant. Of the

Table 9.6 Results from Nine Studies that Have Examined a Correlation Between Imaging With X-Ray, CT, or MR and Response to Lumbar Facet Joint Interventions

Year/Author	N[a]	Imaging	Subject Selection	Treatment	Correlation	Details
1980/Carrera [50]	10	CT	Clinical criteria for facet pain	IA − C + L	Yes	4/4 with arthropathy had relief vs. 0/6 with mild or no arthropathy
1981/Fairbank [69]	25	x-ray	Clinical criteria for facet pain	IA − L	No	All with pain < 3 months, nonresponders had significantly more pain below the knee
1984/Carrera [65]	63	CT	Clinical criteria for facet pain	IA − C + L	Yes	73% with arthropathy had relief vs. 13% of those without arthropathy
1986/Lewinnek [63]	18	x-ray or CT	clinical criteria for facet pain	IA − C + L	yes	13/14 with arthropathy responded vs. 1/4 without arthropathy
1988/Helbig [64]	20	x-ray	Clinical criteria for facet pain	IA − C + L	Yes	5/5 with severe arthropathy responded vs. 9/13 with mild and 1/2 without arthropathy
1988/Jackson [66]	390	x-ray	Clinical criteria for facet pain	IA − C + L	No	Injections done indiscriminately at L4–5 and L5-S1, only immediate effects measured (up to 4 hours)
1992/Revel [70]	40	x-ray and CT	Anyone with back pain	IA − L	No	Multiple clinical criteria examined to evaluate correlation with a positive response to blocks
1995/Schwarzer [67]	57	CT	IA − L & placebo control blocks	IA − L	No	CT scans were evaluated by an independent radiologist using a predetermined scoring system
2007/Cohen [68]	192	MRI	Single MBB	RFN	No	Number of subjects with MRI data and criteria for determining facet arthropathy not specified

Abbreviations: C, corticosteroids; IA, intra-articular injections; L, local anesthetics; MBB, medial branch nerve blocks; RFN, radiofrequency neurotomy.

[a] Number treated with imaging correlated to outcome.
Note that most of the early studies showed a positive correlation whereas more recent studies have not.

three patients with unchanged imaging, two reported no interval change in pain. This led the authors to suggest that in patients with low back pain sagittal STIR images detecting facet joint edema may be useful for planning facet joint interventions.

Other authors have described the ability of fat-saturated T2-weighted sequences and fat-saturated contrast-enhanced T1-weighted images to provide better visualization of potentially relevant degenerative alterations (Figure 9.5). Not only can these methods distinguish between active and inactive degenerative sites, they can also detect activity at sites that otherwise appear normal [77,78]. It remains to be determined how these and other emerging MRI technologies will affect selection of patients for facet joint interventions.

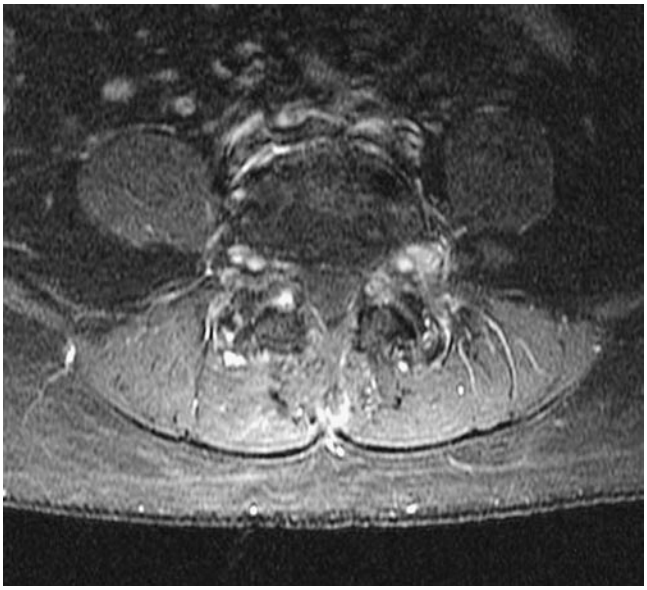

Figure 9.5 Postcontrast axial T1 fat-saturated MRI demonstrating bilateral L5-S1 facet joint edema. The bright signal on these images exposes the inflammation present within the joints, the bone, and the adjacent soft tissues extending into both the right and left neuroforamen.

Nuclear Medicine Imaging

Although SPECT abnormalities can be present in spines of normal volunteers and may appear in locations that do not correspond to a patient's pain, they do so less frequently than abnormalities on routine CT and MRI. Holder et al. demonstrated this in a retrospective study of 43 patients with low back pain suspected to be from the facet joints [57]. All patients had abnormalities on routine imaging by x-ray, CT, and/or MRI, and were considered candidates for facet

joint intervention. Of the 43 patients, only 19 (44%) demonstrated increased tracer accumulation in one or more facet joint, and only 9 (21%) had a positive facet joint finding on SPECT that correlated with his or her clinical symptoms. All nine of these patients were treated with intra-articular injection of anesthetic and corticosteroid and seven (78%) had a positive response based on their pain diaries. Five patients with negative SPECT findings were also treated with injections and only one (20%) responded. In addition, SPECT uncovered other causes of pain in some patients including hip arthritis, iliac fracture, ischial tendinitis, L2 vertebra metastasis, spondylolysis, rib fracture, and vertebral compression fracture.

Dolan et al. prospectively studied 58 patients who met their clinical criteria for facet joint pain [58]. Twenty-two (38%) had one or more positive facet joints on SPECT. Only 20% of the SPECT-positive facets showed signs of degenerative changes on x-ray. In total, 54 subjects were treated. Two were excluded because of technical problems during the facet joint injections, and two were excluded after developing radiculopathy from a disc herniation. Thus, 35/36 patients with negative SPECT findings received intra-articular injections of local anesthetic and corticosteroid into the joints underlying areas of tenderness, and 19/22 patients with positive SPECT findings received the same injections into the SPECT-positive facet joints without regard to areas of tenderness. Interestingly, only 3/19 had a site of tenderness on examination corresponding to the location of the increased uptake on SPECT. Statistically significant improvements in verbal pain scores were reported in the scan positive group only. Of those with positive scans, 94% reported improvement at 1 month and 79% at 3 months, compared to only 47% and 42% of the scan negative patients at the same follow-up periods. Differences at 6 months were not significant.

In a similar prospective study, 47 patients scheduled for facet joint injections were enrolled and divided into three groups based on SPECT findings [79]. All had prior radiographic evidence of facet joint degeneration and clinically suspected facet joint pain. SPECT examination was performed on 31 participants producing the first two study groups: 15 SPECT-positive patients and 16 SPECT-negative patients. The remaining 16 not examined by SPECT comprised the third group. The 15 SPECT-positive patients received injections of local anesthetic and corticosteroid into the positive joints only. All of the non-SPECT and SPECT-negative patients received injections at the facet joints requested by the referring physicians based on a clinical evaluation and prior imaging. The specialty and training of the physicians requesting the injections, and the specific criteria they used to select joints for injections were not specified. As in the previous studies, the group with positive SPECT findings did significantly better than the other groups. This time pain outcomes were based on the validated MODEMS spine survey [80]. At 1-month follow-up, 87% of the SPECT-positive patients improved compared to 13% of the SPECT-negative patients and 31% of the non-SPECT patients. The differences between groups remained statistically significant at 3 months, but not at 6 months. Of note, in the SPECT-positive group the total number of facet joint injections was lowered from the 60 originally planned to only 27 injections. As a result, SPECT not only improved outcomes but also reduced costs. The additional charges of SPECT were more than offset by reducing the number of injections resulting in a savings of more than $320 per patient.

Ackerman and Ahmad studied 46 patients with clinically suspected facet joint pain and positive facet joint SPECT findings [81]. Using a comparative effectiveness study design, participants were randomized equally between injections of local anesthetic and corticosteroid into the facet joints or on to the corresponding facet joints' medial branch nerves. Outcomes were measured using verbal pain scores and the Oswestry Disability Index. The intra-articular injection group demonstrated statistically greater improvements in pain and function leading the authors to conclude that facet joint injections are therapeutically more effective than medial branch nerve blocks in patients with back pain and positive facet joint SPECT findings.

Although these four studies suggest that SPECT can aid the treatment of patients with clinically suspected facet joint pain (Table 9.7) [57,58,79,81], a consensus has yet to be

Table 9.7 Results from Four Studies that Have Examined a Correlation Between SPECT Findings and Response to Lumbar Facet Joint Injections

Study [n]a	Design	SPECT	Randomization	Follow-up	Outcomes	Results
Holder [57]	Retrospective	9 − SPECT (+) 5 − SPECT (–)	Based on SPECT[b]	Not specified	Pain journal	Greater success in SPECT (+) (78% vs. 20%)
Dolan [58]	Prospective RCT	19 − SPECT (+) 35 − SPECT (–)	Based on SPECT*	1, 3 and 6 months	VPS MGQ	SPECT (+) significantly improved at 1 month and 3 months, not at 6 months
Pneumaticos [79]	Prospective RCT	15 − SPECT (+) 16 − SPECT (–) 16 − no SPECT	Based on SPECT*	1, 3 and 6 months	VAS pain	SPECT (+) significantly improved over each of the remaining groups at 1 month and 3 months, not at 6 months
Ackerman [81]	Prospective RCT	All SPECT (+)	23 − IA C + L 23 − MB C + L	3 months	VPS ODI	IA significantly better than MBB for gains in pain and function

Abbreviations: C, corticosteroids; IA, intra-articular injections; L, local anesthetics; MBB, medial branch nerve blocks; MGP, McGill questionnaire; RCT, randomized controlled trial; VAS, visual analog scale; VPS, verbal pain score (0–10).

[a] Number treated with imaging correlated to outcome.

[b] All treated with intra-articular injections of corticosteroids and local anesthetics.

achieved on when, how, or if SPECT should be used in the routine evaluation of chronic low back pain. In addition, SPECT has certain limitations. First, radiation exposure is high. Also, spatial resolution remains a problem, especially when attempting to differentiate between levels in the lower lumbar spine or in patients with severe degenerative deformity [57]. These limitations have led to the fusion of SPECT and CT imaging to combine the functional diagnostic information from SPECT with the anatomic details from CT. This technology has led to more specific interventions in some patients [60]. Finally, SPECT is limited to a single radioactive tracer. In this regard, positron emission tomography (PET) holds further promise because it allows for use of multiple tracers, including newly developed tracers. For instance, if biochemical markers of facet joint disease are uncovered that reliably predict the response to treatment, specific tracers can be developed to accurately identify these joints. Like SPECT, PET can be fused with CT to improve the anatomic details, and has proven able to uncover occult facet arthropathy [82]. At the time this chapter was written, studies on the utility of PET to aid facet joint interventions are entirely lacking.

CONCLUSION

Unlike pain from a large diarthrodial joints such as the knees, or from small superficial joints such as the metacarpophalangeal joints, discerning pain from the lumbar facet joints has proven a clinical challenge. Pain from the facet joints overlaps with other sources of pain, and physical examination is largely nonspecific. Thus, it is tempting to rely on imaging to determine the source of a patient's back pain. Unfortunately, the interpretation of spine imaging for those with back pain is fraught with limitations, mostly due to the high prevalence of abnormalities in asymptomatic volunteers. As a result, routine imaging is often unable to determine the exact source of axial pain. This has not stopped investigators from searching for features that can predict a patient's response to treatment. Although past studies of the link between imaging and response to facet joint interventions produced conflicting results, recent advancements in MRI and nuclear medicine imaging suggest there may be subgroups of responders that can be identified radiographically. Further investigation is required to determine the utility of these findings and their role in guiding facet joint interventions.

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10 Diagnostic Blockade of Symptomatic Lumbar Facet Joints

Venu Akuthota and Rachel Brakke Holman

INTRODUCTION

Pain originating from the lumbar zygapophysial joints (Z-joints), also called facet joints, is a well-recognized etiology of back pain. Estimates on the prevalence of facet joint-generated back pain range between 15% and 45% of those with low back pain [1]. One study showed a high prevalence of Z-joint osteoarthritis with rates as high as 59.6% in men and 66.7% in women [2]. The prevalence increases with age and is most common at the L4-L5 and L5-S1 levels. Eubanks et al. [3] found that 57% had evidence of facet joint arthrosis in cadavers of individuals in their third decade. This rate steadily rose to more than 80% in those older than 70 years of age.

Moreover, not all individuals with imaging evidence of facet joint arthrosis have back pain. Bogduk [4] states that the prevalence of facet-mediated chronic low back pain decreases to less than 10% if proper diagnosis is ascertained by "double-block paradigms" and likelihood ratios are calculated. Thus, confirmatory diagnostic tests of facet-mediated pain are important, especially when directing interventional or surgical care to the facet joints. Unfortunately, the literature has not borne out any specific findings on history or physical examination that can reliably pinpoint facet-mediated back pain from other sources. Many clinicians use exam findings such as pain with extension or rotational motions or focal paraspinal tenderness as suggestive of facet-mediated pain. However, Schwarzer et al. [5] found no specific clinical finding that could be correlated with those who responded positively to diagnostic facet blocks. Because history and physical examination clues for facet-mediated pain remain elusive, diagnostic blockades have emerged as the primary confirmatory diagnostic tests for facet-mediated pain.

DIAGNOSTIC VALIDITY

There are two primary interventional techniques commonly used for diagnosing the presence of facet-mediated back pain: (a) medial branch blocks (MBBs) or (b) intra-articular facet joint injections. These approaches utilize a short-acting local anesthetic to anesthetize or "block" pain originating from lumbar facet sources. MBBs are generally considered to be a more valid diagnostic test than facet intra-articular injections as there is more supportive literature for this approach [6–8]. MBBs have the additional advantage of anesthetizing both intra-articular and extra-articular sources of facet pain, whereas facet intra-articular injections only block sources of pain that arise from within the joint.

The primary basis for the diagnostic validity of lumbar MBBs is through a study by Kaplan et al. In this study, asymptomatic volunteers underwent facet capsular distension with contrast to provoke low back pain [8]. One week later, the authors found that anesthetizing the medial branches to the target facet joint followed by repeat capsular distension of the facet joint did not produce pain in eight of nine subjects. All of those who underwent sham (normal saline) MBBs experienced recurrence of pain with repeat facet capsular distension. In another study, the validity of lumbar MBB was thought to be strengthened because a lumbar facet pain diagnosis was sustained for 2 years, based on sustained treatment effect after successful treatments directed toward the lumbar facet joints [9]. Others have questioned the validity of MBBs and nearly all spinal diagnostics (including discography, magnetic resonance imaging, physical examination) based on the fact that no universally accepted standard exists to diagnose facet pain [10]. At minimum, MBB must be performed with extreme precision to be diagnostic and specific.

ANATOMY

Lumbar facet joint anatomy, including its innervation scheme, dictates lumbar Z-joint diagnostic techniques. The Z-joints are typical diarthrodial synovial joints surrounded by capsule and lined with synovium. The anterior portion of the facet joint capsule is actually the ligamentum flavum. The capsule extends both superiorly and inferiorly with recesses shaped like mushrooms. Capsular rents and synovial cysts can extend through the Z-joint capsule at various locations. Anatomy and these rents can explain the variety of contrast patterns that can occur with intra-articular Z-joint injections:

- Linear joint line pattern
- Capsular "dime" pattern
- Predominant filling of capsular recesses
- Capsular rents allowing flow to pars and epidural space

These joints resist forward displacement of one lumbar vertebra on another and prevent rotatory dislocation. A "distended facet" with increased synovial fluid can be seen on imaging when excessive motion is occurring at that spinal segmental level, such as from spondylolisthesis [11].

The facet joint bony articulations include the inferior articular process (IAP) of the more cephalad vertebra with the superior articular process (SAP) of the more caudal vertebra. Paradoxically, the IAP is the top of the facet joint while the SAP is the bottom of the joint. The Z-joint lines transition from a more sagittal orientation in the upper lumbar spine to a more oblique or coronal orientation in the lower lumbar spine. Thus, more oblique rotation is needed for visualizing the lower facet joint lines on fluoroscopic visualization. The lumbar Z-joints can also be viewed on lateral fluoroscopic imaging, just posterior to the intervertebral foramen.

The dorsal rami stem from the short spinal nerve housed in the intervertebral foramen. The dorsal rami then divide into the medial, intermediate, and lateral branches. (The L5 dorsal rami only divides into a medial and lateral branch.) The intermediate and lateral branches innervate the iliocostalis lumborum and longissmus thoracis, respectively. All Z-joints except one are innervated by the medial branches of the dorsal rami. The L5-S1 Z-joint is the one exception because this joint is innervated by the L4 medial branch and the L5 dorsal ramus. The innervation scheme nomenclature differs from the cervical facet joints (e.g., the C4-5 facet joint receives innervation from the C4 and C5 medial branches whereas the L4-5 facet joint receives innervation from the L3 and L4 medial branches). The typical lumbar medial branches stem from their respective dorsal rami and then course inferiorly between the SAP and the transverse process (Figure 10.1) [12]. Therefore, the typical target points for MBB are on the level below the named level of the medial branch (e.g., the L4 medial branch "lives" on the L5 vertebrae). The nerve then hooks medially around the SAP, crosses further medially toward the lamina (at the base of Z-joint) via a course underneath the mamillo-accessory ligament and divides into multiple branches. Sometimes a foramen or notch is formed and visualized on fluoroscopic images when the mamillo-accessory ligament ossifies. The medial branch innervates multifidus, interspinales, intertransversarii medialis muscles, interspinous ligament, and two Z-joints. The L5 dorsal ramus is more amenable to blockade than its respective medial branch. This L5 dorsal ramus crosses the sacral ala in lieu of the transverse process and is typically blocked at this location. Of note, some authors have argued that there is variability with the medial branch innervation to the various Z-joints (e.g., the L5-S1 facet joint may receive innervation from the L3,4,5 medial branches/dorsal rami) and paraspinal musculature. To perform MBB of a given Z-joint, two medial branches that innervate that joint should be targeted. For example, the right L4-L5 Z-joint is innervated by the right medial branches of L3 and L4. These medial branches run on the L4 and L5 vertebrae.

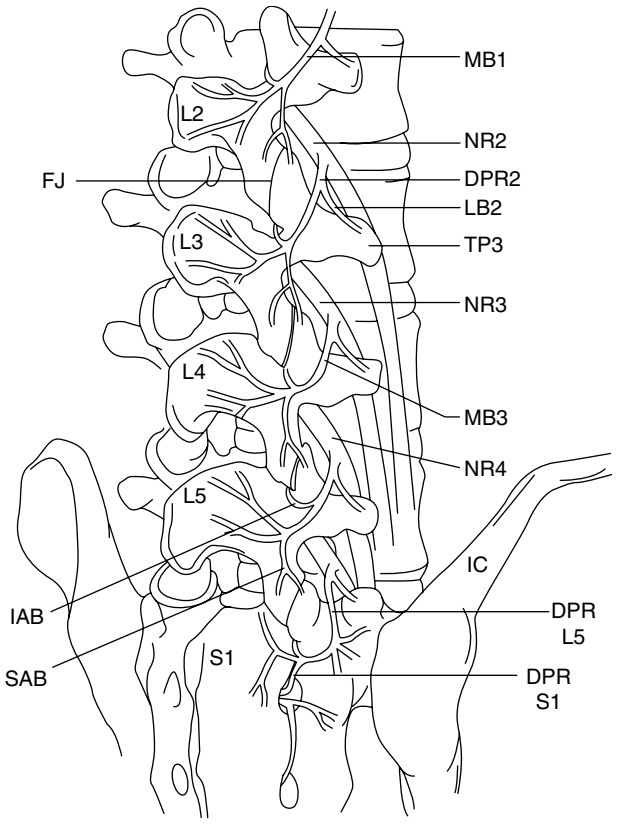

Figure 10.1 This figure shows the branching of the L3 medial branch (MB3) off of the L3 dorsal ramus. This medial branch descends and lies between the superior articular process and the transverse process of the L4 vertebra.

TECHNIQUE

The purpose of MBB is to deliver a local anesthetic specifically around the medial branch to verify whether a patient's low back pain is subserved by a targeted Z-joint, which would be supported by a corresponding reduction in the patient's index low back pain after the MBB. The ideal target point for MBBs is the space between the superior border of the transverse process and the mamillo-accessory notch [4]. This target more accurately localizes the medial branches and is associated with less epidural and foraminal flow [6]. Other more proximal target points have been suggested in the past. The target for the L5 level is the dorsal ramus which runs along the ala of the sacrum.

Medial Branch Blocks

Typically, a 22 or 25 g, 3.5-inch spinal needle is inserted after gaining an oblique fluoroscopic image visualizing the target point. Usually 15° to 20° of ipsilateral rotation is needed to properly visualize the target in an image reminiscent of a shallow Scotty dog picture. Multiple other views are needed to ensure that the needle tip is placed at the appropriate medial point (posterior-anterior view) and depth (lateral view) (Figure 10.2 A and B). The needle

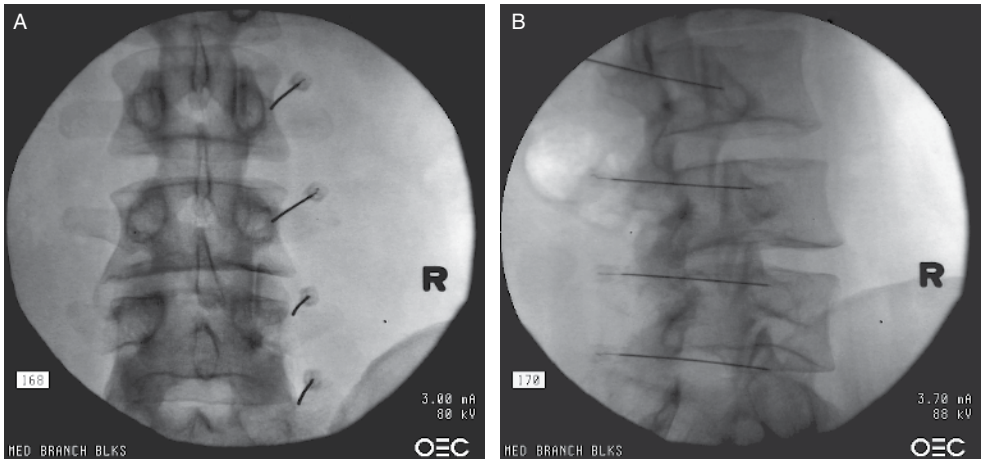

Figure 10.2 (**A**) A PA fluoroscopic image showing proper needle placement for L3 and L4 medial branches and L5 dorsal ramus injections with the target located "behind the eyebrow" of the Scotty dog. (**B**) An oblique fluoroscopic image showing the needle placement for medial branch blocks at the L2, L3, and L4 medial branches and the L5 dorsal ramus.

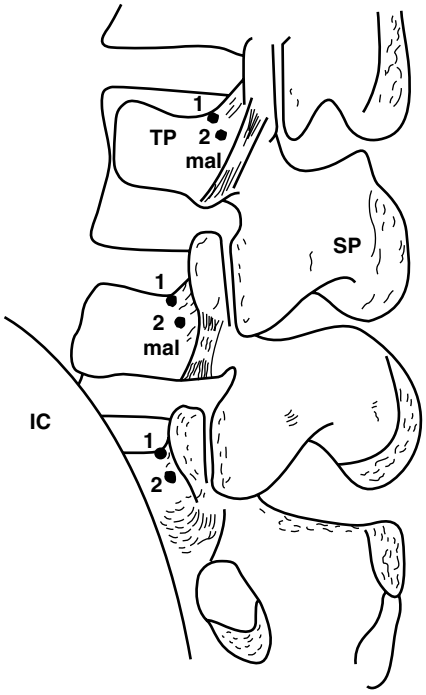

Figure 10.3 An oblique view of the lumbar spine showing the upper (1) and lower (2) target points for L3 and L4 medial branch blocks, and L5 dorsal rami blocks. Of note, the lower target point is preferred. Abbreviations: IC, iliac crest; SP, spinous process; TP, transverse process. From Ref. 6.

should reach an osseous stop; the location of the needle tip at this point should be at the mid-point between the transverse process and junction neck of the SAP (Figure 10.3) [6]. The neck of the SAP can also sometimes be identified with an ossified mamillo-accessory ligament or notch. Some have termed this location "behind the eyebrow of the Scotty dog." A common mistake is to place the needle on the SAP too posteriorly, as there will be an osseous stop felt on the rounded bulk of the SAP rather than its neck. Another mistake is to place the needle tip too laterally where it falls on the thick transverse process. Posterior-anterior and lateral views would reveal these mistakes. Once in correct position, the needle bevel should be directed medial and inferior to avoid intervertebral foramen spread and between 0.1 and 0.3 cc of contrast medium should be injected to ensure no vascular uptake. If there is no venous uptake, then 0.25 to 0.5 mL of concentrated anesthetic should be injected onto the target medial branch [5]. A smaller amount of LA may avoid the complication of unintended spread of medication to the foramen, which would cause a significant loss of specificity of this diagnostic procedure.

L5 Dorsal Ramus Block

The L5 dorsal ramus anesthetic injection follows the same general injection sequence; however, the target point is different due to the sacrum. The target point for this nerve is 5 mm below the superior junction between the sacral ala and the S1 SAP [6]. This "V-shaped notch" target point can be assessed by posterior-anterior view or 15° to 20° oblique view. The needle entry point should be just lateral to this position and the needle directed medially. Once the needle strikes bone, an anteroposterior view should be obtained, which should show the needle tip hugging the SAP of the sacrum. Contrast should be injected as described earlier followed by 0.25 to 0.5 mL of LA if there is no vascular flow [5].

Once the MBB(s) have been performed, it is critical to have the patient assess the pain levels using a pain diary. Pain levels should be assessed immediately prior to the procedure and afterward in a systematic fashion. The patient is instructed to record the low back pain severity every 30 minutes after the procedure for the next 4 hours and then hourly for the remainder of the day. Pain levels should also be recorded for the next two mornings. With the use of LA, the pain relief should correlate with the duration of action of the anesthetic used or longer. Generally, it is felt that 80% pain relief in the time frame appropriate given the half-life of the anesthetic used is considered a concordant and truly positive response. Cohen has advocated for a less

stringent pain relief criteria (50% improvement) based on similar radiofrequency ablation success rates with a 50% relief group versus 80% relief group [13].

Placebo effects are an added variable that complicate the determination of true-positives in response to the MBBs. In one study, the false-positive rate for uncontrolled diagnostic blocks of the lumbar Z-joints was 38% [14]. To compensate for this factor, the double-block technique (or comparative anesthetic block) has been substantiated. The double-block paradigm involves injecting a short-acting anesthetic (e.g., lidocaine) near the medial branches and then another longer-acting anesthetic (e.g., bupivacaine) on another occasion. These two anesthetics should provide pain relief of different durations. If the patient reports concordant pain relief, then the medial branch responses are considered more truly positive and the clinician can be more confident of his or her diagnosis.

Taking this a step further, others have suggested that the double block includes too many false-positive responders and suggest that a triple-block regimen should be used. The triple block is similar to the double block with a third step, the use of a placebo injection that should produce minimal to no pain relief. A concordant responder using this regimen would have 80% or more of pain relief with local anesthetic use in the time frames of relief fitting the duration of action of the anesthetics used. In addition, there should be little to no pain relief with the placebo injection. This regimen is very involved and difficult in clinical practice. Not only is the use of placebo without patient knowledge ethically questionable, payors often will not reimburse for placebo injections. Another issue with the triple-block paradigm is the theoretical effect of washing away inflammatory chemical mediators with placebo (normal saline) injectate, which could produce a true reduction in pain. For these reasons, clinicians generally prefer the double-block paradigm.

Others have questioned the practice of the comparative anesthetic regimen for various reasons. First, the double-block paradigm may not be cost-effective [15,16]. Second, there may be a significant false-negative rate with these double-block or triple-block regimens [17].

Intra-Articular Injections

Another diagnostic injection targeting the facet joint is the intra-articular or pericapsular anesthetic injection. An oblique image is obtained visualizing the facet joint line, then a slightly shallower (less oblique) image is procured (the so-called "modified best view"). The needle is then inserted into the medial border of the facet joint of question and a small quantity (0.2–0.3 mL) of contrast is injected [18]. Various joint contrast patterns may be seen on fluoroscopy, as previously mentioned, to confirm location within the Z-joint. Then, a small amount (0.5–1 mL) of concentrated anesthetic is injected. Lateral border may correspond to the anterior aspect of the joint depending on the shape of the joint; thus, this should not be the target point for entry into the posterior aspect of the joint. However, bony overgrowth (spurs) is often present in the posterior aspect of the SAP, which makes up the medial half of the facet joint. Entry into the joint can be technically difficult when severe arthrosis is present due to posterior spurs, reduced joint space, and calcified capsules. Some clinicians also instill corticosteroid into joint while performing this procedure. However, diagnostic specificity of the procedure may be lost when steroid is given. Similar to the MBBs, a pain diary should be kept and the duration of pain relief should be consistent with the duration of action of the anesthetic used.

One small study compared the pericapsular injection of anesthetic to MBBs and found that those selected using MBBs had better outcomes after denervation procedures suggesting that MBBs are superior to articular diagnostic blocks [19]. In addition, MBB are relatively easier to perform; they are safer and more expedient. Moreover, MBB, rather than facet intra-articular injections, predict success with radiofrequency neurotomy. The one advantage of facet intra-articular injections, if steroid is used, is that they may have more evidence of a therapeutic effect than MBB [20]. In 2010, paravertebral procedure codes (including MBB and facet intra-articular injections) were devalued and bundled with imaging-guidance codes due to an explosive increase in utilization and rampant miscoding [21].

CONCLUSIONS

The diagnosis of lumbar facet-mediated can be elusive through clinical examination; therefore, confirmation with lumbar MBBs can be useful to ascertain diagnosis. The results of a comparative anesthetic regimen can provide useful information into initiating further treatment to the facet joints, such as radiofrequency ablation. Properly performed lumbar MBBs in at least two different sessions help eliminate the high false-positives incurred with this diagnostic test. While facet intra-articular anesthetic injections can also be used as a facet diagnostic test, MBBs appear to be superior based on diagnostic validity testing. In summary, algorithmic care of low back pain appears to be clearly enhanced with the use of lumbar diagnostic blockades.

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11 Therapeutic Intra-Articular Lumbar Facet Joint Injections

Michael B. Furman and Justin J. Petrolla

INTRODUCTION

Potential etiologies for axial low back pain include the intervertebral discs, dura, spinal nerves, lumbosacral musculature, and the facet joints [1]. This chapter focuses on the diagnosis and procedural treatment of the lumbar intra-articular facet joints. Diagnosing lumbar intra-articular facet-mediated pain is challenging and controversial because the history, examination, and even diagnostic blocks have their limitations. The therapeutic procedure, whether with steroids or viscosupplementation, or even when used for joint aspiration and/or rupture, has sparse literature clearly demonstrating long-term benefit.

The vertebrae of the lumbar spine interact with one another through the "three joint complex" the intervertebral discs anteriorly, and the two facet joint articulations posteriorly [2]. This chapter will focus on the posterior aspects of this complex. These posterior articulations are known clinically as zygapophyseal joints but are also referred to as the "z-joints" or the generic term the "facet joints." The term "facet joint" actually refers to these and other true diarthrodial joints in the human body such as the knee or hip joints. Therefore, throughout the rest of this chapter, we refer to these as "lumbar z-joints" or "z-joints."

As the lumbar spine undergoes the degenerative cascade, the intervertebral disc undergoes desiccation and loss of height. This will shift more load onto adjacent posterior elements of the three joint complex, the z-joints [2]. This leads to degenerative changes within the z-joint, which in turn can lead to pain emanating from them.

HISTORICAL BACKGROUND

The lumbar z-joints were first considered as a possible cause of low back pain by Goldthwait in 1911 [3]. More than 20 years later, the term "facet syndrome" was introduced by Ghormely, who also introduced a technique that used an oblique view of the lumbar spine to view and determine the degree of arthritis in the joints [4]. At that time, the assumption was that hypertrophic changes in the facet joint would lead to low back pain through impingement of spinal nerves. These developments were later overshadowed by Mixter and Barr's landmark paper in 1934, which introduced the herniated disc and its associated compression of the spinal nerves as the cause of low back and radicular pain [5]. Little interest was generated in the lumbar z-joint for the next 30 years.

In 1963, Hirsch was able to reproduce low back and lower limb complaints by injecting an irritant, hypertonic saline, around the lumbar z-joints in normal volunteers [6]. Despite these interesting findings, the z-joint did not garner much attention for its possible role in causing low back and lower limb pain.

In 1971, Rees reported a 99.9% success rate in relieving low back pain utilizing a percutaneous procedure to sever the nerves of the lumbar z-joints [7,8]. Although his results could not be replicated by other investigators, this lead to further anatomic studies to delineate the definitive innervation of the lumbar z-joints [9,10]. Interestingly, the concept of percutaneously denervating the lumbar z-joint was introduced by Rees and then Shealy in 1971 and 1974 prior to any published investigations of intra-articular facet injections [8,11]. Although the anatomic basis of these procedures was questionable, the concept of pain emanating from the z-joint became more accepted.

Intra-articular z-joint injections were first described by Mooney and Robertson in 1976, using fluoroscopy to identify and inject these z-joints. These investigators also demonstrated that these joints can cause low back pain in normal volunteers and could relieve the pain of certain patients by anesthetizing the joints [12]. McCall et al. were able to reproduce the finding of pain referral by injecting the z-joints of normal volunteers in l979. They also established that the referral patterns of the lumbar z-joints overlapped the lower lumbar spine, was rarely central, and could even extend down the lower limb. These findings though must be considered carefully because of the large volumes of injectate used in these studies [13]. In 1981, Fairbank et al. were able to reproduce relief of low back and lower limb pain by anesthetizing the lumbar z-joints [14]. These findings sparked further interest and led to further research into the clinical diagnosis, diagnostic injections, and therapeutic treatments of the suspected lumbar z-joint pain. Although there has been a surge in research related to lumbar z-joint-mediated pain, this remains a controversial topic in the physiatric, pain, and orthopedic literature.

PREVALENCE

Kalichman et al. performed the first cross-sectional study to determine the prevalence of lumbar z-joint osteoarthritis in a community-based population. Using computed tomography (CT) scan findings, they demonstrated arthritic z-joint findings in 59.6% of males and 66.7% of females, respectively. They also attempted to associate low back pain with CT scan findings of lumbar z-joint osteoarthritis but could find no association [15]. These findings support previous work which could not identify any associations between low back pain and radiologic imaging [16]. The prevalence of lumbar z-joint osteoarthritis increased with age and by age 60 to 69 had reached 89.2%. The L4-5 level was the most common level involved, which supports previously published results [15,17]. A cadaveric study performed by Eubanks et al. found osteoarthritic changes in the lumbar z-joint to be a universal finding. This study also postulated that by age 30, nearly half of all individuals have osteoarthritic changes in the lumbar z-joint, with the L4-5 level being the most commonly involved [17]. Although the prevalence of lumbar z-joint osteoarthritic changes has been delineated, identifying which joint is contributing to a given patient's low back pain has proven to be a formidable task.

DIAGNOSIS OF LUMBAR Z-JOINT PAIN

Physical Examination

Thus far, multiple attempts have been made, but definitive history or physical examination findings to identify lumbar z-joint-mediated pain have not been identified. Revel et al. reported several clinical features to suggest the lumbar z-joint as the possible source of pain, which included the following: age greater than 65 years; pain not exacerbated by coughing, hyperextension, forward flexion, extension-rotation, or rising from forward flexion; or mitigated by recumbence [18,19]. Subsequent studies, however, were not able to reproduce these results [20,21]. In 2007, Hancock et al. reviewed all recent literature concerning the diagnosis of lumbar z-joint, sacroiliac joint (SIJ), and discogenic pain. Although the authors found some limited diagnostic value for clinical examinations to identify the SIJ and the disc as pain generators, no diagnostic value was found for any test in identifying the lumbar z-joint as the source of pain [22]. Many physicians rely on the so-called Kemp's test, which involves rotation and extension of the lumbar spine, or will rely on concordant pain with deep palpation of the lumbar musculature overlying the lumbar facet joint. Neither of these examination maneuvers have been validated.

Imaging

The use of plain x-rays, magnetic resonance imaging (MRI), CT, or radionuclide bone scanning has not aided in the diagnosis of pain emanating from the lumbar z-joints [23–27]. The single photon emission CT (SPECT) scan has garnered some interest with preliminary studies [26–28]. The SPECT scan was also used in a recent study by Ackerman and Ahmad. In this study, patients with a positive scan had a better outcome with intra-articular lumbar z-joint injections compared with another group of SPECT-positive patients undergoing medial branch blocks. Although these are interesting results, there are several issues with the design of the study. The power of the study is low, and, more importantly, the diagnosis of lumbar z-joint pain was made without the use of diagnostic blockade. Also, a 50% reduction in pain was considered a positive result, and the patients were followed for a short period of only 12 weeks [29]. Regardless of these shortcomings, there may be a role in the future for the use of SPECT scans to aid in the diagnosis lumbar z-joint pain. Interestingly, a recent study by Willick et al. examined the use of the so-called fire scan in the diagnosis of facet-mediated pain. The fire scan is essentially the digital fusion of a CT scan with a bone scan and SPECT imaging of the area of interest. The authors retrospectively reviewed the results of 26 cases in which a fluoroscopically guided injection was undertaken based on physical/clinical examination findings coupled with a positive fire scan. In all cases, the patients experienced immediate and sustained relief following the planned injection [30]. Although these findings are preliminary and retrospective, they are quite interesting. Unfortunately, the so-called fire scan is not ready for widespread use. Thus far there is no clinical examination finding, constellation of symptoms, or imaging modality that has been proven to implicate the lumbar z-joint as the primary pain generator.

Diagnostic Injections

Diagnostic lumbar z-joint injections, directed at either the intra-articular space or the nerve supply to the joint (i.e., medial branch blocks), are considered the most accurate means of diagnosing lumbar z-joint-mediated pain [31–33]. The response is typically monitored using a prospective pain diary completed by the patient. Ideally, this should also include an index activity important to the patient (i.e., bowling, golf, sitting at a desk). Most purists advocate that complete (100%) postprocedure pain relief is considered "diagnostic" with lumbar z-joint injections. However, many physicians use softer criteria in clinical practice (50% or 75% relief). Of note, a reduction of 50% in patient's pain has been found to improve a patient's quality of life [34].

When a single diagnostic block is used, the false-positive rate has been found to be as high as 38% [35]. Because of the high rate of false-positives, many physicians advocate using dual blocks to diagnosis lumbar z-joint-mediated pain. In this paradigm, blinded patients undergo two separate diagnostic injections with two anesthetics differing in pharmacologic duration of anesthetic effect. The patient's anesthetic and functional response to each injection is recorded prospectively. Those patients with "appropriate" response would undergo a therapeutic z-joint procedure, either intra-articular z-joint injections with a mixture of anesthetic and steroid or a radiofrequency (RF) procedure in the hopes of creating a longer response.

Interpreting the response to the dual blocks can be challenging. Because the "short" and "long" term acting anesthetics overlap in their time duration, the pain relief

extent is not considered tantamount to a positive diagnosis. Some patients even describe long-term (days, weeks, or months) benefit form the diagnostic injection. Likewise, although interpreting a positive response to only one of the two blocks should be considered a negative response, some physicians use their "clinical judgment" anyway treating this as a "soft positive" thereby negating the rationale of dual blocks. Others even begin doing more blocks making interpretation even more challenging.

Alternatively, patients who have a positive response to the initial single diagnostic injection could undergo a therapeutic injection with anesthetic and steroid in an attempt to limit the amount of injections the patient will receive. The second injection could also be included in the diagnostic protocol. Proponents of using a only a single-block [36] advocate that a double-block protocol creates too many false negatives, thus depriving patients of a potentially beneficial therapeutic z-joint treatment, that is, RF.

In theory, a triple-block paradigm, two different anesthetics plus a placebo, could be utilized to truly reduce the placebo response and improve specificity and sensitivity, but this regiment is not feasible outside of pure research. The double-block paradigm is a reasonable alternative to a single-block regiment (high false-positives) and a triple-block paradigm (increased cost, procedures) with the caveat that there may be some false-negative patients with this regimen [32]. Many patients prefer not going through the expense, time, or discomfort associated with the second block, especially after an extremely successful (positive) single block. Ultimately, though the decision as to diagnosis and treatment is up to the treating physician and patient, clinical judgment should be used prudently.

There is no protocol to decide which joints should be injected first. The L4-5 and L5-S1 joints are generally accepted as the most commonly involved joints, and thus, most physicians will start by investigating these joints [20,37]. In our practice, when the patient has bilateral involvement, we will try to identify the most painful side and start our investigation on that side. This is an attempt, on our part, to have the least amount of variables involved when diagnosing and treating lumbar z-joint pain. Unfortunately, circumstances do not always allow for such a regimented structure, and we will investigate both sides when needed.

REVIEW OF PERTINENT ANATOMY

An intimate understanding of the anatomy of the z-joint is needed for a successful intra-articular injection, especially considering that individuals getting this injection may have suboptimal anatomy. The lumbar z-joints are true diarthrodial synovial joints that contain a joint space, hyaline cartilage, and fibrous capsule [31]. The joint itself is created from the interface of the superior and inferior articular processes of sequential lumbar vertebrae. The joint is curved in a crescent-like or C-shape with the C facing the spinous process (Figure 11.1). The posterior portion of the joint is oriented sagittally as compared with the anterior portion which is oriented more coronally (Figure 11.2). This is especially relevant for injections because the posterior portion of the joint is where the needle enters for an intra-articular injection. The lumbar z-joints' orientation changes as they proceed from L1 through S1. The upper lumbar joints are oriented more sagittally to resist axial rotation, and the lower joints are more coronally oriented to resist shearing forces [38].

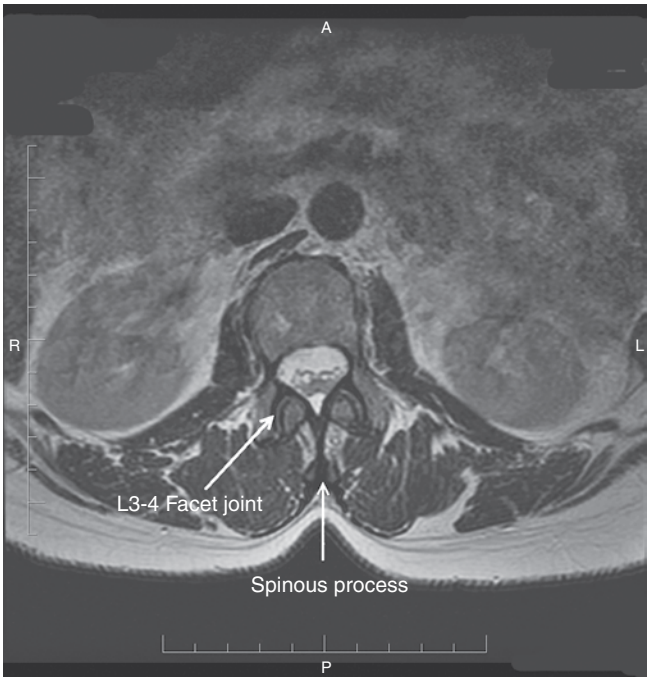

Figure 11.1 T2 MRI axial image of the L2-3 lumbar facet joints. Note the crescent or "C" shape of the facet joints with the curve oriented towards the spinal process.

The fibrous capsule is roughly 1 to 2 mm thick and quite strong [37–39]. The capsule blends with the ligamentum flavum anteriorly [40]. The anterior capsule can, at times, form a cyst which can lead to compression of the spinal nerves and radicular complaints [41,42]. Posteriorly the capsule is covered by the mutlifidus muscle, and fascicles of this muscle help to reinforce the joint [40,43]. The posterior capsule eventually blends into the cartilage of the interior joint [39]. Two recesses are created at the superior and inferior aspects of the posterior z-joint. The inferior recess is larger and thus is the usual target for intra-articular injections [44]. Though smaller, the superior recess is contiguous with the joint space and can be used as a target when needed. Although the superior recess can be used for joint entry, the close proximity of the spinal nerve makes it a less desirable target (Figure 11.3). Synovial lined adipose tissue is present at these recess areas and extends in villi or fold like meniscoid inclusions. The role of this tissue is unclear, but it may have some role in pain originating from the z-joint [40,44].

Each lumbar z-joint is primarily innervated by two medial branches of the dorsal rami [38,45]. Although the innervation appears simple, it can be somewhat confusing.

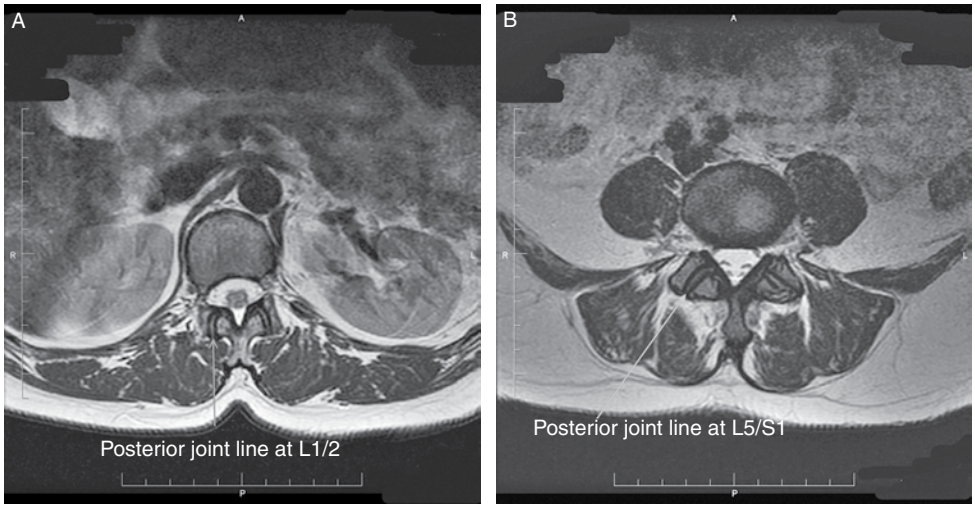

Figure 11.2 T2 MRI axial images of the L1-2 **(A)** and L5-S1 **(B)** facet joints. Note the angle change as the joints descend from superior to inferior in the lumbar spine. This will directly affect the angle used for joint entry.

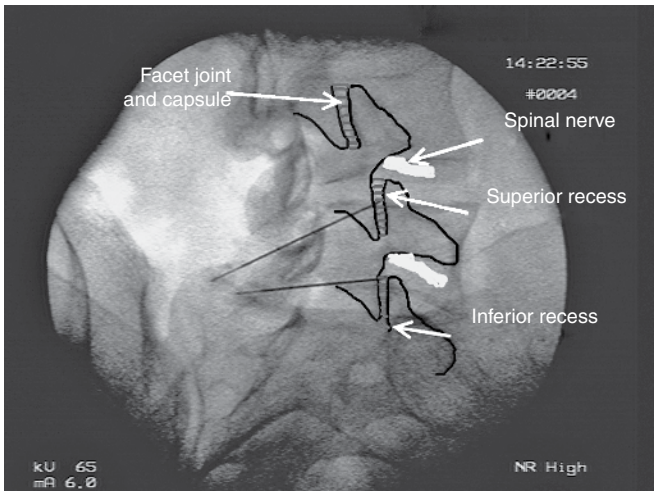

Figure 11.3 Fluoroscopic oblique image of the L3-4, L4-5, and L5-S1 facet joints. Note the close proximity of the spinal nerve to the superior recess of the facet joint.

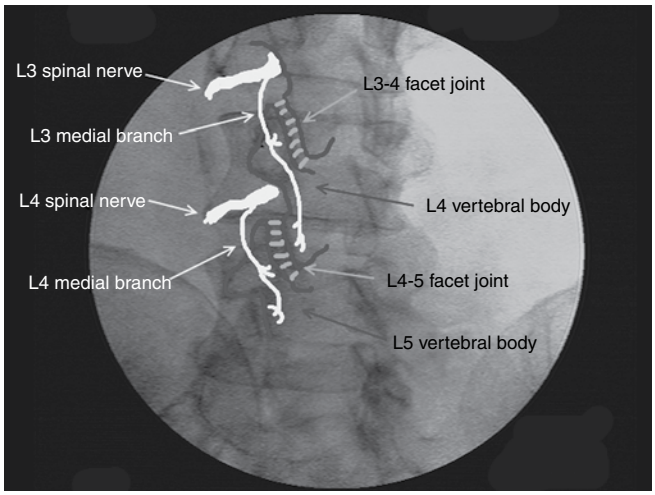

Figure 11.4 Fluoroscopic imagines of the lumbar facet joints. Note the innervation of the L4-5 facet joint is from the L3 and L4 medial branches.

Although each joint is innervated by the segmental levels superior and inferior to it (Figure 11.4), the actual numbering can be confusing. For example, the L4-5 z-joint is innervated by the L3 and L4 medial branches (Figure 11.4). This is true for all lumbar z-joints except for L5-S1. The L5-S1 facet joint is innervated by the L4 medial branch and the dorsal ramus of L5.

The capsule of the z-joint itself has been found to have a complex innervation [46]. Substance P along with autonomic fibers has also been found within the z-joint, subchondral bone, and capsule [47,48]. Overload of the capsule with subsequent activation of these fibers and substances has been suggested as possible cause of z-joint pain. The synovium has been shown to contain nociceptive fibers, but the role of these nerves is debated [49–51].

TECHNIQUE OF FACET JOINT INJECTIONS

This procedure is typically performed with the patient in the prone position on the fluoroscopic table. Because of the difficulty in diagnosing z-joint-mediated pain and the potential for false-positives, sedation is typically not recommended [52]. Intravenous placement is optional in nonsedated patients.

The correct level(s) is (are) identified in anteroposterior (AP). The superior and inferior end plate can be squared but this is not a necessary step. The C-arm image intensifier is then obliqued ipsilaterally until the posterior joint space is barely visualized. This is an important point because over-rotation obliquely will bring the anterior joint space into view which can not be accessed from a posterior approach (Figure 11.5). Also,

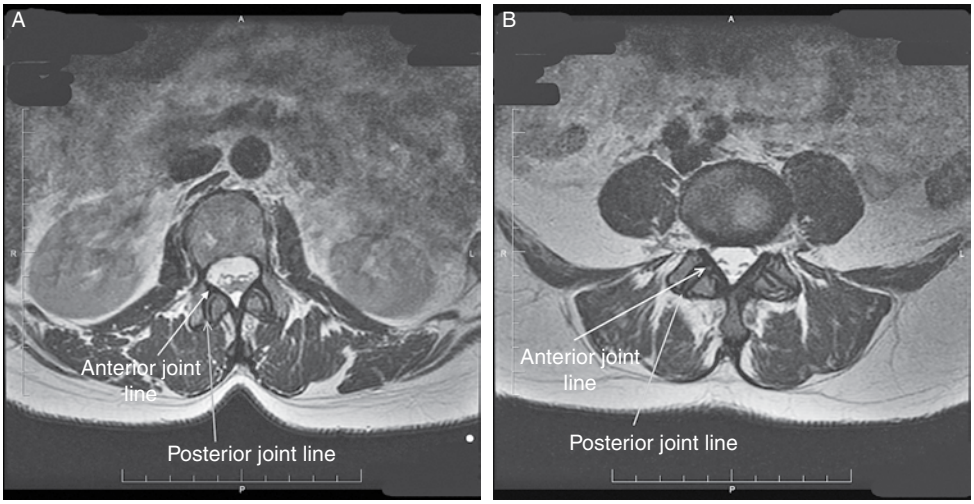

Figure 11.5 Note the anterior versus posterior joint space lines for **(A)** L2-3 and **(B)** L5-S1. This is paramount when setting up the trajectory view. If the anterior joint space is targeted the needle will not enter the joint. The posterior joint space is generally closer to midline than one would expect.

although the joint is typically oriented so the posterior entry is more lateral, it is easier to enter the joint from its more medially oriented opening. For the upper lumbar z-joints, the angle can be 10° to 30°, and for the lower z-joints, the angle can reach 45° or more. Because there is significant variability in the z-joints of each individual, care must be taken to properly use the C-arm's obliquity and tilt to properly identify each individual z-joint's entry point.

Once the fluoroscopic trajectory image has been identified, the area is prepped with a betadine, alcohol, or other cleaning solution, and a sterile drape is applied. A skin wheal can be raised around the target site using 1% lidocaine or a local anesthetic of the practitioner's choice. A 22- or 25-gauge spinal needle is then inserted parallel to the angle of the beam (trajectory view) toward the joint space. As noted earlier, the inferior joint space recess is optimal because of the larger size of this area and the spinal nerve exits closer to the superior recess which can cause discomfort for the patient. Of note, the tip of the spinal needle can be bent with the bevel of the needle for greater control but is not specifically needed for this injection. The needle is advanced until periosteum is touched by the needle or the give of the z-joint capsule is felt. At this point, we advocate triplanar imaging (AP, oblique, and lateral) to guide final needle positioning and gauge needle location before contrast is administered. The lateral view will identify that the needle tip is truly intra-articular and not too far dorsal or ventral (Figures 11.6, 11.7, and 11.8).

Once needle placement is confirmed with the proper imaging, a small amount, roughly 0.2 to 0.5 cc, of nonionic contrast is administered under live fluoroscopy observing for any vascular, venous, or arterial uptake. Only minimal contrast should be utilized so that the joint is not filled before placing the final injectate. The z-joint arthogram classically appears as an S-shaped image but may appear linear. The z-joint will generally hold roughly 1.0 to 1.5 cc of injectate prior to leakage or rupture, but leakage can occur with volumes as low as 0.5 cc of injectate [53,54]. If a larger volume of contrast and/or injectate is used the capsule of the joint may rupture and leakage of injectate onto the surrounding structures will occur. If contrast is leaked then the fluoroscopic images could be compromised. If injectate is leaked this would theoretically reduce the diagnostic accuracy of the injection and should be avoided especially for diagnostic z-joint injections. Once the appropriate images are saved, the injectate is administered and an "end-feel" is appreciated as the joint is filled. The maximal injectate used is roughly 1.0 to 1.5 cc for each joint. We administer a mixture of 1 cc of 1% lidocaine with steroid. We typically use a total of 80 mg of triamcinolone or depomedrol divided equally among all joints injected. We will use more concentrated steroid (80 mg/cc) when injecting only one or two joints allowing for sufficient anesthetic as well.

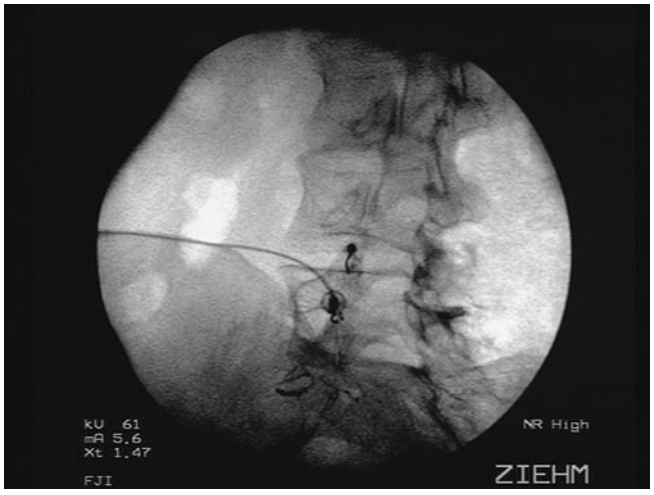

Figure 11.6 Note needle position is oblique or trajectory.

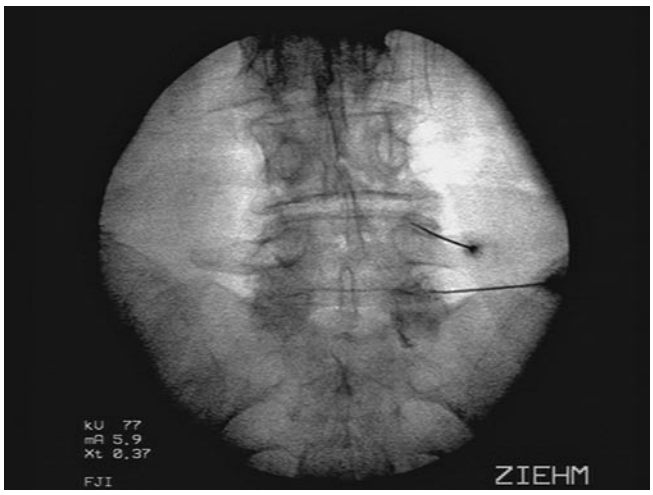

Figure 11.7 Note needle position is anteroposterior.

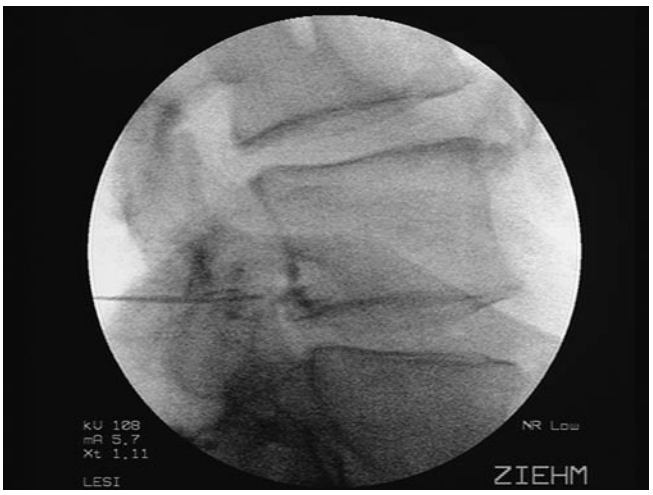

Figure 11.8 Note needle position is lateral.

When performing intra-articular lumbar z-joint injections, we suggest that a 25- or 22-gauge needle, either 3.5 inch or 5 inch, is used. The 25-gauge is an easier fit into the joint space, especially with the more arthritic joint, and tends to be less painful for the patient. A 22-gauge needle will allow improved needle directionality and more "feel" when entering the joint capsule.

EFFICACY OF THERAPEUTIC INJECTIONS: STEROIDS AND VISCOSUPPLEMENTATION

As with the diagnosis of lumbar z-joint pain, the treatment of this condition is controversial. Unfortunately, the literature is not robust and consists of multiple uncontrolled studies intermixed with few controlled trials. A complete review of all relevant literature for intra-articular lumbar z-joint injections is beyond the scope of this chapter, and several excellent reviews have already been completed by numerous authors [31,55]. The literature supporting therapeutic intra-articular lumbar z-joint steroid injections is scarce. In a recent review, therapeutic intra-articular z-joint steroid injections support was deemed "weak or not recommended at all" [55]. Some authors have stated that, because of the poor research and lack of definable therapeutic benefit, there is no role for intra-articular lumbar z-joint injections whether diagnostic or therapeutic [56]. Although it is true that the research in support of z-joint injections (diagnostic or therapeutic) is not robust, there is still room within evidence-based medicine for clinical judgment and expert opinion when the research is found to be lacking [57].

The role and mechanism of intra-articular steroids is also controversial. No study has specifically looked for inflammatory cells within the facet joint of patients with presumed z-joint-mediated pain. Also, the exact mechanism of intra-articular steroids is only presumptive at this time.

Because of these questions and the specific anatomy of the joint itself, several authors have looked at the role of viscosupplements in the treatment of this condition. In theory, viscosupplements offer a means of halting or reversing the degenerative changes that occur within the z-joint itself. Studies by Fuchs and Clearly unfortunately failed to produce positive results [58,59]. A major flaw in these studies was a lack of presumed diagnostic accuracy with the use of dual diagnostic blocks. Also, these studies did not use multiple treatments with viscosupplementation similar to treatment of knee osteoarthritis. A recent pilot study did utilize a dual intra-articular diagnostic block paradigm to select patients for viscosupplementation and used multiple treatments. This study had more positive results and hopefully will lead to further research in the role of viscosupplements for painful lumbar z-joints [60].

SPECIAL CASES: FACET CYSTS

As mentioned earlier, z-joints may occasionally develop a cyst on their anterior aspect. Generally, these cysts are asymptomatic and simply noted on MRI or during surgery (Figure 11.9). Symptoms can occur if the cyst becomes large enough to displace or crowd the neural structures. Generally, the symptoms caused by a facet cyst will be radicular or similar to spinal stenosis. Attempts have been made to aspirate facet cysts and/or inject them with a steroid/anesthetic mixture, but the results have been mixed with this technique. Rupturing the cyst though has been shown to be highly effective treatment for this condition. In a retrospective review by Allen et al., a 72% success rate with initial treatment was achieved. The recurrence rate was 37.5% in their study, and 45% of these patients who underwent a second rupture achieved a success. In this study, success was defined as improvement in symptoms and the avoidance of surgery. Failure was defined as lack of improvement in symptoms and the need for surgical intervention. Failures were attributed to incomplete rupture or loculations within the cyst itself, but no correlating

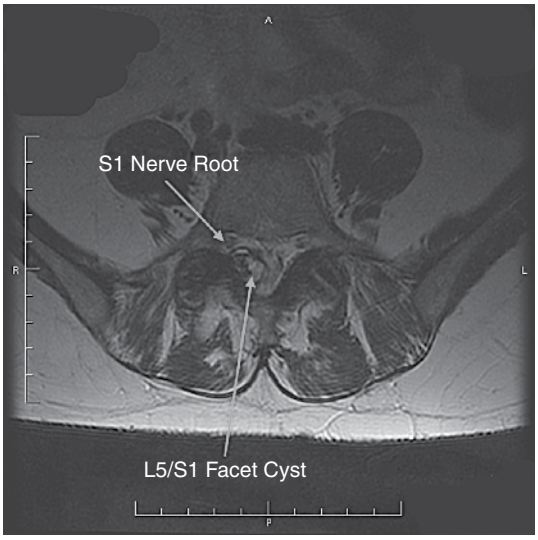

Figure 11.9 Axial T2 MRI image depicting an anterior L5-S1 facet cyst which is abutting the right S1 nerve root.

factors could be identified [61]. A recent large retrospective review found the success rate to be roughly 81% for rupture of a z-joint cyst and 46% avoided surgical intervention. Both injection and surgical groups reported statistically significant reductions in low back pain, leg pain, and Oswestery Disability Index (ODI). The cyst rupture group did, however, report a slightly higher ODI score compared with the surgical group, but this was not statistically significant [62].

In our practice, we employ a technique that is very similar to what the authors Allen et al. describe [61]. Because of the close proximity of the spinal nerve to the cyst, a transforaminal injection is preformed first. Care must be taken to treat the correct level. A facet cyst at L4-5 generally will affect the L4 spinal nerve. In our practice, we perform the injection with anesthetic only in an attempt to reduce any discomfort the patient may experience with the cyst rupture. After performing the transforaminal injections, we follow our previously described technique for an intra-articular injection. We mix 1% lidocaine with our contrast for a facet cyst rupture. After identifying proper needle placement with triplanar imaging, we administer contrast to obtain an arthrogram. We then inject up to 6 cc of a 1:1 mixture of lidocaine and contrast until epidural flow is noted. Occasionally, a give can be felt when the cyst is ruptured. We take live fluoroscopic images during the procedure, but intermittent pictures can be taken once the arthrogram is identified. In our experience, the patient will experience very little discomfort if the spinal nerve is anesthetized prior to cyst rupture.

RECOMMENDATIONS FOR INTRA-ARTICULAR FACET JOINT INJECTIONS

The role for intra-articular z-joint injections in the diagnosis and treatment of axial low back pain is controversial. Further challenging is that there are no pathognomonic findings (history, examination, or imaging) for lumbar z-joint-mediated pain. The literature is sparse and many of the studies have significant flaws, making interpretation difficult. Although lumbar intra-articular injections can be very effective in the diagnosis of pain mediated from the z-joint, identifying the appropriate patients is difficult. The role of therapeutic intra-articular steroid injections appears limited; however, the use of viscosupplements in the treatment of this condition has shown some promise. As technologies and further research are developed, the proper identification and treatment of this condition will be even better understood. Therefore, the intra-articular z-joint procedure is still an effective tool for the diagnosis and treatment of axial low back pain when used judiciously.

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12 Radiofrequency Denervation for Lumbar Facet Joint Pain

Frank J. E. Falco and Stephanie Geffert

INTRODUCTION

Goldthwait [1] first considered the facet joint as a source of low back pain in 1911. He believed that the lumbar facet joint was responsible for low back pain, lumbar spine instability, and leg pain. Putti [2] supported the concept that facet joint generated low back and leg pain in 1927. Ghormley [3] coined the term "facet syndrome" in 1933, which is still used today to describe this condition. The prevalence of symptomatic lumbar facet joints has been reported as 8% to 45% in chronic low back pain [4–6].

Mooney and Robertson [7] evaluated the lumbar facet joint in a group of individuals with and without low back pain with provocative hypertonic saline facet joint injections. The subjects described the pain produced from the injections as deep, dull, and vague. The location of the pain produced by stimulating the facet joints with the intra-articular injections was recorded, and pain referral maps were constructed for both groups (Figure 12.1). The information derived from these maps has been helpful in the assessment of patient pain drawings [8,9]. The pain drawing is often used in clinical practice to help the physician evaluate lumbar disorders.

Facet syndrome has been described as nonspecific low back pain with a deep and achy quality. Facet joint dysfunction can result from osteoarthritis or trauma. The syndrome is considered in a patient with low back pain that radiates into the buttock and posterior thigh. Patients might complain of symptoms that increase with twisting the back or bending backward. Physical examination typically reveals local lumbar paravertebral muscle tenderness to palpation. Radicular findings are commonly absent. The pain can often be increased with hyperextension and rotation of the lumbar spine consistent with historical complaints as described earlier. Although these findings might be somewhat helpful in evaluating for lumbar facet syndrome, no history or physical examination findings are unique to this condition [10–13].

CLINICAL ANATOMY

The zygapophyseal joints, also known as facet or "z" joints, are paired posterolaterally along the lumbar spine as well as the entire vertebral column. The facet joints are diarthrodial with articular cartilage, menisci, a synovial membrane, and a richly innervated capsule (Figure 12.2). The lumbar facet joint is formed by the inferior articular process of the vertebra above and the superior articular process of the vertebra below. The orientation of the lumbar facet joint is different within the lumbar spine region as well as in the cervical and thoracic spine. The upper lumbar facet joints are oriented in a sagittal plane, whereas the lower facet joints approach a more frontal orientation. The zygapophyseal joint controls specific motion of each vertebral segment depending on the orientation. Resistance to forward displacement is greatest with the lower lumbar facet joints, and rotation is resisted most by upper z joints.

The lumbar spinal nerves exit through the root canals and divide into the anterior and posterior rami. The anterior rami form the lumbar plexus to supply innervation to the lower limbs, torso, and genitorectal areas. The posterior lumbar rami divide into the medial, intermediate, and lateral branches except for the L5 dorsal branch, which only forms the first two branches. These nerves innervate the posterior back structures including the deep postvertebral muscles, ligaments, skin, and zygapophyseal joints. The medial branch nerves course posteriorly over the subjacent tansverse process, providing innervation to the multifidus

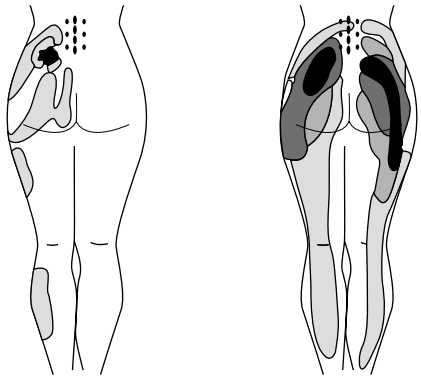

Figure 12.1 Lumbar facet joint pain mapping.

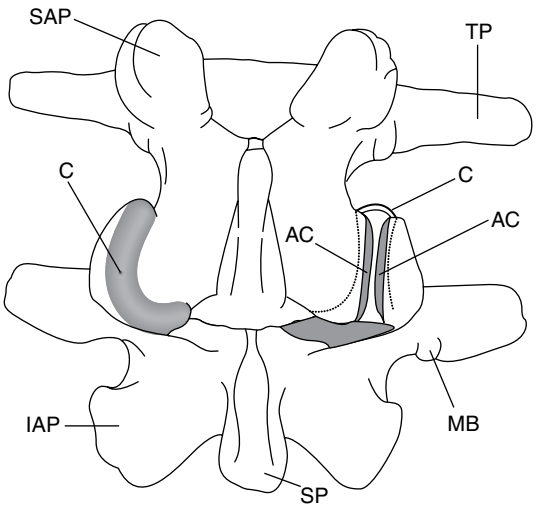

Figure 12.2 Lumbar facet joint anatomy. AC, articular cartilage; C, capsule; IAP, inferior articular process; MB, mammillary body; SAP, superior articular process; SP, spinous process; TP, transverse process. Adapted from Bogduk N, Twomey LT, eds. *Clinical Anatomy of the Lumbar Spine*. 2nd ed. New York, NY: Churchill Livingstone; 1991.

muscle, interspinous muscle, interspinous ligament, and the z joint above and below (Figure 12.3).

RADIOFREQUENCY ABLATION NEUROTOMY

The basic equipment needed to produce a radiofrequency (RF) tissue lesion from high-frequency waves includes a voltage generator, alternating current, and active and reference electrodes. The patient's tissues serve as a resistor within the circuit and provide impedance. The active electrode is an insulated needle with an exposed tip, whereas the reference electrode is a large surface self-adhesive pad. This configuration leads to the greatest current concentration and heat being next to the tip, with diffusion of the current and heat at the large reference electrode. The current causes vibration of the electrons in the tissues in the vicinity of the RF probe, resulting in an increase in temperature. The greater the voltage and the tissue impedance, the higher the temperature that develops within the targeted tissues.

The advantages of RF include controlled lesion size, accurate temperature monitoring, limited need for anesthesia, precise probe placement, low incidence of morbidity or mortality, and rapid post-procedure recovery. The lesion size is dependent on the probe diameter, length of the uninsulated tip, temperature, the time, and the tissue vascularity. In general, the lesion size is greater with a larger probe diameter, longer uninsulated tip, higher temperature, lower tissue vascularity, and longer lesioning time.

Pulsed RF uses 10 to 30 ms bursts of high-frequency alternating current. Lesions created by pulsed RF are low temperature (cold RF) and are nondestructive lesions. When creating a RF lesion the tissue that surrounds the tip of the electrode is exposed to an electromagnetic field.

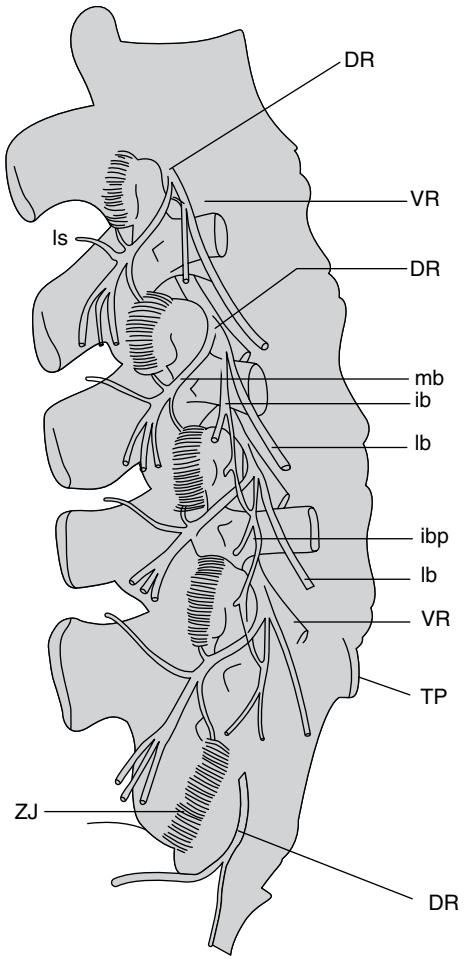

Figure 12.3 Lumbar facet joint innervation. a, articular branch; DR, dorsal ramus; ib, intermediate branch; ibp, intermediate branch plexus; is, interspinous branch; lb, lateral branch; mb, medial branch; VR, ventral ramus; ZJ, zygapophyseal (facet) joint. Adapted from Bogduk N, Twomey LT, eds. *Clinical Anatomy of the Lumbar Spine*. 2nd ed. New York, NY: Churchill Livingstone; 1991.

Although the mechanism by which pulsed RF treatment works is not known, there are several theories. One theory is that the electromagnetic field might have a clinical neuromodulation effect rendering the nerve less likely to transmit painful impulses. Another possibility is that it works in a similar manner to transcutaneous electrical nerve stimulation, activating both spinal and supraspinal mechanisms, which can reduce pain perception.

INDICATIONS

Patients with functionally limited spinal facet joint pain that is resistant to at least 3 months of conservative treatment are candidates for RF neurolysis or radiofrequency ablation. This condition cannot be definitively diagnosed by history, physical examination, or imaging studies. The current method for diagnosis is through facet joint injections or medial branch (facet joint) nerve blocks (see

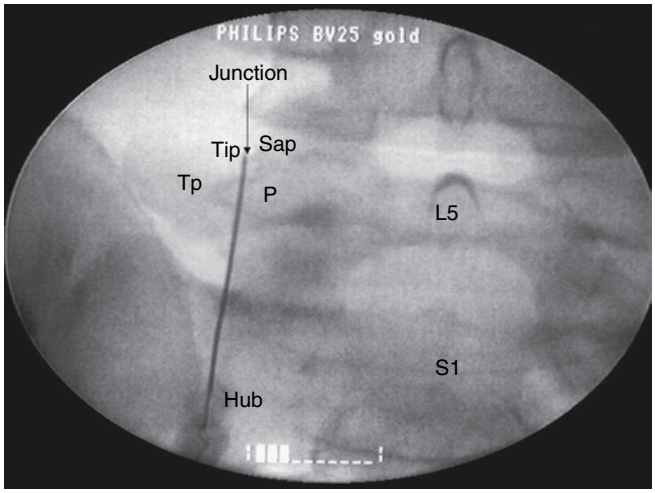

Figure 12.4 Radiofrequency neurolysis lumbar facet joint. Radiofrequency probe in position for left L4 medial branch RF neurotomy AP view. hub, RF probe hub; junction, junction of sap and tp; L5, L5 vertebra; P, pedicle; sap, superior articular process; S1, S1 vertebra; tip, RF probe tip; tp, transverse process.

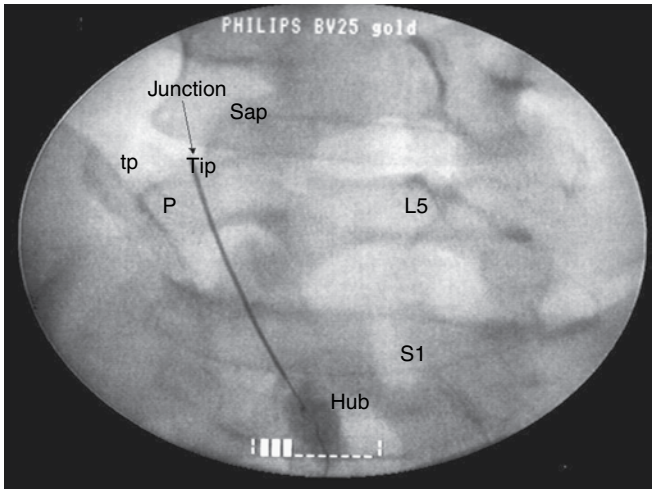

Figure 12.5 Radiofrequency neurolysis lumbar facet joint. Radiofrequency probe in position for left L4 medial branch RF neurotomy oblique view. hub, RF probe hub; junction, junction of sap and tp; P, pedicle; sap, superior articular process; L5, L5 vertebra; S1, S1 vertebra; tip, RF probe tip; tp, transverse process.

chapter 9). The nerves that supply the facet joints to the lumbar spine are the medial branches of the dorsal rami and the L5 dorsal ramus. The lumbar medial branch blocks have been shown to be target specific if anesthetic solutions are injected carefully at specific osseous target points, and contrast is necessary to ensure that inadvertent venous uptake does not occur. A dual injection paradigm of the medial branch nerves is recommended for a more accurate diagnosis of facet joint pain because of the false-positive rates associated with single medial branch nerve blocks.

TECHNIQUE

The patient is placed prone for RF lesioning of the lumbar facet joints. The RF probes are placed parallel to the nerves as opposed to the perpendicular approach used for medial branch nerve blocks. This allows for optimal denervation of the medial branch nerves. The probe is placed inferior and lateral to the targeted medial branch and advanced under fluoroscopy until contact at the junction of the superior articular process and the transverse process (Figure 12.4). An oblique "Scottie dog" view (Figure 12.5) is then obtained, and the needle should be seen to reside parallel to the target nerve in the osseous groove. The needle is then advanced to the proximal junction of the superior articular process and transverse process for the L1–L4 medial branch nerves (Figure 12.5), and the proximal junction of the S1 superior articular process and the sacral ala for the L5 dorsal ramus. A lateral view is then obtained to ensure the needle is placed no further anterior than the posterior aspect of the foramen (Figure 12.6). The C-arm is then finally repositioned in an anteroposterior projection to verify that the needles did not stray laterally while being advanced under oblique imaging with a final needle tip position just medial to the lateral silhouette of the

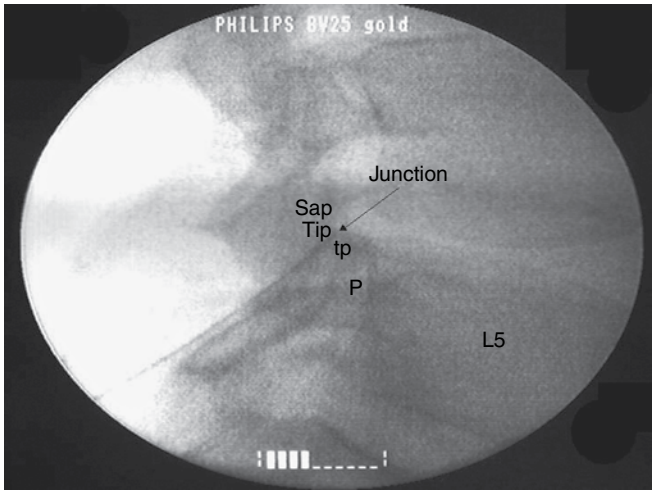

Figure 12.6 Radiofrequency neurolysis lumbar facet joint. Radiofrequency probe in position for left L4 medial branch RF neurotomy lateral view. junction, junction of sap and tp; L5, L5 vertebra; P, pedicle; sap, superior articular process; tip, RF probe tip; tp, transverse process.

superior articular process. Sensory and motor nerve fiber electrical stimulation is done as a safety precaution, and then the area is anesthetized. Typically, sensory stimulation at 50 Hz produces low back discomfort or pressure between 0.3 and 0.8 V. Multifidus contractions are elicited between 0.7 and 1.1 V, and voltage is increased to approximately two to three times the level at which sensory symptoms were noted. The needle tip must be repositioned if radicular pain and/or myotomal contractions are encountered during electrical stimulation. The radiofrequency ablation lesion is created by hearing the probe to 80 to 90°C for 90 to 120 seconds.

EFFICACY

A recent exhaustive systematic review of the literature was published regarding the efficacy of therapeutic lumbar facet joint procedures in treating low back pain [14]. A computerized search of the literature in the English language was performed for studies of therapeutic intra-articular facet joint injections, lumbar facet joint nerve blocks, and lumbar facet joint nerve RF neurotomy from 1996 to December 2008 using Medline and EMBASE as well as manual searches of bibliographies of known primary and review articles.

The studies were evaluated using the Agency for Healthcare Research and Quality criteria for observational studies [15,16] and by modified Cochrane Musculoskeletal Review Group criteria for randomized trials [17]. Only studies with a score of 50 or more on a weighted score of 100 was included in the analysis for the systematic review. The level of evidence from the literature for RF neurotomy for the treatment of lumbar facet joint pain was determined based on five categories developed by the Agency for Healthcare Research and Quality US Preventive Services Task Force as shown in Table 12.1 [18]. The recommendations for the use of RF neurotomy for the treatment of lumbar facet joint pain based upon the analysis of the literature was graded based on Guyatt's criteria listed in Table 12.2 [19].

Studies selected for this review had to include subjects who met diagnostic criteria for lumbar facet joint pain with controlled diagnostic blocks leading to a minimum of 80% pain relief and the ability to perform previously painful maneuvers. All studies had to have outcome evaluations of treatment after at least 6 months. The primary outcome measure was short-term pain relief (up to 6 months) and long-term relief (greater than 6 months). Other outcome measures included functional status improvement, psychological status improvement, return to work, and the use of opioids. Only studies scoring at least 50 of 100 on the weighted scoring criteria were included for analysis according to the Agency for Healthcare Research and Quality criteria for observational studies and modified Cochrane review criteria for randomized trials. The literature search produced 1438 studies for review regarding therapeutic intra-articular facet joint injections, lumbar facet joint nerve blocks, and lumbar facet joint nerve RF neurotomy. There were only two of eight observational studies (Table 12.3) and one of seven randomized trials (Table 12.4) identified by the literature search for lumbar facet joint RF neurotomy that met the inclusion criteria.

Table 12.1 AHRQ USPSTF Quality of Evidence

I:	Conclusive. Evidence obtained from at least one properly randomized controlled trial or multiple properly conducted diagnostic accuracy studies.
II-1:	Strong. Evidence obtained from one well-designed controlled trial without randomization or at least one properly conducted diagnostic accuracy study of adequate size.
II-2:	Moderate. Evidence obtained from at least one properly designed small diagnostic accuracy study.
II-3:	Limited. Evidence obtained from multiple time series with or without the intervention. Dramatic results in uncontrolled experiments (such as the results of the introduction of penicillin treatment in the 1940s) could also be regarded as this type of evidence.
III:	Indeterminate. Opinions of respected authorities, based on clinical experience descriptive studies and case reports or reports of expert committees.

Adapted and modified from the AHRQ USPSTF. From ref. [16].

An observational study published by Dreyfuss et al. [20] consisted of 15 patients with chronic low back pain. There were 460 individuals who responded to announcements regarding this study through the local medical community, local newspapers, radio, and television. Telephone interviews were conducted with the 460 people who responded to the announcements, and 138 were identified to be eligible for the study. These 138 potential subjects underwent a physical examination, completed a pain drawing and Beck Depression Inventory, and lumbar spine x-rays if none had been done in the past year. These evaluations narrowed the field down to 41 persons who underwent a diagnostic medial branch blocks with 2% lidocaine of whom 22 had greater than 80% pain relief. The 22 volunteers returned a week later and underwent medial branch blocks with 0.5% bupivacaine. There were 15 of these 22 people who had 80% pain low back pain relief that established the diagnosis of lumbar facet joint low back pain.

All 15 patients then underwent RF medial branch neurotomy of the diagnosed lumbar facet joints. Outcome measures included visual analogue scale, SF-36 (physical function, bodily pain), Roland-Morris, McGill Pain Questionnaire, Beck Depression Inventory, floor to waist lift, above shoulder lift, push, pull, and treatment satisfaction were documented prior to and after the RF neurotomy.

These same outcome measures were repeated at 1.5, 3, 6, and 12 months after the medial branch neurotomy procedure. Needle electromyography was also performed on the multifidus muscle before and after the RF procedure to assess the accuracy of the neurotomy. There were 13 of the 15 patients who experienced at least 60% pain relief at 12 months and 9 of the 15 patients had at least 90% pain relief at the 1-year assessment. Pain relief correlated well with presence of needle electromyography findings of denervation potentials in the mutifidus muscles supplied by the medial branch nerves treated by the RF procedure. On the other hand, electrical stimulation of the medial branch nerve prior to the RF neurotomy of the medial branch nerves had no bearing on the outcome measures. Rather, the anatomical positioning of the RF probes prior to the neurotomy of the medial branch nerves was imperative to the success of the medical branch neurolysis.

The performance in lifting, push, and pull tasks improved slightly but not to a statistically significant degree. There was statistically significant improvement seen in visual analogue scale, SF-36, Roland-Morris, and McGill Pain Questionnaire scores. Fourteen of the subjects did not require or use any other type of treatment during the 1-year follow-up period. One individual had 18 chiropractic treatment sessions at 10 through 12 months post the RF neurotomy. This same person used codeine alternatively with hydrocodone for treatment of residual low back pain at the 12-month follow-up.

In the other observational study published by Gofeld et al. [21], a prospective assessment was conducted over

Table 12.2 Guyatt Grading Recommendations

Grade of Recommendation/ Description	Benefit vs Risk and Burdens	Methodological Quality of Supporting Evidence	Implications
1A/strong recommendation, high quality evidence	Benefits clearly outweigh risk and burdens, or vice versa	RCTs without important limitations or overwhelming evidence from observational studies	Strong recommendation, can apply to most patients in most circumstances without reservation
1B/strong recommendation, moderate quality evidence	Benefits clearly outweigh risk and burdens, or vice versa	RCTs with important limitations (inconsistent results, methodological flaws, indirect, or imprecise) or exceptionally strong evidence from observational studies	Strong recommendation, can apply to most patients in most circumstances without reservation
1C/strong recommendation, low quality or very low quality evidence	Benefits clearly outweigh risk and burdens, or vice versa	Observational studies or case series	Strong recommendation but may change when higher quality evidence becomes available
2A/weak recommendation, high quality evidence	Benefits closely balanced with risks and burden	RCTs without important limitations or overwhelming evidence from observational studies	Weak recommendation, best action may differ depending on circumstances or patients' or societal values
2B/weak recommendation, moderate quality evidence	Benefits closely balanced with risks and burden	RCTs with important limitations (inconsistent results, methodological flaws, indirect, or imprecise) or exceptionally strong evidence from observational studies	Weak recommendation, best action may differ depending on circumstances or patients' or societal values
2C/weak recommendation, low quality or very low quality evidence	Uncertainty in the estimates of benefits, risks, and burden; benefits, risk, and burden may be closely balanced	Observational studies or case series	Very weak recommendations; other alternatives may be equally reasonable

Adapted from ref. [19].
RCT, randomized clinical trial.

Table 12.3 Koes Modified and Weighted Cochrane Methodologic Quality Assessment Criteria

Criterion	Weighted Score	Nath et al. [22]
1. Study population	35	8
A Homogeneity	2	2
B Comparability of relevant baseline characteristics	5	2
C Randomization procedure adequate	4	4
D Drop-outs described for each study group separately	3	–
E <20% loss for follow-up	2	–
<10% loss for follow-up	2	–
F >50 subjects in the smallest group	8	–
>100 subjects in the smallest group	9	–
2. Interventions	25	20
G Interventions included in protocol and described	10	10
H Pragmatic study	5	–
I Co-interventions avoided	5	5
J Placebo-controlled	5	5
3. Effect	30	17
K Patients blinded	5	5
L Outcome measures relevant	10	10
M Blinded outcome assessments	10	–
N Follow-up period adequate	5	2
4. Data-presentation and analysis	10	5
O Intention-to-treat analysis	5	–
P Frequencies of most important outcomes presented for each treatment group	5	5
Total score	100	50

Adapted from ref. [17].

a 10-year period from January 1991 to December 2000 on patients who underwent RF neurolysis of the lumbar facet joints for the treatment of low back pain. All patients with a history low back pain of more than 6 months with or without nonradicular lower extremity pain were included in this study. The eligible patients then underwent a self-reported pain questionnaire, physical examination, and review of imaging studies. The patients underwent comparative double diagnostic blocks to determine the diagnosis of lumbar facet joint low back pain. Those diagnosed with lumbar facet joint low back pain based on the controlled medial branch nerve injections underwent RF neurolysis of the lumbar facet joints. The degree of pain reduction on a scale of 0% to 100% experienced by patients was recorded at 1.5, 6, 12, and 24 months after the RF procedure.

There were 209 patients who underwent RF neurolysis of their lumbar facet joints in this study for treatment of their low back pain. All follow-up data were collected in 174 of the participants in this study. The data from the remaining 35 subjects were not included in the final analysis because of insufficient data collection or loss to follow-up. There were 91 women and 83 men who made up the final 174 patients who completed this study. There were 119 of the 174 subjects who reported good to excellent pain relief at 6 months post the RF procedure. Forty-five (25.9%) patients were characterized as having excellent pain relief, with a greater than 80% reduction in pain. Seventy-four (42.5%) patients had good pain relief, with more than a 50% decline in pain. The remaining 55 (31.6%) of the 174 subjects were considered not to have a

TABLE 12.4 AHRQ Quality Assessment Criteria for Observational Studies

Criterion	Weighted Score	Dreyfuss et al. [20]	Gofeld et al. [21]
1. Study question	2	2	2
• Clearly focused and appropriate question			
2. Study population	8	5	5
• Description of study population	5	5	5
• Sample size justification	3	–	–
3. Comparability of subjects for all observational studies	22	8	8
• Specific inclusion/exclusion criteria for all groups	5	5	5
• Criteria applied equally to all groups	3	–	–
• Comparability of groups at baseline with regard to disease status and prognostic factors	3	–	–
• Study groups comparable to nonparticipants with regard to confounding factors	3	–	–
• Use of concurrent controls	5	–	–
• Comparability of follow-up among groups at each assessment	3	3	3
4. Exposure or intervention	11	8	8
• Clear definition of exposure	5	5	5
• Measurement method standard, valid and reliable	3	3	3
• Exposure measured equally in all study groups	3	–	–
5. Outcome measures	20	15	15
• Primary/secondary outcomes clearly defined	5	5	5
• Outcomes assessed blind to exposure or intervention	5	–	–
• Method of outcome assessment standard, valid and reliable	5	5	5
• Length of follow-up adequate for question	5	5	5
6. Statistical analysis	19	17	7
• Statistical tests appropriate	5	5	–
• Multiple comparisons taken into consideration	3	3	3
• Modeling and multivariate techniques appropriate	2	2	–
• Power calculation provided	2	–	–
• Assessment of confounding	5	5	2
• Dose-response assessment if appropriate	2	2	2
7. Results	8	8	8
• Measure of effect for outcomes and appropriate measure of precision	5	5	5
• Adequacy of follow-up for each study group	3	3	3
8. Discussion	5	5	5
• Conclusions supported by results with possible biases and limitations taken into consideration	5	5	5
9. Funding or sponsorship	5	5	5
• Type and sources of support for study	5	5	5
Total score	100	73	63

Adapted and modified from ref. [15].

significant response from the RF neurotomy, with pain relief lasting for less than 6 months.

In the group of 119 patients who experienced low back pain relief from the RF neurotomy, there were 81, 36, and 2 patients who reported pain relief for 6 to 12, 12 to 24, and greater than 24 months, respectively. The median pain relief for all 174 patients was 9 months, but the median pain relief was 12 months for the 119 patients who had good to excellent results for more than 6 months. All 119 patients had an increase in physical activities, and 99 of them were able to reduce their consumption of analgesics. The use of analgesics remained the same in the other 20 patients.

Nath et al. [22] completed a randomized controlled study of percutaneous RF neurotomy in 40 patients with chronic low back pain (20 active and 20 controls). All patients were examined by an orthopedic surgeon before and 6 months after the treatment (sham or active). Inclusion criteria were three separate positive facet blocks. Denervation was achieved by multiple lesions at each level in an effort to provide effective denervation. The active treatment group showed statistically significant improvement not only in back and leg pain but also back and hip movement as well as the sacroiliac joint test. Preoperative sensory deficit and weak or absent ankle reflex normalized ($P < .01$) and ($P < .05$), respectively. There was significant improvement in quality of life variables, global perception of improvement, and generalized pain. The improvement seen in the active group was significantly greater than that seen in the placebo group with regard to all the aforementioned variables. None of our patients had any complication other than transient postoperative pain that was easily managed. Our study indicates that RF facet denervation is not a placebo and could be used in the treatment of carefully selected patients with chronic low back pain.

SAFETY

No long-term complications or serious adverse effects to the patient have been described with RF facet ablation

procedures, when motor stimulation was performed before lesioning to prevent inadvertent ventral ramus or nerve root injury [23]. Patients can feel increased soreness and local pain especially in the first 3 to 5 days, but these symptoms usually disappear within 2 weeks. Other postoperative symptoms can include itching, burning, and hypersensitivity especially if the lateral branch is lesioned during the neurotomy that usually subside in approximately 4 to 6 weeks. Gabapentin, pregabalin, or tricyclic antidepressants can be very helpful for this condition. Improper needle placement without prior motor stimulation can potentially lead to permanent injury of the ventral or dorsal ramus, leading to limb weakness, permanent sensory deficit, or persistent neuritis.

Radiation safety is an aspect of this procedure that the physician must prepare for as is any other interventional procedure that involves fluoroscopic guidance. Proper shielding such as a lead apron, thyroid shield, glasses, and gloves should be used during the procedure. The use of pulsed fluoroscopy, collimators, and proper patient positioning will all limit radiation exposure. A dosimeter should be used on the hand, neck, and body to and changed on a quarterly basis to track radiation exposure.

DISCUSSION

The treatment of lumbar facet joint-derived low back pain with RF neurotomy of the medial branch nerves was assessed in a recent comprehensive systematic review of the literature [14]. Only three studies met the methodological quality assessment for inclusion in the analysis of the review. The three studies demonstrated both short-term (≤6 months) and long-term (>6 months) pain relief, increased function, and reduced medications use. Nath et al. also showed in their study that the benefits from the treatment of lumbar facet joint pain with RF neurotomy is not a placebo effect.

Although there was a limited number of studies from the literature for analysis, the level of evidence based on the two observational reports and the one randomized controlled trial was strong to moderate (II-2/II-3) for RF neurtomy in the treatment of lumbar facet joint pain. The recommendation for the use of RF neurotomy to treat lumbar facet joint pain based on Guyatt's criteria was strong (1B/1C), indicating that the benefits clearly outweigh the risk and burdens. These recommendations apply to most patients in most circumstances without reservations.

CONCLUSIONS

RF medial branch neurotomy is a valid, effective, and safe method for the treatment of lumbar facet joint low back pain. Although the literature is limited, the evidence and recommendations based on the analysis of the few publications that met the methodological quality assessment are strong for the use of RF neurotomy to treat lumbar facet joint pain. The studies show that RF neurotomy of the lumbar facet joints provides both short-term and long-term relief with improved function and a decrease in the use of pain medication.

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13 Diagnostic Imaging of Painful Sacroiliac Joints

Joseph D. Fortin, Sayed E. Wahezi, Douglas Mintz, and Anthony Chang

INTRODUCTION

During the turn of the 20th century, the sacroiliac joint (SIJ) was considered the leading cause of low back pain (LBP). This idea fell out of favor in 1934 when Mixter and Barr published their landmark paper describing herniated nucleus pulposus (HNP). However, in the last few decades there has been a resurgence of interest in SIJ-mediated LBP [1–4]. Perhaps this is because modern imaging studies such as magnetic resonance imaging (MRI), computed tomography (CT), bone scintigraphy, and arthrography are allowing us to examine the SIJ with previously unmatched detail [5–9].

Prevalence reports suggest that at least one-fifth of patients with LBP have an SIJ component [10,11] (see Chapter 1). However, SIJ pain remains a controversial topic in part because, the SIJ is geometrically and topographically complex [1,7,9]. Gender, body habitus, activity, and age-influenced osteochondral changes within the joint can make it difficult to distinguish a normal versus pathological condition [12]. Therefore, painful SIJs are not always radiologically evident and a decision to treat should not be based solely on imaging characteristics. A careful history and physical examination can assist to diagnose SIJ-mediated pain. Patients presenting with clinically suspected SIJ-mediated LBP based on the former and latter despite imaging corroboration are labeled to have "SIJ dysfunction" [1,13,14].

The SIJ is the largest joint of the spine and is central to human locomotion. It transfers loads between the trunk and lower extremities. Similar to other joints, biomechanically incompetent or structurally altered SIJs may cause pain.

ANATOMY

The SIJ is characterized as a large, auricular shaped, amphidiarthrodial synovial joint partially covered with hyaline cartilage and enveloped within synovium [1,7]. In the adult male, the SIJ has an average surface area of 17.5 cm^2 [1,15,16]. The sacral surface is lined with hyaline cartilage three to five times thicker than the iliac surface (3 to 5 mm:1 mm, respectively), offering the sacral surface relative preservation from degeneration; however, iliac subchondral bone is thicker than sacral, as displayed in Figure 13.1. Preferential sacral face erosion may indicate a nondegenerative pathological process. A network of ligamental connections encloses the SIJ. The posterior capsule is rudimentary or, in certain individuals, absent. The dorsal ligamentous structure forms the main structural bridge between the sacrum and pelvis via sacrotuberous and sacroiliac ligaments [17]. Some investigators have indicated that this ligamentous network functions to prohibit SIJ motion. However, cadaveric dissections, arthrograms, and postarthrographic CT scanning display redundant capsular tissue (recesses) teleologically suggested to permit joint movement [18,19] (see Figure 13.2).

The innervation of the SIJ has been debated for decades. Despite conflicting reports, most agree that S1-S4 dorsal rami innervation exists [4]. Cadaveric studies identified 26 nociceptive afferents within the posterior SIJ. The anterior SIJ contained two. Thus, ventral innervation may be insignificant toward the study of SIJ-mediated pain [4].

The sacrum consists of five dorsally and ventrally fused vertebrae. Dorsally, the fused spinous processes form the median sacral crest. The sacral hiatus is a defect

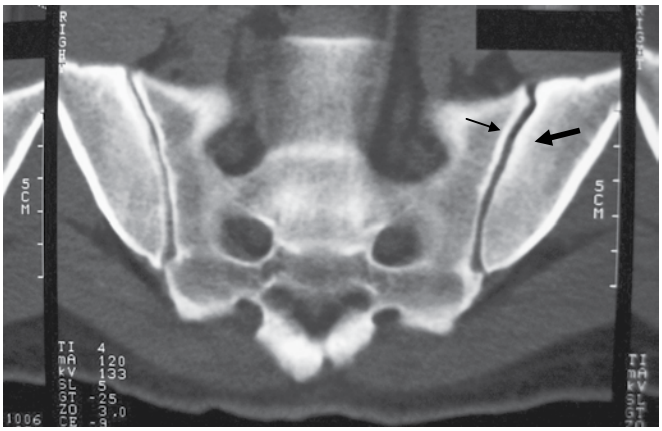

Figure 13.1 Coronal reconstruction CT of a normal SIJ demonstrating the relative thickness of the subchondral iliac (thick arrow) and sacral (thin arrow) faces. Note the opacity within the right joint margin because of contrast enhancement.

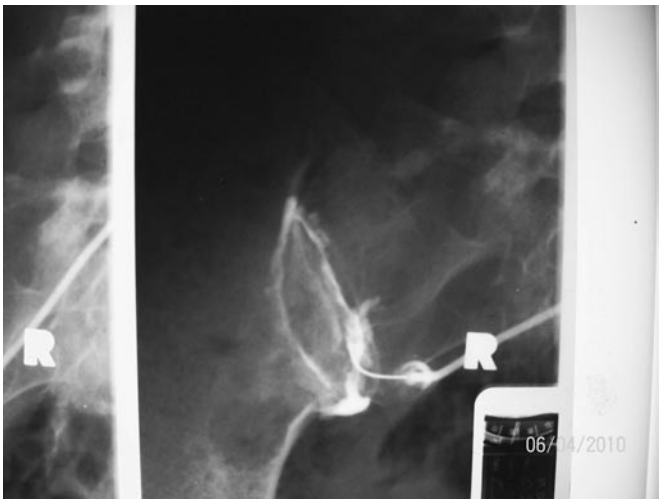

Figure 13.2 AP projection of SIJ arthrography displaying contrast pooling within the inferior SIJ recess (arrow).

in the dorsal wall of the sacrum at the S5 level. S1 is the largest sacral body and is densely populated with trabeculae arranged in a cruciate pattern. This histological design gives the S1 vertebra superior support during axial loading. Lateral masses are paired blocks of bone paramedian to the sacral bodies. Sacral compression fractures are common in this location because of low-density trabeculae [1]. The fractures are identifiable with MRI, CT, or bone scan [7] (see Figures 13.3A and 13.3B).

IMAGING MODALITIES

The four primary modalities used to image the SIJ are radiographs, CT, MRI, and bone scintigraphy. Each technique has its own strengths and limitations [9,17,20–24].

Plain radiographs are often recommended as the first imaging test when working up patients with suspected SIJ-mediated pain [9]. Radiographs allow a unique and inexpensive assessment of osseous morphology and texture, and can complement other radiological tests. For example, radiographically suspicious osseous changes can clue investigators in to performing CT or MRI versus scintigraphy for a more detailed examination. However, the complex configuration of the SIJ limits the sensitivity of early disease recognition [25,26]. The Ferguson view accommodates the SIJ architecture but has not proven to be superior to AP or PA radiographs. The patient is positioned supine and the tube is angled 25° to 30° cranially. With this projection, the symphysis pubis overlaps the sacrum. Individual joint visualization is then possible, as demonstrated in Figure 13.4. Unfortunately, each joint is oriented 40° to 60° dorsolaterally and is heavily invested with ridges. Given this uneven topography, radiographic analysis may only clearly display a portion of the joint [25]. Thus, there is no consensus regarding the radiographic evaluation of the SIJ.

CT uses radiographic technology to create cross sectional images. CT is more sensitive and specific than x-ray

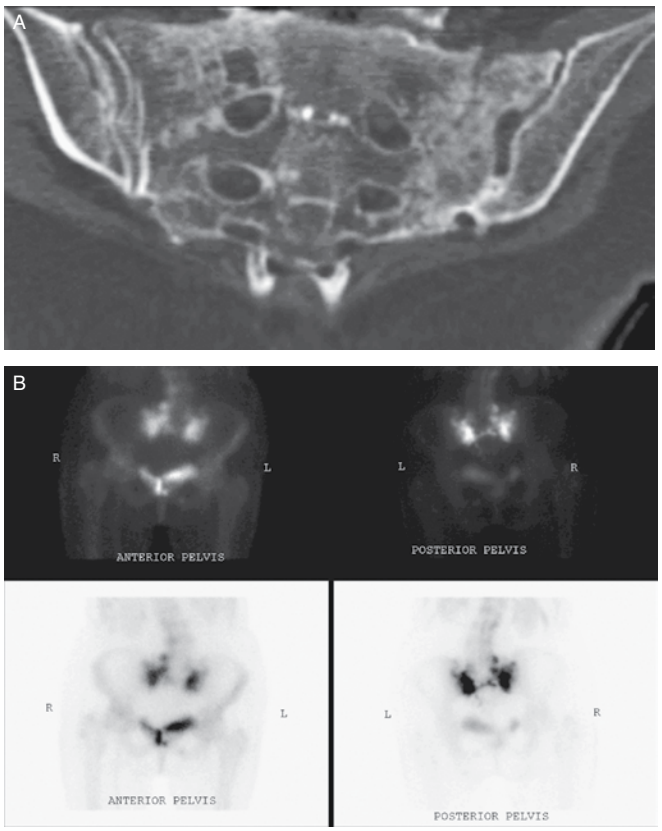

Figure 13.3 **(A)** Coronal CT of a 69-year-old female who presented with nontraumatic, sudden onset left sided LBP. Here we see a sacral fracture along the left S1 lateral mass (arrow). **(B)** Radionucleotide scan of a patient with bilateral sacral (arrows) and pubic rami (arrowheads) fractures. Note the increased uptake at both SIJs.

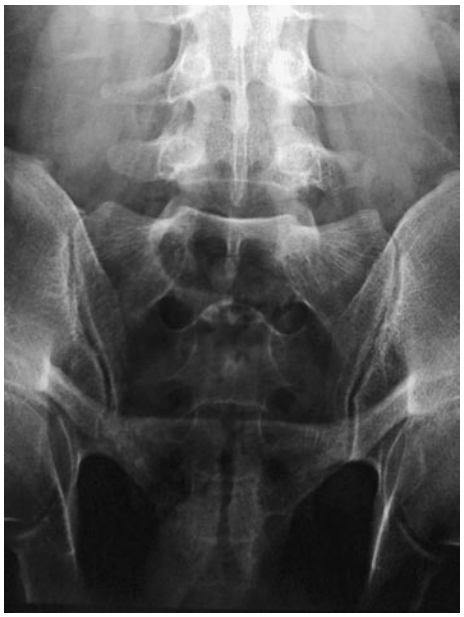

Figure 13.4 Ferguson view radiograph of a 38-year-old patient with normal SIJs. The patient is positioned supine and the tube is angled 25° to 30° cranially. This angle allows individual SIJ analysis.

for diagnosing subtle or complex architectural changes in patients with suspected SIJ disease [27]. Cross sectional anatomy also provides knowledge about intrapelvic pathology, which may mimic SIJ pain [28]. However, CT has three major limitations when used for SIJ diagnosis. First is the inability to demonstrate the acute changes that accompany inflammation such as synovitis and bone marrow edema. Second, the higher radiation dose (four to five times the dose of an x-ray) is a concern especially for young female patients, because their ovaries are within the primary CT beam. Third, outside of malignant disease or primary erosive and degenerative disorders CT has yielded equivocal results when diagnosing painful SIJs [17,20,29] (see Figure 13.5).

An MRI image is generated by exploiting the magnetic properties of protons in tissues. These subatomic particles are aligned by a superimposed magnetic field generated by radiofrequency pulse sequences and produce unparalleled soft tissue contrast. Hence, it is the preferred tool to view marrow space and nonmineralized tissue disorders. At times, the magnetic properties of specific pulse sequences or adjacent tissues may lead to distortion of osseous borders on MRI. Based on its distinct physical properties, CT provides greater contour resolution for viewing bone cortex and internal osseous architecture [20,24,30]. However, the detail of CT is achieved at the expense of exposing the patient to ionizing doses of radiation. MRI can be safely performed in most patients without risk of ionizing radiation.

Although MRI is the preferred modality for soft tissue analysis, thin ventral ligaments and discontinuous dorsal bands limit the utility of MRI for capsular investigation. Furthermore, while MRI may display degenerative marrow alterations, the characteristic cortical changes of degeneration are often obscured on MRI as variable signal intensity (within degenerative ridges), as seen in Figure 13.6. Although appropriate for vascular and inflammatory-mediated SIJ pathology, MRI has not shown to be helpful in the diagnosis and treatment of the vast majority of patients who present to Spine centers without these underlying problems, perhaps because most patients presenting with SIJ-mediated pain fit into radiologically occult classes [1,11,31]. Thus, osseous and capsular SIJ pathology are poorly defined by MRI and may be an important contributing factor limiting our understanding of SIJ-mediated pain.

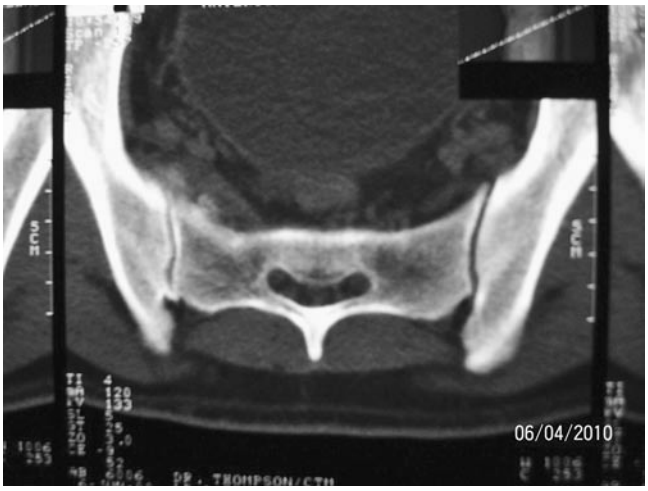

Figure 13.5 Right sided postarthrographic axial SIJ CT, displaying a normal SIJ on the left.

Bone scintigraphy displays increased signal in areas of inflammatory cell recruitment. Unfortunately, signs of metabolic upregulation remain long after active inflammation has ceased allowing the lines between acute, subacute, and chronic pathology to blur [32,33]. Moreover, although radionucleotide scanning rivals the sensitivity of MRI for inflammatory disorders, it lacks specificity. Hence, the scintigraphic changes of acute sacroiliitis and chronic degenerative changes may be indistinguishable. However, this imaging modality is not without value. Scintigraphy in Figure 13.7 displays characteristic signal enhancement of sacral fractures and the ability to selectively view focal and global anatomic pathology makes scintigraphy uniquely suited to easily evaluate metastatic lesions [9,17,20,26,34,35].

ARTHROGRAPHY AND SIJ DYSFUNCTION

The SIJ capsule can be assessed with arthrography [1–3,36]. The sacrum and pelvis are transfixed at multiple sites through a web of intricate ligamentous connections. Rents in the capsular ligaments may result from chronic imbalanced loading, high impact forces, or pelvic structural asymmetry [1–3]. Damage to these ligaments potentially causes LBP [6,13,29,37]. However, not all arthrographic changes are mechanical, because the normally thin ventral ligaments, and discontiguous dorsal ligamentous bands may allow contrast to seep from the capsule irrespective of mechanical loading to failure [36,38].

SIJ dysfunction is, in part, an abnormality of SIJ articular surfaces and ligaments [6,39]. Current noninvasive radiological techniques have proven to be poor identifiers of pain. This may be due to their inability to accurately assess perisacral ligaments and the inherent difficulty of recognizing normal SIJ articular surfaces [13,29,40,41]. Physical examination can assist a diagnosis of SIJ dysfunction but studies suggest that the clinical yield is moderate when used alone [42–45]. However, the Patrick's test, ischial compression, Fortin finger test, pubis compression, and Gaenslen's maneuvers assess SIJ integrity and can be adjunctive to increase the sensitivity of the radiological examination (see Chapter 3) [14,17,43]. Arthrographic capsular stimulation can identify SIJ-mediated LBP when used in conjunction with physical examination [2,3]. The author decides a patient's candidacy for SIJ arthrographic investigation if three of the five aforementioned tests are positive. Confirmation of SIJ involvement is then determined if provocative pain during physical examination rivals the symptoms elicited upon an intra-articular fluid challenge and they reliably experience pain relief after therapeutic intra-articular medication delivery [2].

The physical examination should corroborate any arthrographic capsular stimulation, because SIJ pain

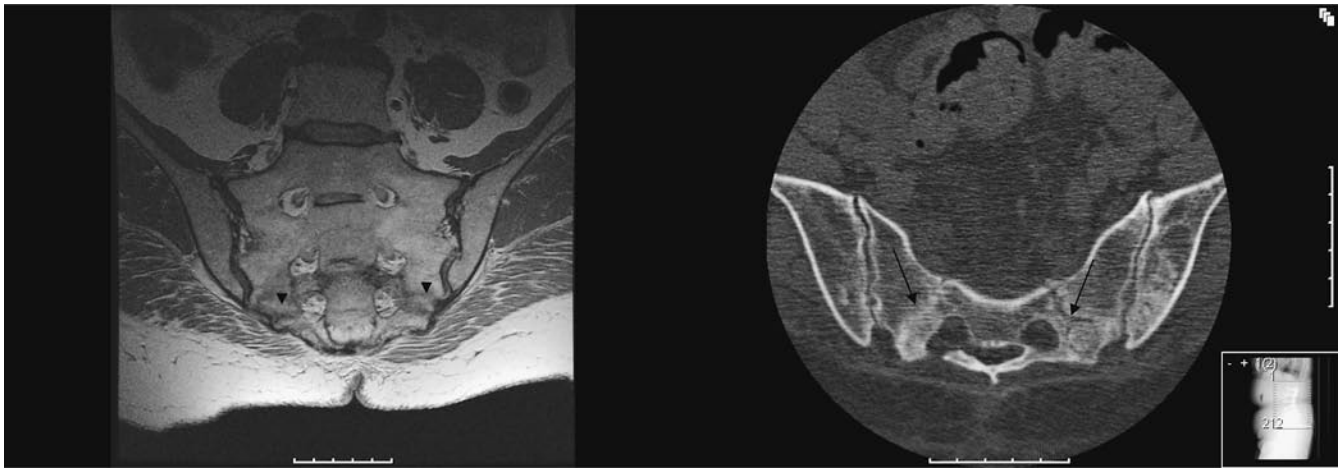

Figure 13.6 Left: Coronal T1 weighted longitudinal axis MRI of a fractured sacrum (arrowheads). Right: Axial CT scan of the same patient. Notice how corticomedullary defects are better visualized with MRI, yet cortical outlines are better seen on CT.

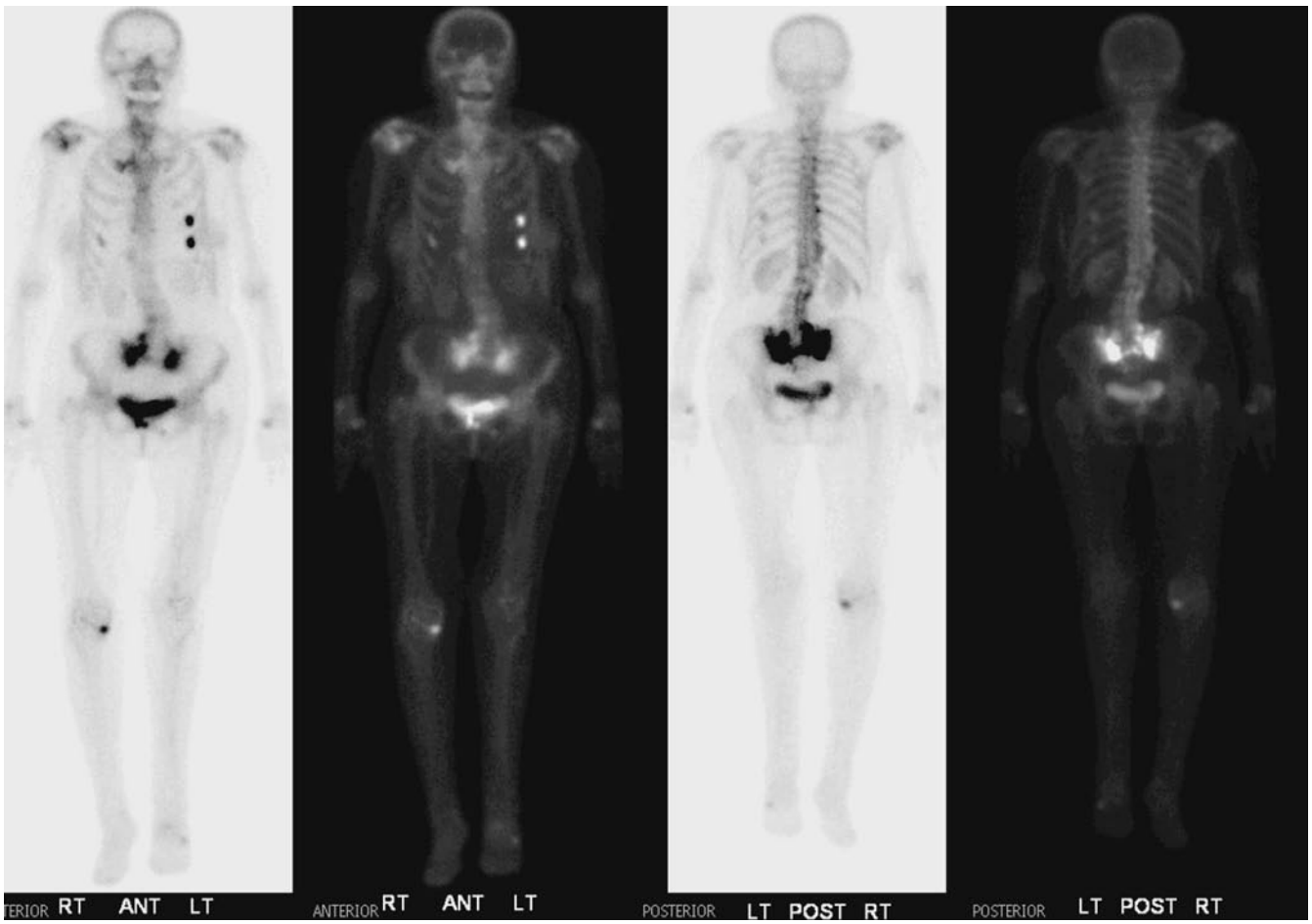

Figure 13.7 Scintigraphy of a patient with bilateral SIFs. Also, notice the nonspecific irregular uptake in the ribs, shoulders, and knee, which can represent degenerative, inflammatory, or metastatic lesions (arrows). Often a confirmatory MRI or CT is performed due to the lack of scintigraphic specificity. In this case, confirmation was done with the CT and MRI as seen in Figure 13.6.

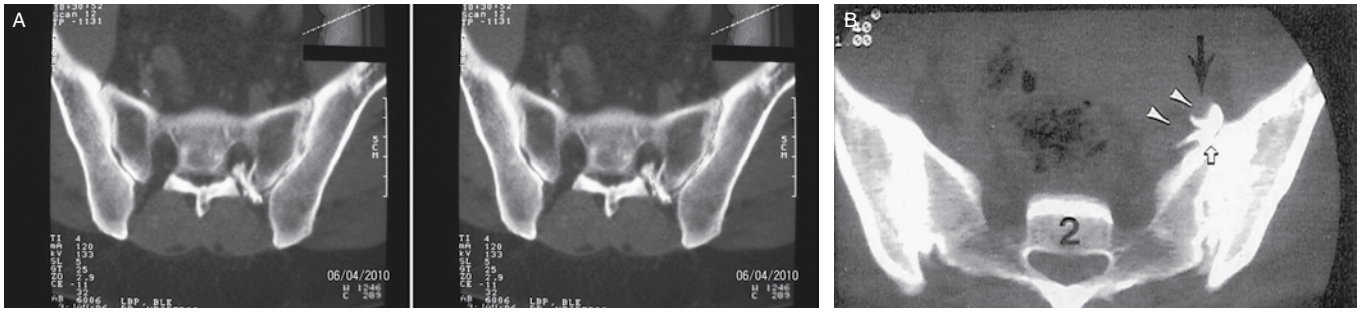

Figure 13.8 **(A)** Postarthographic axial CT of the pelvis displaying dorsal sacral foramina extravasation after left sided SIJ injection (left). The dashed line outlines the flow of contrast from the SIJ to the dorsal sacral foramina (right). **(B)** Axial postarthrographic SIJ CT scan of a 42-year-old male with LBP after bilateral injection of dye. Notice the ventral extravasation of contrast (arrows), indicating a ventral capsular tear.

referral patterns can mimic radiculopathy, bursitis, and facet joint arthropathy. This may be due to adjacent structures that are irritated in settings of SIJ capsular rupture [1,36,38]. Arthrographic distribution of contrast extravasation may explain the radicular pain referral patterns commonly seen in SIJ dysfunction [2,3]. For example, dorsal sacral foramina extravasation, shown in Figure 13.8A, may cause gluteal and trochanteric pain by irritation of the posterior sacral nerves; ventral rents may cause irritation of the lumbar plexus and cause posterior leg pain, as demonstrated in Figure 13.8B; finally, superior recess extravasation of contrast may affect the L5 nerve root and provoke leg symptoms [36].

Arthrography in Figure 13.9 outlines the SIJ and allows detailed, capsular investigation. The author uses a fluoroscopic guided inferior posterior approach to cannulate the SIJ, where the portal of entry is the inferior third of the joint. Rotating the C-arm obliquely by 5° to 10° past midline in both directions allows a better 3-dimensional perspective of the joint to select the "window" for optimal needle entry. This lucency in the inferior aspect of the joint is used as a target for needle placement and allows the least resistance upon needle passage. Following verification of initial needle position, contrast is injected to a volume commensurate with firm endpoint or fluroscopically visualized joint extravasation [2,3]. CT adjunctive investigation serves as modality to verify needle placement and prevent the misinterpretation of ligament disruption [28,46]. Postarthrography CT complements plain film arthrography and can serve as an invaluable tool to identify joint contrast extravasation in cases of degenerative joints with attenuated capsules (particularly those resulting in the pain referral patterns described earlier) [1–3,47,48].

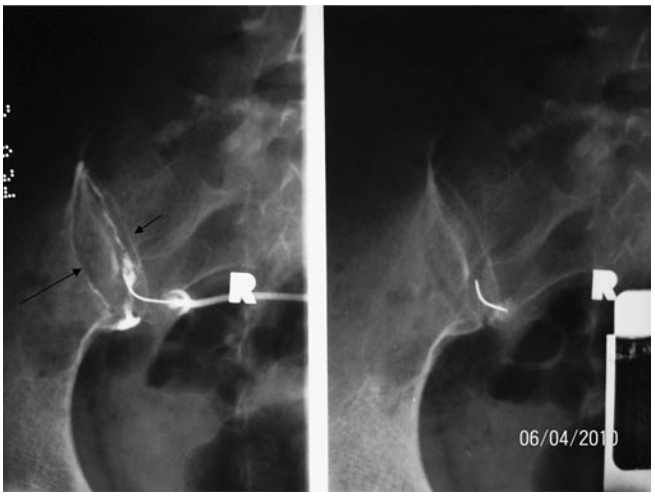

Figure 13.9 Right to left radiographic series displaying a 30° ipsilateral oblique modified Fenton projection. The SIJ arthrogram was performed using the inferior SIJ cannulation technique. Note how the oblique projection allows a clear image of anterior (small arrow) and posterior (large arrow) capsular integrity.

SACRAL AND SIJ PATHOLOGY

It is important to be able to recognize benign and malignant pathology when considering SIJ-mediated LBP. Following are some pathological entities encountered during SIJ radiological investigation.

Congenital SIJ Abnormalities

Transitional vertebrae are developmental variants of the spine, which usually affect the lumbar vertebrae. The L5 vertebrae can be "sacralized," wherein there are four true lumbar vertebrae and six sacral vertebrae. Accordingly, the S1 vertebrae can be "lumbarized," giving the appearance of six lumbar vertebrae and four sacral vertebrae. Some studies have demonstrated 30% overall incidence of lumbar anatomical variation [49].

The neural network is embryologically determined and is predicated by the myotome, not the ossified level. For example, in most cases the L5 nerve root exists above the sacrum; however, in a patient with a "sacralized vertebrae" (i.e., the patient has four unfused lumbar vertebrae) the L4 nerve exits directly above the sacrum. On the other hand, in a patient with a "lumbarized S1" (i.e., the patient has six unfused vertebrae) the S1 nerve root exits above

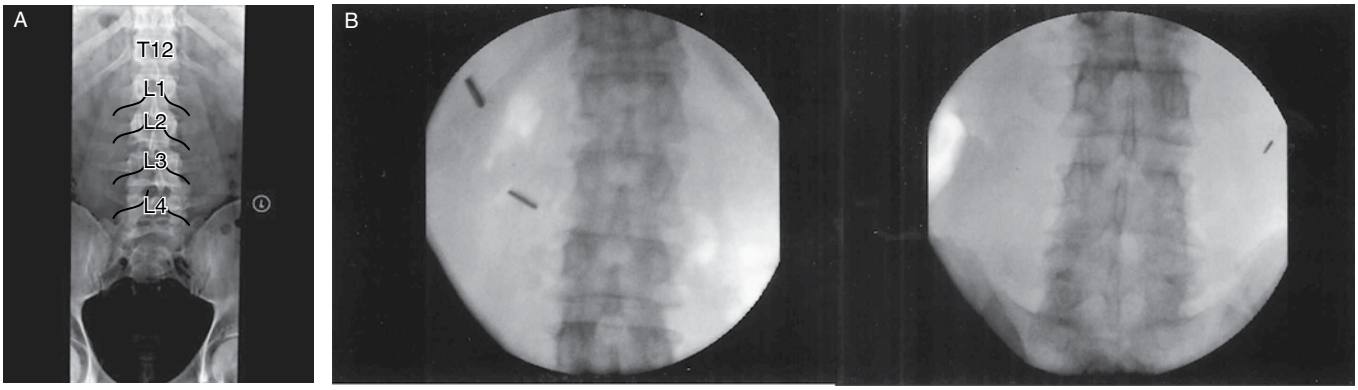

Figure 13.10 **(A)** Fluoroscopic image of a patient with a sacralized L5 vertebrae (the patient has four true lumbar vertebrae). The L4 nerve root exits directly above the sacrum, masquerading as an L5 nerve root (Image courtesy of Fritsch and Thompson, Radiologist, Diagnostic Radiology of Houston, Texas, USA). **(B)** Fluoroscopic image of a patient with a lumbarized S1. The S1 nerve exists directly above the subadjacent fused or immobile sacral segments. This anatomy changes the normal landmarks for performing facet blocks, discography, perineural injections, and dorsal rhizotomies.

the sacrum [50,51]. Figures 13.10A and 13.10B demonstrate both anatomical variations. When considering a procedure involving lumbar or sacral neural elements, it behooves the clinician to recognize lumbarization of the sacrum or sacralization of the lumbar spine to correctly perform the intended neurological block.

Accessory SIJs are formed from a rudimentary iliac transverse tuberosity usually lateral to the second sacral foramen [52]. It is a false joint remote and dorsal to the true synovial portion of the SIJ [52–54]. This area is a frequently attempted site for SIJ needle entry and may create an obstacle for proper SIJ cannulation. It is unknown if accessory SIJs are congenitally normal variants or whether they may be pathologically acquired fibrocartilagenous joints resulting from chronic weight-bearing stress.

Furthermore, accessory SIJs may not be rare. Reports suggest an incidence ranging from 4% to 16% [53,54]. The variability may be due to normal joint architectural and degenerative changes misinterpreted as accessory SIJs, imaging protocols, and the population base chosen for the studies [53]. Therefore, it is important for the interventional spine practitioner to be able to identify these anomalous structures before performing SIJ cannulation. An accessory SIJ is shown in Figure 13.11.

Divergent inferior SIJ articular surfaces, as demonstrated in Figure 13.12, are congenital SIJ variations, which can also complicate SIJ cannulation. Although this finding has not been reported, it has been encountered by the authors. The normal inferior SIJ is oriented caudomedially, whereas divergent joints are directed caudolaterally. Therefore, when needle access is attempted from the inferior SIJ it is important to determine whether the joint opens medially or laterally. The appropriate anatomical site of entry may not be easily seen on fluoroscopy because of degenerative SIJ and gas artifact. However, axial CT scans easily identify this variant. Although the incidence of the divergent inferior SIJ has not been studied, it is important to be aware that its presence can complicate SIJ access.

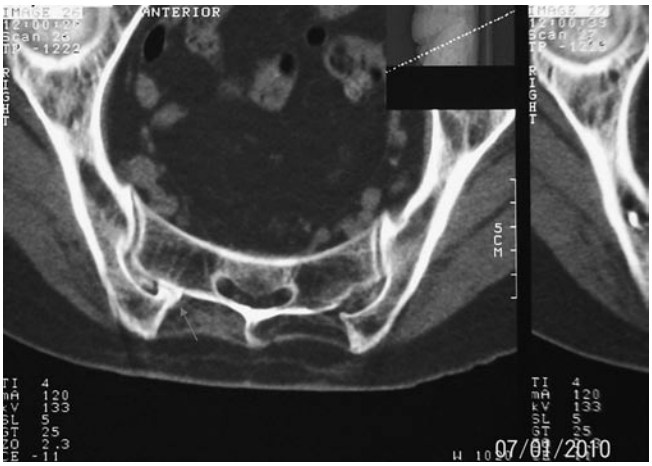

Figure 13.11 Right sided accessory SIJ (arrow). Accessory SIJs are typically found at the S2 level and can obstruct cannulation.

Congenital Dorsal Sacral Pathologies

Congenital dorsal sacral pathologies are indicated in the following section. It is important to recognize these entities because when executing most sacral injections, the clinician relies on posterior sacral surface landmarks to perform a myriad of sacral procedures such as transforaminal, intraosseous (i.e., bone biopsy and sacroplasty), and caudal injections. Posterior elemental irregularity can distort the clinician's visualization of sacral markers and can lead to misguided cannulation of sacral elements.

Sacral agenesis is also known as caudal regression syndrome. This condition is a severe form of abnormal sacral development and occurs in 0.005% to 0.01% of the population. It has mostly been observed in children of diabetic mothers. Associated abnormalities include: syrinx, lipoma, lipomeningocele, and cord tethering [55–57]. There are four types of sacral agenesis categorized by the severity of the agenesis. In type I, there is partial unilateral

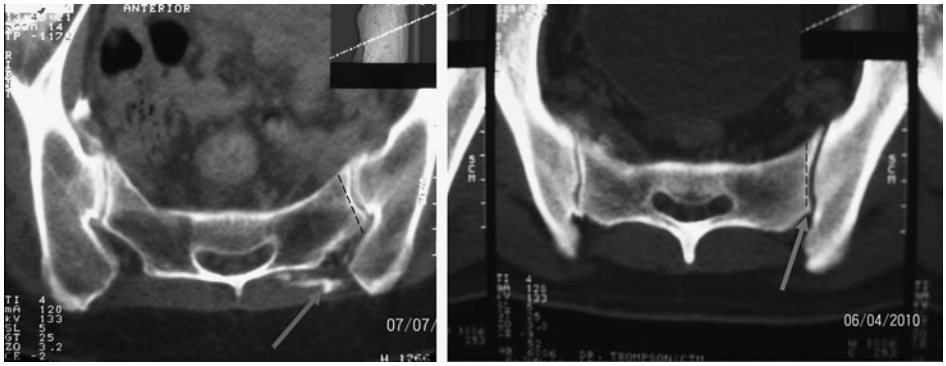

Figure 13.12 Left: Axial postarthrographic CT displaying bilaterally divergent SIJ (dashed line parallel to SIJ). Right: Axial postarthrographic CT in a patient with normally aligned SIJs (dashed line parallel to SIJ). The commonly employed medial to lateral approach of SIJ cannulation failed when attempting to cannulate the patient on the left (gray arrow displaying contrast material outside of SIJ). Superior or inferior cannulation techniques are recommended in patients with laterally divergent SIJs.

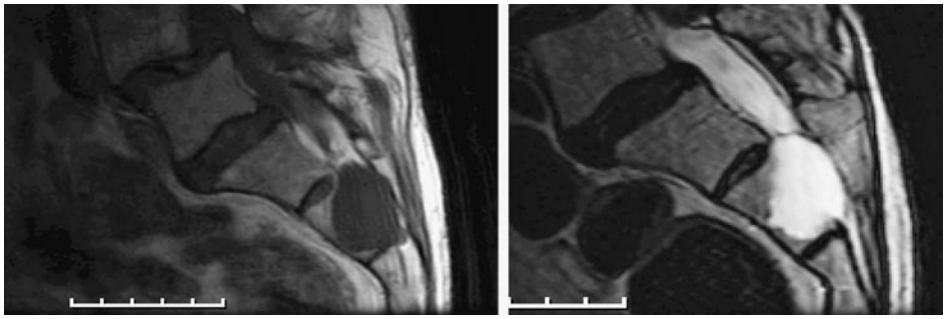

Figure 13.13 T1- (left) and T2- (right) weighted sagittal MRI of a patient with a Tarlov cyst (perineural cyst). Tarlov cysts are fluid filled sacs of spinal fluid and can cause pain if large enough to compress adjacent nerves or erode through bone, as demonstrated here.

agenesis localized to the sacrum or coccyx. In type II, there are bilateral symmetric defects in the sacrum; the iliac bones articulate with S1 and the distal sacrum, and coccyx do not develop. In type III and IV, there is total sacral agenesis [56].

Sacral meningoceles are described as ventral or dorsal osseous defects (dysraphism) that cause protrusion of membrane-lined thecal contents from the spinal canal. An anterior meningocele consists of herniation of cerebrospinal fluid (CSF) through a sacral defect or foramen. These disorders are embryological defects in ossification of laminar components during somite progression and chondrification [50,51]. Anterior meningoceles can manifest as symptomatic or asymptomatic pelvic masses, depending on their size. Posterior meningoceles are much more common and may be classified based on the contents of their herniated sac. Herniated contents can include meninges, spinal nerves, or spinal cord and can penetrate the skin (dermal sinus). Spinal cord herniation is not typically seen in adults unless the breach is in the midthoracic spine. In infancy, the tip of the spinal cord is positioned in the lower lumbar spine and then ascends to the upper lumbar spine as the vertebrae grows into adulthood [50]. MRI and CT are used to asses the soft and hard tissue defects. Complete spinal imaging may be necessary to properly assess any associated defects such as Arnold-Chiari malformations or cord tethering, as these may be a cause for LBP or headache.

Sacral meningeal cysts are abnormal dilations of the meninges within the sacral canal or foramina. They are reported to occur in 5% of the adult population and are often observed during incidental axial imaging of the lumbosacral spine [58,59]. Tarlov cysts are a subtype of meningeal cysts, which freely communicate with the subarachnoid space. Figure 13.13 demonstrates that these cysts can erode and remodel the sacral canal or sacral foramen. CT or MRI exhibits expansile and erosive deformation of the adjacent canal. Although the cysts are commonly asymptomatic, large cysts can manifest painful neurologic symptoms. MRI demonstrates that symptomatic cysts tend not to communicate with the subarachnoid space [56,58]. Percutaneous treatment strategies for symptomatic cysts include aspiration and fibrin glue therapy.

Osteoporosis/Osteopenia and Insufficiency Fractures

Sacral insufficiency fractures (SIF) are most often pathological fractures related to alterations in bone metabolism and consequently present following minimal or no trauma. Women above the age of 80 comprise more than 90% of all SIFs [60]. Corticosteroid-induced osteopenia and radiation therapy are also implicated as common risk factors, although all postmenopausal women, patients with a history of renal osteodystrophy, hyperparathyroidism, lumbar fusion, pregnancy, and Paget's disease are high risk groups. Furthermore, new evidence suggests that muscle imbalances, leg length discrepancies, and Trendelenberg gait may predict the type and location of SIFs. Therefore, the mechanics of ambulation correspond to varying areas

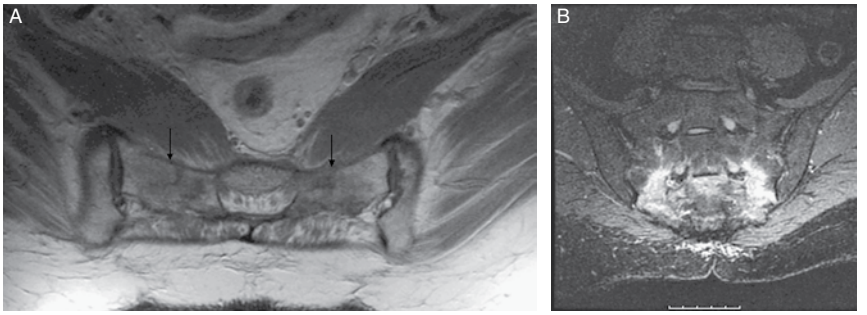

Figure 13.14 **(A)** Axial view of T1 MRI displaying bilateral SIFs. Edema is represented by a low-intensity signal on T1 MRI, whereas marrow and fat are represented by moderate or high-intensity signals. Note the low edema signal within the sacral cortex (arrows). **(B)** Coronal view of fat suppression MRI displaying bilateral SIFs at the lateral bodies of S2 and S3. Fat suppression images dull an otherwise high-intensity marrow signal, yet sustain the signal intensity of edema.

of SIF locations and can be predicted by biomechanical gait analysis [61].

The sacrum consists of five dorsally and ventrally fused vertebrae. Sacral bodies are densely populated with trabeculae and arranged in a cruciate pattern. The sacral lateral masses are paired blocks of bone paramedian to the sacral bodies with less dense trabeculae. This design creates stress concentrations at the sacral body-lateral mass junction [60,61]. Thus, the sacral lateral masses are the most commonly involved structure in SIFs. Low load stress to demineralized or low-density bone is thought to compromise its micro-architecture without evoking pain or inflammation. However, repetitive low-impact loads may disrupt the macro-architecture. The result is localized marrow edema and pain. Over time, healing reactive sclerotic change can be seen on CT and x-ray [60]. Thus, sacral fractures can appear as different histological entities depending on the time of diagnosis.

The Denis classification has been adopted from traumatic sacral injuries to aid in the prognosis and diagnosis of these fractures. According to the Denis classification, zone 1 fractures are the most commonly described SIF type and involve the sacral ala. Zone 2 involves one or more sacral foramen and affects the ipsilateral lumbosacral nerves. Zone 3 fractures affect the body of the sacrum and involve the central canal as well as bilateral lumbosacral nerves. Zone 3 fractures may present with saddle anesthesia and loss of sphincter tone (shown in Denis classification pic) [60].

Radiographs are usually inadequate to demonstrate SIFs before the development of healing calcification. SIFs can mimic osteoblastic or osteolytic disease, depending on their stage of healing. MRI is the most sensitive screening tool for SIFs. Nonhealed lesions demonstrate low signal intensity on T1 and high intensity on T2-weighted images, as shown in Figure 13.14A. On the other hand, Figure 13.14B demonstrates that sensitivity can be further improved with T2-weighted short tau inversion recovery (STIR) sequences. STIR sequences depress the fat signal within vertebral marrow and yield enhanced contrast to view normal and abnormal bone [20].

Bone scintigraphy is a sensitive, yet nonspecific technique to diagnose SIFs. Intravenous technetium-99 medronate methylene diphosphonate (MDP) is injected and uptake is measured by bone scan. These areas of uptake represent acute inflammatory sites and may correspond to a fracture. The classic "H" sign is shown in Figure 13.15, indicating bilateral inflammatory cell flares around the central canal [62,63].

CT is a useful alternative to the latter two diagnostic modalities when evaluating subacute or chronic SIFs, as demonstrated in Figure 13.16. Sclerotic healing and interdigitating fracture lines can be easily seen. Trabeculae can be visualized, excluding confounding diagnoses such as malignant or infectious disease.

Tumors

Tumors of the sacrum account for 6% to 8% of all clinically apparent spine tumors [64]. The majority of sacral tumors are secondary metastases of distant lesions. The most common primary malignant sacral tumor is the chordoma, accounting for more than 50% of primary sacral tumors. Giant cell tumors represent the most common benign lesion of the sacrum [64,65].

Radiographic imaging of tumors is limited by sacral curvature, frequent presence of overlying bowel gas, lack of sacral trabecular organization, and concomitant osteopenia. Radiographic tumor pathology specificity may be as low as 17%. Despite this, x-ray is considered the first-line imaging study after failed conservative therapy and continued pain. The Ferguson view has demonstrated 100% sensitivity for metastatic sacral disease [56]. However, clinical judgment should supercede convention; if the suspicion for malignancy is high, MRI or bone scintigraphy should be performed first.

MRI and CT are preferred visualization methods for sacral tumors, as trabeculae, cortex, edema, and other reactive tissue changes can be seen to aid the diagnosis.

Sacral tumors can be classified into four categories: congenital, primary osseous, metastatic, and primary neurogenic.

Congenital tumors of the sacrum include dermoid cysts, anterior and intrasacral meningoceles, Tarlov cysts, temartomas, hamartomas, and chordoma. Of these, chordoma is the most prominent. Chordomas represent 3% of all malignant bone tumors. Sacral chordomas represent 60% of all chordomas [64,66]. It is a remnant of the embryologic caudal end of the notochord and therefore is found in a midline or paramedian location. Chordomas are slow growing, locally invasive tumors that can reach a large size before causing symptoms. Slow tumor growth

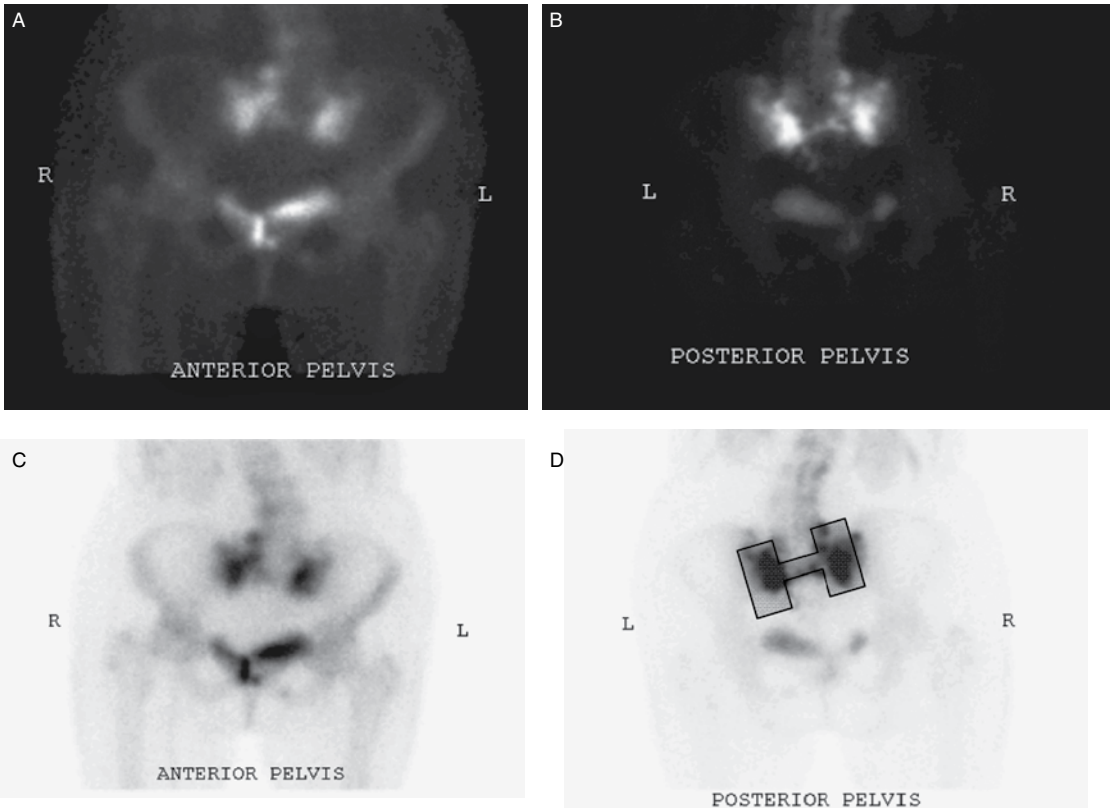

Figure 13.15 Bone scintigraphy of bilateral SIFs. Note the classical "H" sign outlined in image D.

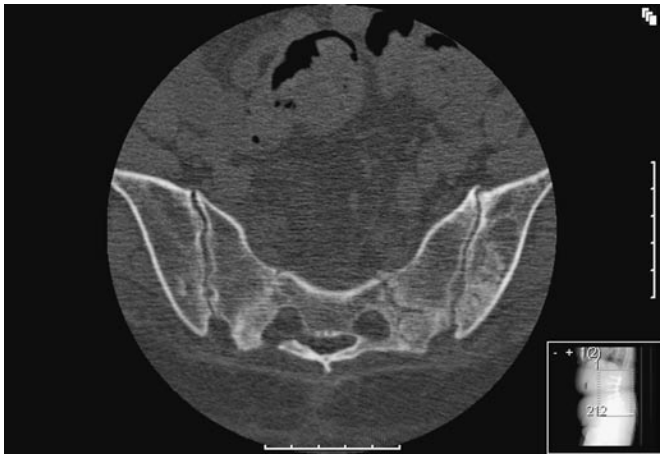

Figure 13.16 Axial CT displaying the cortical disruptions in a patient with bilateral sacral fractures (arrows).

denies the body of reactive inflammatory changes that would otherwise cause LBP, sciatica, constipation, or lower extremity paresis. Radiographic imaging usually displays a large lytic lesion with internal calcifications and adjacent soft tissue mass. T2 MRI findings, shown in Figure 13.17, include very high signal intensity, heterogeneous signal distribution, and enhancement with gadolinium.

Metastatic lesions are the most common neoplasm affecting the sacrum. These lesions are most often hematogenously spread from multiple myeloma, breast, lung, prostate, or colon carcinomas. Metastatic lesions are usually diagnosed earlier than primary lesions because rapid angiogenesis and subsequent growth causes painful reactive inflammatory changes within affected bone. Bone scintigraphy is the preferred imaging modality to screen for metastatic lesions because of its ability to evaluate inflammation within large anatomical regions [64–66]. However, lytic lesions may be obscure or indiscernible on scintigraphy.

Hematogenous bone tumors such as lymphoma, plasmacytoma, and multiple myeloma account for 18% of malignant bone tumors [67]. The distribution of hematopoetic bone marrow has a major impact on the site of malignant tumors. The sacrum, containing hematopoetic marrow, is a common site of metastases and hematological malignancy. Lymphoma is an aggressive lesion associated with extensive moth-eaten osteolysis, reactive sclerosis, and soft tissue invasion. Massive bony invasion on MRI and a large soft tissue mass on CT with relative sparing of the cortex suggests lymphomatous malignancy [65]. Figure 13.18 shows that multiple myeloma creates an aggressive lesion, creating diffuse multiple round lytic bony changes without reactive sclerosis on CT. Plasmacytomas are rare tumors, thought to represent solitary myelomas. A plasmacytoma is shown in Figure 13.19 [64–66].

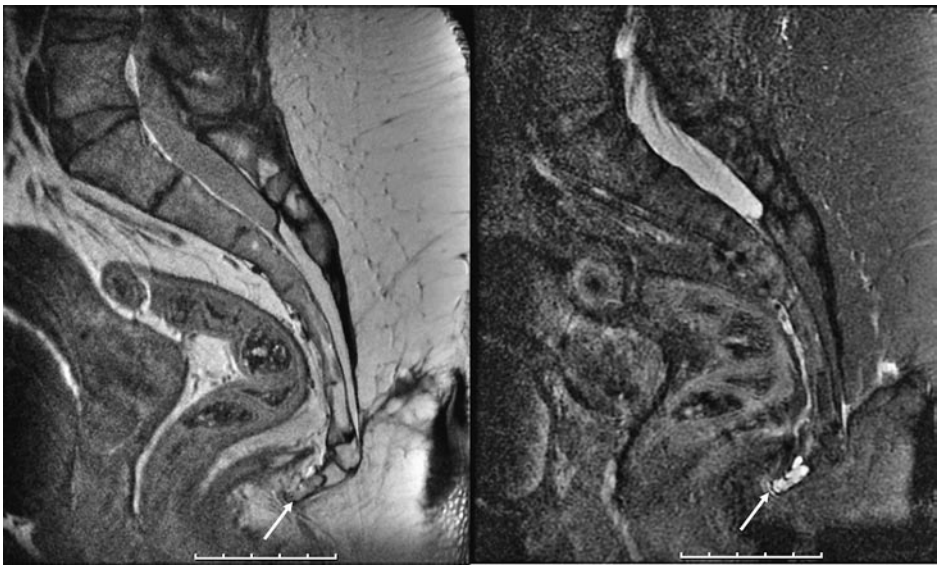

Figure 13.17 Sagittal proton density (left) and T2 fat suppression (right) weighted MRIs displaying chordoma (arrows). Chordomas are remnants of the embryologic caudal end of the notochord and therefore are almost always found in a midline or paramedian location in relation to the spine. High signal intensity and heterogeneous signal distribution on T2-weighted MRI is a classical feature of chordoma.

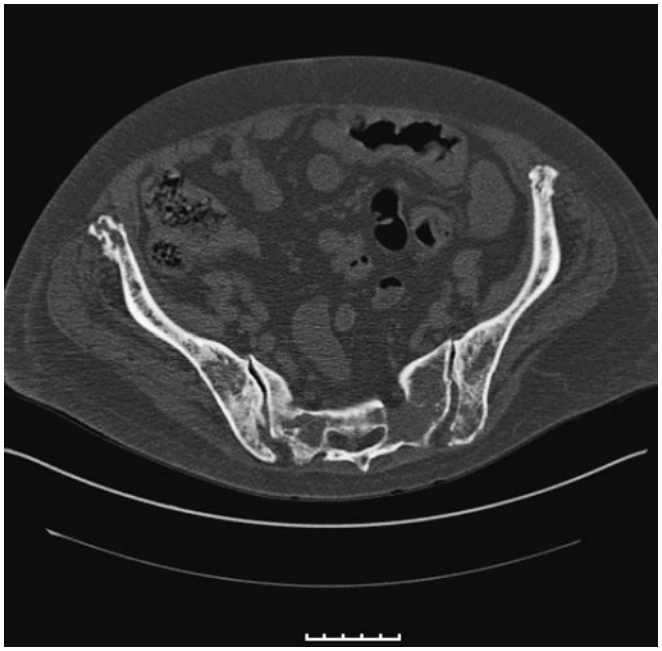

Figure 13.18 Pelvic axial CT scan in a patient with multiple myeloma. Notice the moth-eaten appearance within the sacrum and pelvis.

Primary osseous tumors are uncommon in the spine and sacrum. Less than 10% of all primary bone tumors are found in the axial skeleton. Histologically, these tumors are divided into different grades depending on their rate of growth.

Low-grade tumors include osteoid osteomas, osteoblastomas, osteochondromas, and aneurysmal bone cysts. Osteoid osteomas have a classic "olive pit" appearance owing to a small calcific nidus surrounded by a lytic ring with periphery of dense reactive bone. However, the dense centrum may be absent if the nidus is entirely lytic. This mineralized infrastructure is best observed by CT scan [64,65]. Osteoblastomas are lytic lesions with multiple focal calcific sites and a sclerotic rim due to reactive bone formation around the tumor. This is demonstrated in Figure 13.20. Variable sclerosis and expansile properties distinguish them from osteoid osteomas. T2 MRI displays a defining intermediate lobular signal distribution with adjacent soft tissue and marrow edema.

High-grade lesions include chondrosarcomas and osteosarcomas, both of which present with pain and radiographically visible invasive lytic lesions with arcuate, calcified, lobulated matrices and absent rim of reactive change. Chondrosarcomas have high signal intensity on T2 MRI [65]. Septated enhancement with gadolinium demonstrates the vascular properties of the tumor.

Giant cell tumors represent 60% of all benign bony sacral lesions. Giant cells are subchondral, locally invasive, and have a high rate of reoccurrence [68]. The most common presenting signs are pain and bowel/bladder dysfunction caused by mass effect. Upon surgical excision, the lesions appear heterogeneous because of necrosis, hemorrhage, and cystic spaces within the tumor. The CT in Figure 13.21 displays the lytic characteristics of a giant cell tumor, which crosses the sacral midline and is surrounded by a thin rim of reactive sclerosis. T1 and T2 MRI imaging has low signal intensity with heterogenous signal distribution and no edema [64,65,68].

Primary neurogenic tumors include schwannomas, neurofibromas, ependymomas, and ganglioneuromas.

Schwannomas and neurofibromas are benign lesions thought to arise from the neoplastic transformation of nerve sheath cells. They are both nonlocally invasive, slow growing encapsulated lesions often with dumbbell-shaped intradural and extradural components. The most common presenting complaint is LBP with associated radiculopathy in the distribution of the affected nerve.

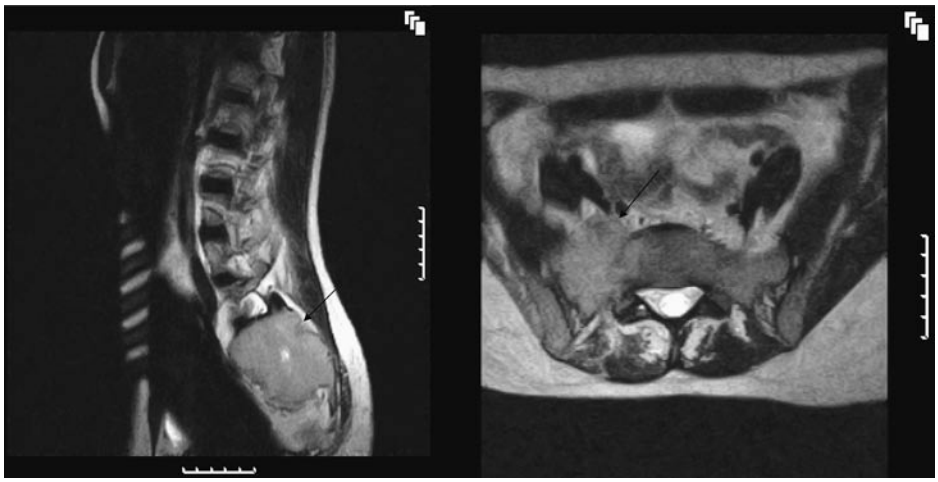

Figure 13.19 Sagittal (left) and axial (right) T2-weighted MRI displaying sacral plasmacytoma (arrows) with soft tissue invasion and obliteration of the right SIJ. Plasmacytomas are fast growing hematogenous lesions derived from plasma cells, thought to be solitary myeloma.

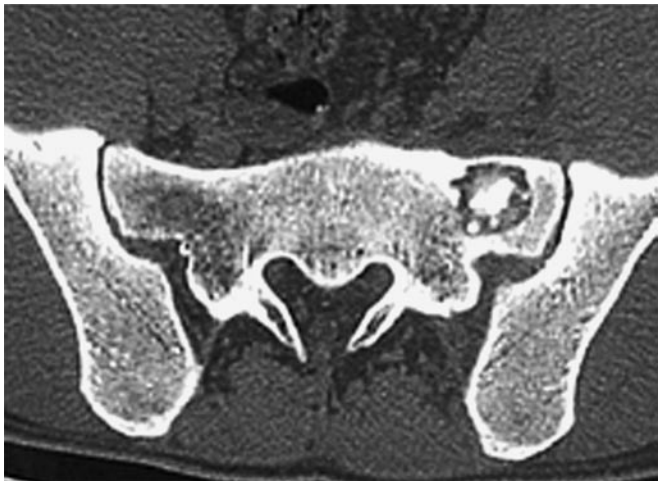

Figure 13.20 Osteoblastoma of the sacrum in a 37-year-old man with sacral pain. CT scan shows a hypoattenuating area with multiple central calcifications and minimal surrounding sclerosis. Courtesy of MH Rodallec.

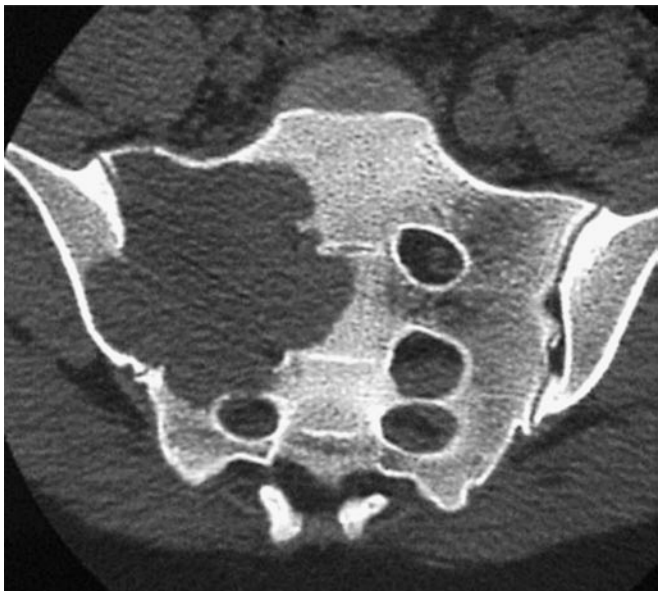

Figure 13.21 Giant cell tumor of the upper sacrum in a 33-year-old woman. Coronal reformatted CT image shows a well-defined lytic lesion of the right upper part of the sacrum with extension through the right sacroiliac joint and absence of a sclerotic rim. Courtesy of MH Rodallec.

T2-weighted MRI displays a homogeneous hyperintensity encasing the nerves, which defines their intradural component [56,65]. The homogeneity is due to intermingling cystic and necrotic regions of the tumor, whereas reactive edema is identified by high T2 signal, as seen in Figure 13.22 [63].

Ependymomas and ganglioneuromas are rare. They represent less than 1% of primary neurogenic tumors. Ependymomas are tumors of CSF production that may involve significant bone destruction [66]. Patients may present with LBP or radicular symptoms. Ganglioneuromas are slow-growing tumors of the sympathetic nervous system that usually arise in the abdomen but can also grow in the pelvis from sacral extensions of the sympathetic chain. They can extend through the sacral foramen and cause sacral nerve compression.

Nerve sheath tumors have a low signal center with high signal periphery on T2 MRI. They share heterogeneous signal distribution, have no enhancement with gadolinium, and have no edema [65].

Normal bone has well-defined margins on CT and displays a consistent trabecular pattern. Poorly delineated osseous borders seen on CT scan may indicate a malignant process. On the other hand, disruption of the trabecular arrangement, an intact cortical rim and absence of local tissue invasion suggest a benign entity [56]. Nerve sheath cell tumors may disrupt the bony borders of its exiting canal, causing irregular enlargement of sacral foramen. A large tumor with extensive osteolysis, heterogeneous necrotic cysts, and hemorrhage is suggestive of a giant cell tumor. MRI, which identifies a sacral tumor with a lobular high-intensity T2 signal

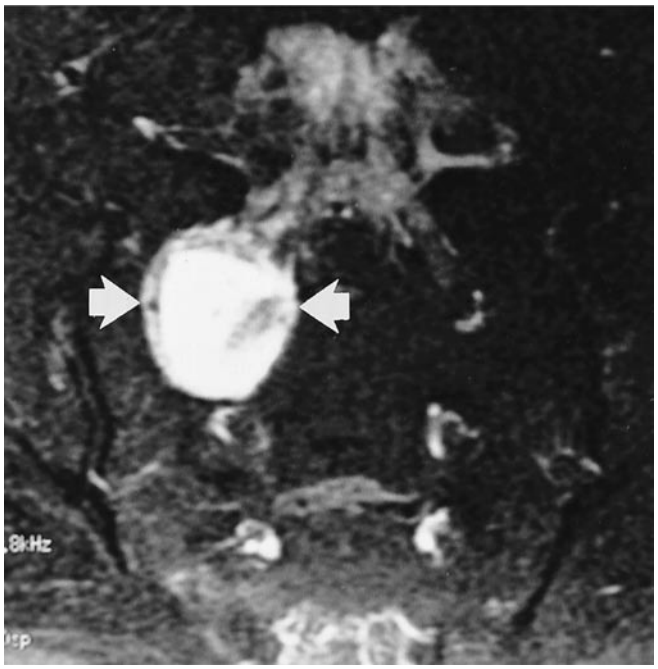

Figure 13.22 Sacral nerve neurofibroma in a 68-year-old woman with low back pain. Coronal T2-weighted MRI with fat suppression image shows increased signal intensity within an expansile neural foraminal mass (arrows). Courtesy of J Diel.

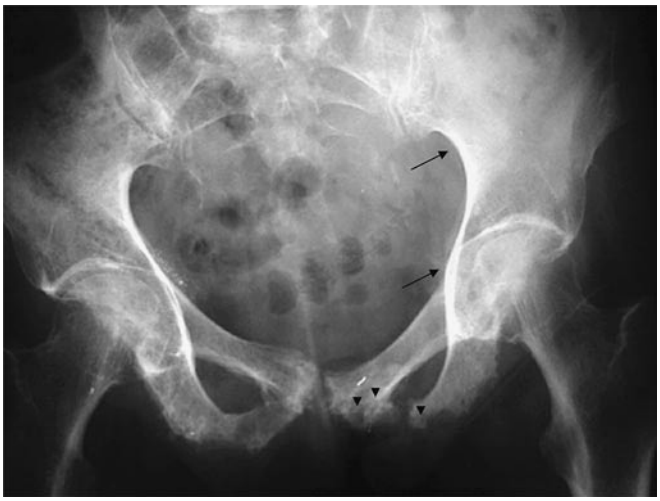

Figure 13.23 Anteroposterior pelvis view of a patient with late osteoradionecrosis of the pelvis and sacrum. The irregular osseous borders of the pubis are due to fatty trabecular infiltration (arrowheads). The sclerosis seen at the left pubic rim and acetabulum (arrows) are due to deregulated repair of radionecrotic trabeculae. Courtesy of University of Washington, UWMC Roosevelt Clinic, Musculoskeletal Radiology.

and contrast enhancement of the septa is suggestive of chondrosarcoma or chordoma.

Radiation-Induced Changes

The pelvis and sacrum are commonly included in the radiation field for prostate cancer treatment and gynecologic neoplasms. Radiation-induced changes of the pelvis include osteopenia, increased bone density, and SIJ widening and irregularity [64,66]. Skeletal complications include osteoradionecrosis, pathologic fracture, and radiation-induced neoplasms. Osteoradionecrosis of the spine results in the replacement of hematopoietic cellular elements with fat—a consequence of impaired osteoblast function. Radiographically, this is manifested as osteopenia because fat is poorly represented on x-ray. Osteoradionecrotic changes are high-intensity homogeneous signals on T1 and T2 MRI typically seen 1 year after radiation [35,69]. Several years after radiation therapy, the affected bone attempts to repair itself and results in bone deposition on ischemic trabeculae. The radiographs in Figure 13.23 reveal heterogeneous bone density with punctuate areas of increased density, osteopenia, and coarse trabeculation. Although dense bone is deposited, fractures are common in these areas because of bone absorption along non–weight bearing lines. Therefore, radiation is a common predisposing factor for SIFs [35].

Changes secondary to growth arrest in the spine may be seen following irradiation doses of 1000 to 2000 centigray (cGy). Horizontal growth arrest lines are identified 9 to 12 months after therapy. Irreversible irregularity of the vertebral endplate associated with decreased vertebral height become evident with doses of 2000 to 3000 cGy. Devascularizaion of bone occurs at doses above 5000 cGy [35].

Osteochondromas represent the most common benign radiation-induced tumor and occur exclusively in children below 2 years of age. Radiation-induced osteochondromas are radiologically and clinically identical to spontaneous osteochondromas. Radiation-induced osteosarcomas are a rare consequence of radiation therapy, representing about 2% of all primary bone sarcomas [66]. They can be more invasive than their idiopathic counterpart and appear similar on radiographic studies.

SIJ Arthropathies

The SIJ is characterized as a large, auricular shaped, amphidiarthrodial joint covered with hyaline cartilage and contained within synovium. The sacral surface is lined with hyaline cartilage three to five times thicker than the iliac surface (3 to 5 mm:1 mm, respectively), offering the sacral face relative preservation from sheer force and axial load degeneration during weight bearing [16,70].

Many inflammatory conditions can involve the sacrum by affecting the SIJ as part of a systemic or local inflammatory process. Examples include: ankylosing spondylitis (AS), inflammatory bowel disease (IBD), psoriatic arthritis, Reiter's syndrome, osteoarthritis (OA), rheumatoid arthritis (RA), and crystal deposition arthropathy. CT and radiographs are the preferred aids for visualizing associated cortical and sclerotic changes, whereas MRI is favored for staging reactive marrow space edema and

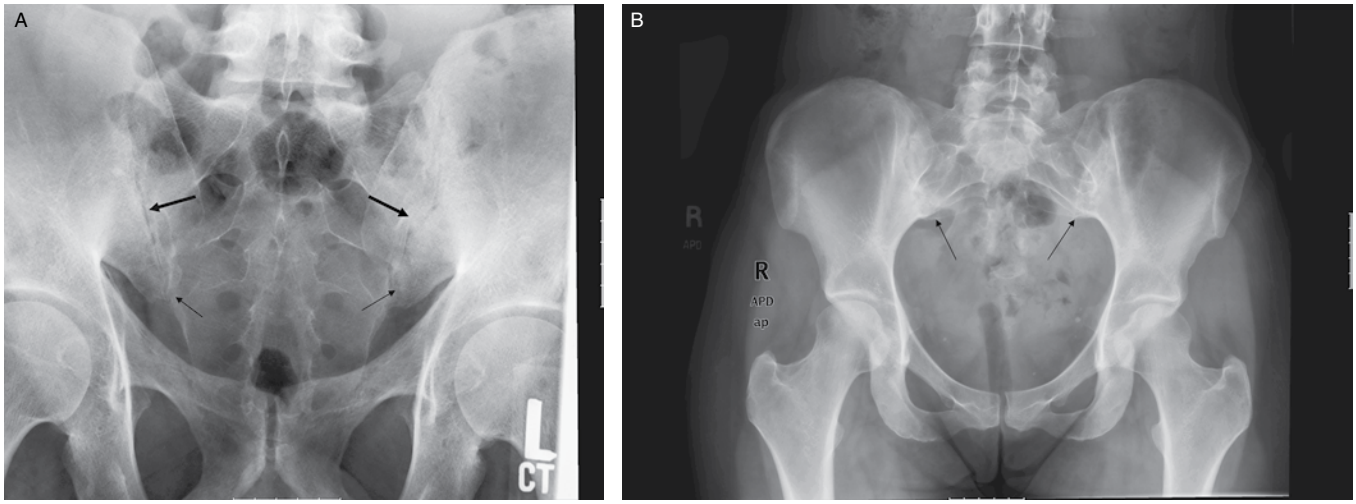

Figure 13.24 **(A)** Ferguson radiograph of a 30-year-old patient with AS who presented with a 5-year history of low back "stiffness." The x-ray displays symmetrical ankylosed inferior SIJs (thin arrows), joint space narrowing, and blurring of SIJ lines (thick arrows). **(B)** A case of IBD with symmetric SIJ ankylosis. Notice the blurring of iliac and sacral borders, especially at the inferior joint margins (arrows).

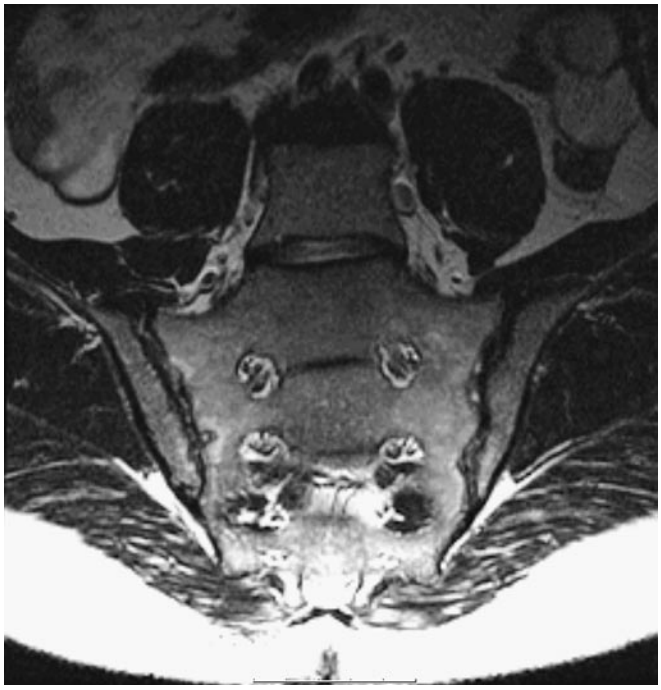

Figure 13.25 Coronal T2 MRI of the sacrum displaying symmetric hyperintense periarticular SIJs in a patient with acute ulcerative colitis (arrows). Perirectal hyperintensity is incidentally noted (arrowheads).

inflammation. Although nonspecific, radiographic imaging of arthropathies are made possible using a modified Ferguson view [20]. Periarticular erosions, subchondral sclerosis, and joint ankylosis can allow differentiation of the aforementioned arthropathies. In all of the aforementioned SIJ-modifying entities, the iliac side of the joint is affected to a greater degree than the sacral side because of thinner articular surface. It is postulated that the thinner permeable lining of iliac articular cartilage exposes the subchondral vessels to inflammatory mediators, recruiting and sustaining a local inflammatory response within the SIJ [22,30,71].

AS and IBD cause symmetric SIJ inflammatory pathology, as demonstrated in Figures 13.24A and 13.24B. Figure 13.25 exemplifies that acute and subacute inflammation appear as hyperintense periarticular zones on T2 MRI [71]. Erosive changes are characteristic early in the disease process, but later this leads to subchondral sclerosis and eventual ankylosis, as seen in Figure 13.26. This is best displayed with CT in Figure 13.27, as the SIJ margins blur with time. OA, psoriatic arthritis, and Reiter's syndrome are the only three noninfectious arthropathies that can present with asymmetric bilateral SIJ involvement. Figures 13.28A and 13.28B demonstrate these phenomenon. OA, seen in Figure 13.29, may present radiographically without erosions, although all may have a component of joint space narrowing, subchondral sclerosis, and osteophyte formation.

CT scan is preferred to visualize cartilagenous and cortical changes. However, the MRI in Figure 13.30 demonstrates the extent of intra- and periarticular inflammation.

Osteitis condensans ilii (OCI) is a disorder of symmetric iliac bone sclerosis commonly seen in postpartum females with LBP. It is a benign, and often painless, condition sometimes confused with AS on radiographs. However, CT can reliably differentiate between them if there is any doubt in the diagnosis. OCI is essentially a radiological diagnosis defined by triangular shaped nonerosive symmetrical sclerosis along the inferior iliac border of the SIJ. In OCI, axial CT sections display preserved

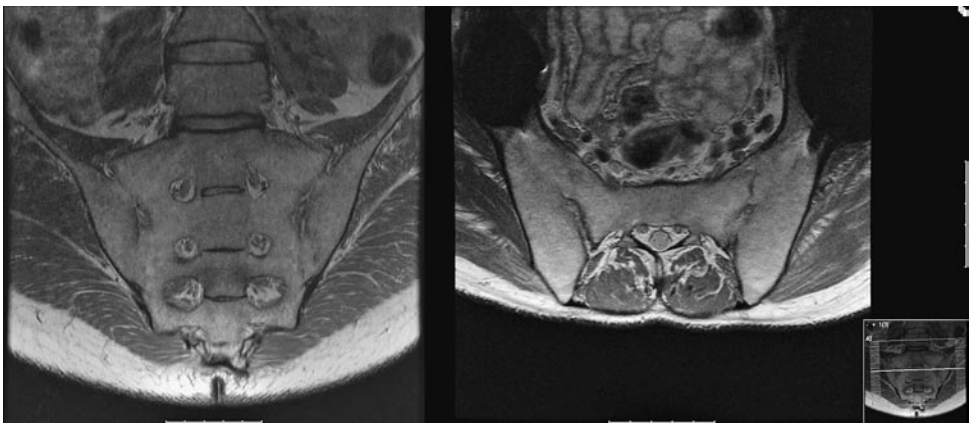

Figure 13.26 Coronal (left) and axial (right) T1 MRI displaying symmetrical SIJ ankylosis in a patient with AS.

SIJ space and articular margins, as seen in Figure 13.31, whereas ankylosis and sclerosis are seen in the spondyloarthropathies [72].

Infection

Infection of the SIJ is often due to spread from an adjacent infection. Pelvic abscesses can disrupt the anterior articular capsule of the SIJ or the periosteum and cortex of the ilium or sacrum. Infection may also spread from the bladder, intestines, genitourinary tract, or from intravenous injections in drug abusers via Batson's plexus [56,73,74]. Sacral decubitus ulcers, trauma, sacral biopsy, and iatrogenic injury from gluteal injections can also predispose patients to SIJ infections. Tuberculosis also has an affinity for the spine (Pott's disease) and the SIJ.

Infections of the musculoskeletal system are tuberculous in nature in 4% of cases. The SIJ is involved in approximately 10% of those cases [75]. Tuberculous sacroiliitis is

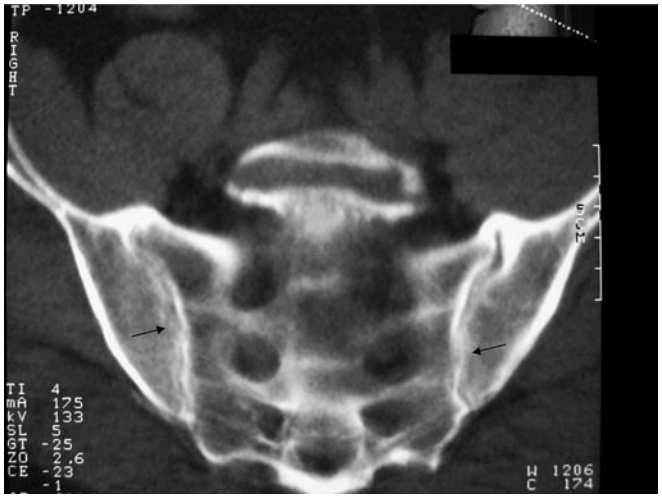

Figure 13.27 Axial CT in patient with longstanding AS displaying bilateral SIJ ankylosis. Notice the blurring of articular lines (arrows).

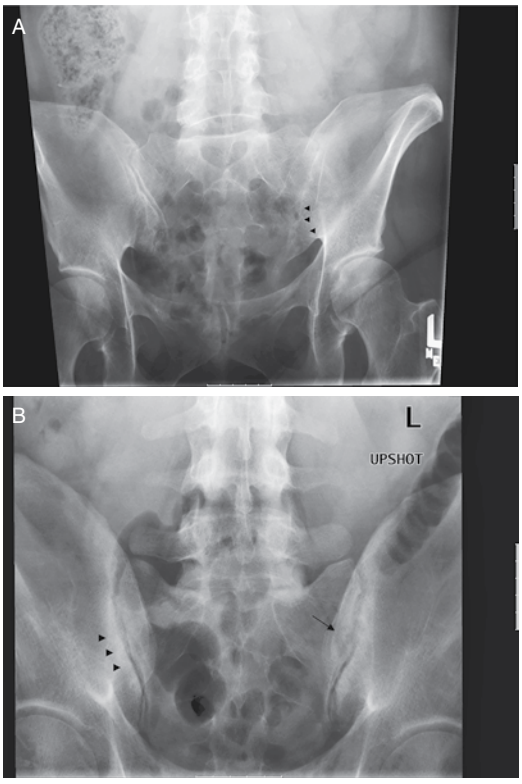

Figure 13.28 **(A)** Ferguson view x-ray displaying asymmetric SIJ involvement in this patient with longstanding psoriatic arthritis. Note the blurring of SIJ lines on the left (arrowheads). **(B)** Ferguson view radiograph demonstrating asymmetric subchondral sclerosis (arrowheads) and bony erosions (arrow) in a patient with Reiter's syndrome.

a noncaseating granuloma involving the SIJ. Radiographs may display erosive expansion and reactive sclerosis of a tuberculous infected SIJ.

SIJ infections are commonly caused by *Staphylococcus aureus*, a locally invasive pathogen with soft tissue digesting capacity. T1 MRI displays low intensity within infiltrated marrow and intermediate signal in fluid filled pockets. In Figure 13.32A, high-intensity signal is seen on

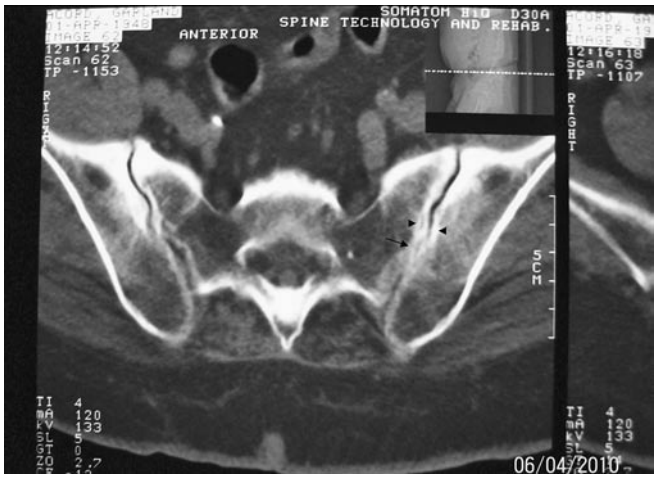

Figure 13.29 Axial CT scan of the pelvis in a 65-year-old patient with asymmetrical SIJ OA. Note the left sided subchondral sclerosis (arrowheads), joint space narrowing, and osteophyte formation (arrow).

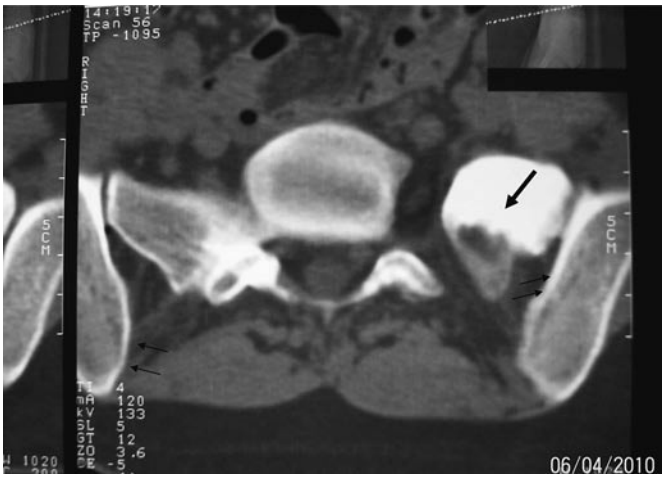

Figure 13.31 Axial CT scan of a young postpartum female with osteitis condensans ilii (OCI). Note the symmetrical rim of nonerosive iliac sclerosis (small arrows) and, in this case, the left lateral body of S1 (thick arrow).

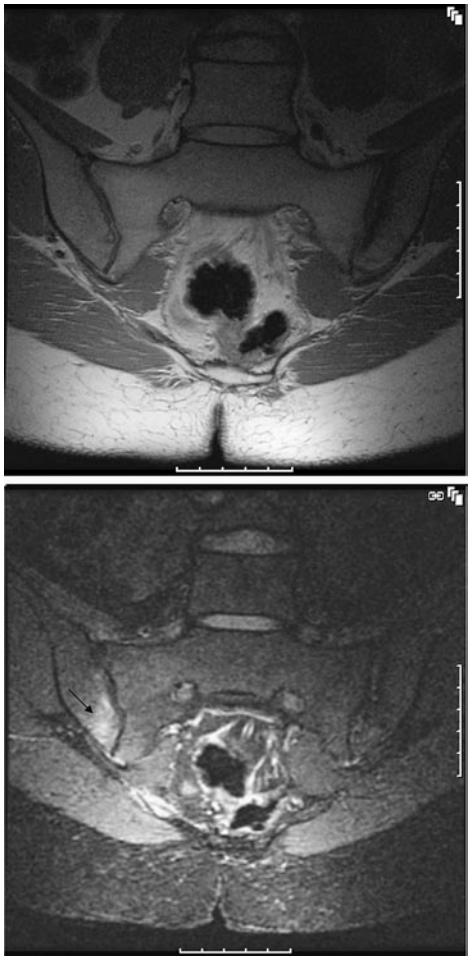

Figure 13.30 Axial T1 (top) and T2 (bottom) SIJ images of a patient with psoriatic arthritis. T1-weighted imaging defines SIJ architectural integrity. T2-weighted imaging highlights active inflammatory processes. Notice the preferential iliac face involvement (arrow) commonly seen in inflammatory SIJ conditions.

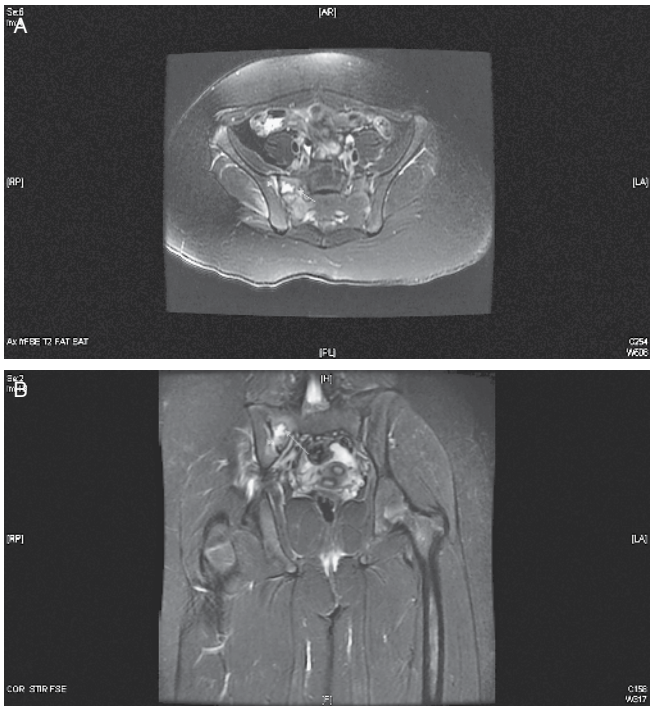

Figure 13.32 **(A)** Axial T2 fat suppression MRI of a 10-year-old female with right sided infectious sacroiliitis and osteomyelitis who presented with right sided hip pain and fever. Abnormal high-intensity signal is seen within the right iliac bone, sacral ala, and right SIJ (arrowhead). **(B)** T2 STIR coronal MRI of the same patient in (A). A high-intensity fluid signal is seen within the right SIJ (arrowhead) and the sacrum (arrow), representing pyarthrosis. (Image courtesy of Wahezi, DM, Children's Hospital at Montefiore Medical Center, Albert Einstein College of Medicine).

T2 MRI, representing areas of edema. STIR images, seen in Figure 13.32B, commonly demonstrate high-intensity signal at sites of infection and edema [75]. The SIJ normally harbors much of the infiltrate as it is a low-pressure site wherein inflammatory products collect.

SUMMARY

The osseous, ligamentous, and cartilaginous components of the SIJ can cause pain. Each requires a different radiological modality for proper evaluation and is sometimes complicated by anatomical variations. We propose that the SIJ is an under-recognized cause of LBP, in part due to our lack of appreciation for the anatomic and radiological complexity needed to understand this joint. Perhaps we need to reform our clinical looking glass in order to understand SIJ-mediated LBP.

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14 Diagnostic Blockade of Symptomatic Sacroiliac Joints

Jean-Yves Maigne

INTRODUCTION

For a long time, musculoskeletal medicine gave little or no recognition to the fact that the sacroiliac joint (SIJ) might be the possible source of nonspecific low back pain (LBP) [1]. According to the osteopathic literature on the other hand, up to one third of occurrences of LBP could be directly related to such dysfunction [2]. In another study of 1293 patients, the prevalence of LBP resulting from SIJ dysfunction was 22.5% [3]. The only way to provide an objective answer to this question was to carry out diagnostic blockade of the putatively symptomatic SIJ. The first study, published in 1995, was that of Schwarzer et al., [4] who performed diagnostic sacroiliac blocks (SIJB) on 43 unselected patients with LBP, 30% of whom afterward expressed a "gratifying relief of their pain." The second was that of Maigne et al., who selected 54 patients with LBP in the form of pain in one buttock only and carried out an initial block with lidocaine. Then, if this was positive, a confirming block of bupivacaine was carried out 7 days later to limit the false positives relating to a placebo effect [5]. Eighteen percent responded to the two successive blocks (and 35% to the first) with more than 75% pain relief as measured on a visual analog scale (VAS), confirming the place of the SIJ as being one of the possible sources of nonspecific lower back pain, even if the exact prevalence of this syndrome is still under debate. To date, anesthetic block remains the standard treatment for a diagnosis of pain originating in the SIJ. The technique, its complications, its indications, and interpretation of the results are what we are going to discuss in the present chapter.

THE TECHNIQUE OF DIAGNOSTIC BLOCKADE OF THE SIJ

A technique of injection into the upper part of the joint was described in 1938 [6], but the approach currently used, by puncture in the lower part, dates from 1979 [7], and the injection of radio-opaque contrast medium to check the correct position of the needle dates from 1982 [8].

Technique

If an anesthetic block is to provide results which can be properly interpreted, it needs to be carried out at a time when pain is present. The patient is therefore asked to do everything he or she can to provoke the customary pain on the day the block is to be carried out. No analgesics or anti-inflammatory medication should be taken in the morning, and potentially painful situations should not be avoided. Before the injection, a VAS test needs to be carried out, with scores from 1 to 10 according to the intensity of pain during the most painful position or activity at that time. It could be walking, going up or down stairs, standing, or any other position or posture. This needs to be explained clearly to the patient.

The patient should be placed face down. The lower part of the SIJ space must be visible. If it is not clearly visible, a cushion should be placed under the contralateral anterior superior iliac spine. The effect of this simple axial torsion is to place the SIJ space to be reached in the sagittal plane. For heavily lordotic subjects, it is also possible to turn the beam of the x-rays toward the feet so that the beam is perpendicular to the sacrum, or to place a cushion under the stomach. A metal marker placed on the skin will enable the puncture point to be seen clearly, approximately 1 cm above the tail end of the joint space (illustrative figure). The skin should be prepared and disinfected. We generally use a 22-gauge (0.7 mm) 5-cm-long intramuscular needle. For obese patients, a 10-cm spinal needle will be required. A local anesthetic is not useful. If given, it should remain superficial because anesthesia in the depths of the ligament and capsule would hinder interpretation of the results of the block. The needle passes successively through the skin, the most medial fibers of the gluteus maximus muscle, and the dorsal sacroiliac ligament. It then penetrates the joint. The implementation is not always easy, as the injection of contrast medium shows. The needle can remain outside the joint if an osteophyte (invisible on the anteroposterior view) bars its way. It can also enter the capsule but become positioned in the joint cartilage (quite thick), or a little too far forward, in the very thick ventral sacroiliac ligaments. An injection then becomes impossible. Last, it can be intraosseous, particularly in elderly patients whose bone is osteopenic. In this case, injection is possible but no trace of intra-articular SIJ contrast medium appears. Instead, a periosteal or intramedullary contrast pattern becomes apparent (illustrative figure).

If positioning is not correct, the operator should start by pivoting the needle bevel 90° or 180° to prevent it from

possibly becoming plugged. If he or she is still not in the joint, the needle should be withdrawn 1 to 2 mm very slowly, until the injection flows easily. This move, often effective, means that the needle was positioned too anteriorly. This is the most frequent cause of error. The hip joint can also be positioned in maximum internal rotation which, in theory, has the effect of separating the SIJ edges. Despite all these precautions, approximately one in 10 injections is not into the joint. This means that the operator needs to place the needle a little higher or a little lower, or be satisfied with an extra-articular injection (which limits the interest of the block). The proceduralist then injects approximately 0.5 to 1 cc of radiographic contrast medium which will fill the whole joint from back to front and from bottom to top (Figure 14.1) but which sometimes stagnates in the caudal end. Ideally, the least possible amount should be injected, to leave as much space as possible for the anesthetic, but the operator often desires to obtain an excellent arthrogram. Two percent lidocaine (approximately 1–2 cc) is then injected. The SIJB is finished. If so desired, a periarticular steroid can be injected in association. The needle should be withdrawn a few millimeters so that it is injected alongside the joint.

Postblock Evaluation

The patient gets dressed and waits approximately 15 minutes before trying to provoke the customary pain again in all ways possible and to give it a VAS assessment. Questioning will enable assessment of improvement, or will reveal absence of improvement. We have considered the figure of a 75% decrease in pain as testifying to a positive block [5]. Telephone contact of the patient within 24 hours can be useful, because some improvements are felt, or better assessed, with a delayed effect. Last, the effect of the steroid, determined 3 to 4 weeks later, is interesting to consider but much less specific.

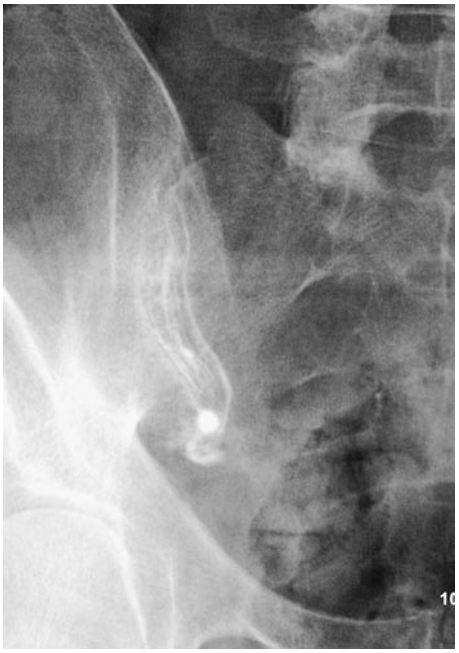

Figure 14.1 Correct positioning of the needle in the right sacroiliac joint after injection of contrast medium. The thickness of the cartilage and the slenderness of the joint space should be noted.

COMPLICATIONS AND CONTRAINDICATIONS

The complications of diagnostic SIJBs are those of any infiltration, that is, allergy to iodine and infection. There are practically no painful postinjection reactions. Pregnancy is an absolute contraindication. Although essential, this contraindication is detrimental to our knowledge, for it is probable that a certain amount of lumbosacral pain during pregnancy originates from the hypermobile state of the SIJ.

ASSESSMENT OF RESULTS

A number of factors make the assessment of the result of an anesthetic block difficult. They are in particular responsible for false positives.

False Positives

The first is the placebo effect. In the study by Maigne et al., [5] 19 patients out of 54 found pain relief with an initial block, but only 10 out of 19 found relief with a second block 7 days later. In almost one out of every two cases, therefore, the first positive response could not be duplicated by the second diagnostic SIJB. This figure is very high and one that, we believe, is not taken into account enough in the articles published about diagnostic SIJBs. It means that one should not put a blind trust in these blocks because the rate of positive response can be overestimated.

Communication between the joint cavity and the neighboring nervous structures has been observed: ventral leakage toward the lumbosacral plexus (16% of arthrographies, Figure 14.2), dorsal leakage toward the first sacral foramen (8% of arthrographies), or upper leakage toward the L5 root (3% of arthrographies) [9]. It is not possible to say whether they preexisted the arthrography, or if they were provoked by the injection under pressure temporarily tearing the capsule or a diverticulum (Figure 14.2). The role is diversely appreciated. For Fortin et al., [9] who described it, this communication may explain the referral of pain away from the SIJ into the lower limb through the leakage of the chemical mediators of inflammation outside the joint. Conversely, for Berthelot et al., [10] it could be a possible source of false positives during SIJB because the leakage is likely to relieve authentic lower limb radicular or sciatic neuropathic pain. The sciatic nerve does indeed pass less than 10 mm in front of the inferior part of the sacroiliac space [11]. Although the presence of communication between the joint and the sheath of the sciatic nerve does not ensure that the contrast medium reaches far enough cephalad to the lumbosacral junction, it does emphasize the importance of checking the absence of a lumbar source for the pain before making a decision to perform a SIJB.

Third and last, the difficulty of assigning a pain intensity score on the patients level of LBP, which may require

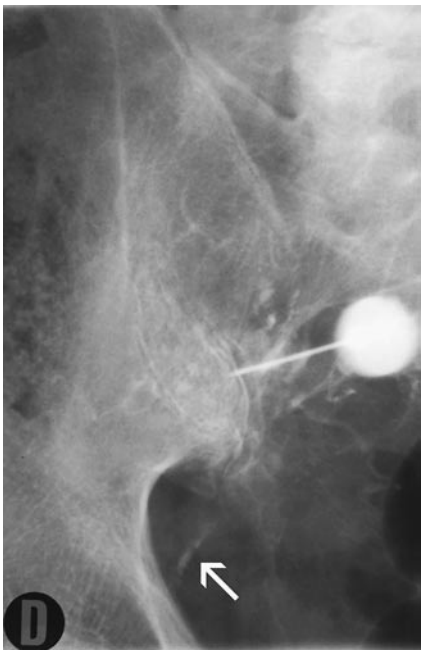

Figure 14.2 Diverticular aspect of the joint. Possible leakage of contrast medium along the sciatic nerve.

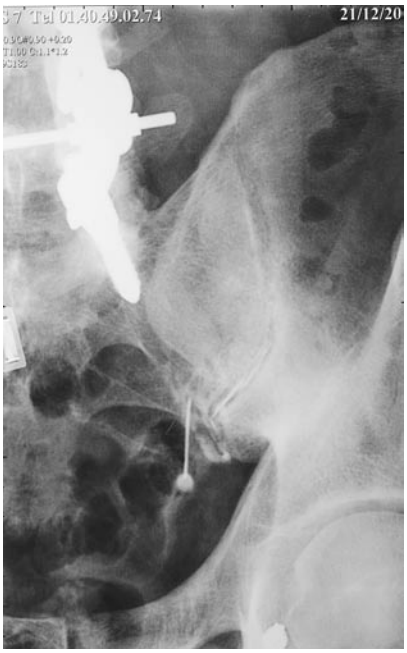

Figure 14.3 Left sacroiliac block in a patient with lumbar fusion. Note a possible leakage through the first sacral foramen.

some time for the patient to provoke, needs to be taken into account. The anesthetic is only active for a maximum of 30 to 60 minutes, and some patients need more time to assess the intensity of their pain.

False Negatives

False-negatives are also possible, in particular if the injection was painful, because the patient can have difficulty in differentiating the customary pain from that of the procedure, or if the anesthetic does not spread properly throughout the joint, which can occur in 20% to 25% of cases. It can sometimes remain in the caudal area.

WHEN SHOULD A DIAGNOSTIC SIJ BLOCK BE PERFORMED

Anesthetic block of the SIJ is an invasive procedure where interpretation can be difficult. It should therefore only be performed when it will provide a benefit to the patient. This benefit consists in the cessation of additional explorations once the origin of the pain has been identified. It can also be a therapeutic benefit because, despite the absence of controlled, randomized studies, intra-articular infiltration of a steroid is presumed to be effective [5,12,13].

A typical indication is that of chronic unilateral lower back pain (sometimes bilateral for some authors [9]) distributed over the SIJ area with or without referral into the lower limb, whose persistence is not explained by the biopsychosocial model and where it is thought, based on clinical examination and on lumbar imaging, that it does originate in the lumbar spine [14]. Its functional repercussions can justify performing a block which is all the more likely to be positive if the topography of the pain is compatible with a sacroiliac source [15], if the pain is not influenced by repeated mobilization of the lumbar region in various directions (e.g., the subjects' LBP does not "centralize") [14], and if several of the maneuvers to provoke sacroiliac pain are simultaneously positive [14]. Imaging plays no part in this decision. Bone scintigraphy alone has shown specificity (89%) but not sensitivity (46%) [16].

Persistence of lower back pain after lumbar fusion may be explained by painful SIJs (Figure 14.3) [17]. A block has all the more chance of being positive if lumbar pain after fusion is different from that which gave rise to the fusion in the first place, that it occurred after a pain-free period of several weeks or months after the fusion surgery, and that the L5-S1 disc was included in the fusion [17].

ALTERNATIVES TO ANESTHETIC BLOCK OF SIJ

The intra-articular anesthetic block favors pain relief in the very short term, sometimes difficult to quantify, and blocks only the joint itself. Other diagnostic techniques have been suggested, either including an injection of a steroid, or blocking periarticular structures.

Intra-Articular Injection of Anesthetic and Steroid

Liliang et al. added triamcinolone acetonide, a long-acting steroid, to the anesthetic. Both were intra-articular

injections. Two thirds of their patients found relief within 6 weeks [13]. However, in theory, the combination of steroid with the anesthetic can dilute the second agent, reducing its anesthetic affect. Additionally, limited intra-articular volume of the joint will not accommodate additional volume introduced by combining steroid with the anesthetic. Thus, diagnostic SIJ blocks should be performed by injecting solely anesthetic without steroid to maintain the accuracy of the blockade.

Guided Periarticular Block

The periarticular injection of an anesthetic under fluoroscopic guidance was suggested by Murakami et al. [18]. The rationale was that some pain may come not from the joint in the strict sense (cartilage and synovial membrane) but from the dorsal sacroiliac ligaments. These authors selected patients having at least one maneuver provoking positive sacroiliac pain and performed this injection, which was positive in 96% of cases. They remark that the injection into the middle of the periarticular area was the most effective.

Our opinion is that the results of this type of block are difficult to interpret, given the absence of control over the spread of the anesthetic agent. Anatomical structures such as the sacral dorsal rami or the paraspinal and gluteus maximus muscles can be anesthetized. The pain relief which may appear after this anesthetic does not mean that the pain is related to a dysfunction of the SIJ or of its ligaments. Hence, the periarticular approach may suffer from inferior specificity.

Combined Intra- and Periarticular Block

The second alternative is to perform a combined block. Borowsky et al. compared the rate of positivity of intra-articular injections (anesthetic and steroid) to that of combined intra and periarticular injections. Assessment of patients 1 hour after the injection enabled the effect of the anesthetic to be isolated. The positive rate of response was respectively 42.5% and 62.5%, that is, a better rate for the combined blocks. The authors conclude that intra-articular diagnostic blocks underestimate the prevalence of pain in the sacroiliac region [19].

The same remarks as earlier can be made. Anesthesia in periarticular structures lacks specificity. A pain relieved by blocking a muscle or a nerve in the sacroiliac region can originate in a more distant structure, as for example the lumbosacral region. This is even more probable if one follows the hypothesis of these authors, that is, a pain not relieved by an intra-articular SIJB.

CONCLUSION

Despite its interest, anesthetic block of the SIJ remains an experimental tool. But it is still the gold standard for confirming pain originating in the SIJ.

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15 Therapeutic Intra-Articular Injections for Painful Sacroiliac Joints

Amish R. Patel

INTRODUCTION

The sacroiliac joint is generally accepted as a potential source of low back pain and/or buttock pain with or without lower extremity pain. Several studies have attempted to establish the prevalence of sacroiliac joint syndrome using history and physical examination [1–3] and recently using a diagnostic fluoroscopically guided intra-articular injection using single- [4] and double-block paradigms [5]. Multiple treatments for sacroiliac-related pain have been adopted by the varying disciplines caring for patients with low back pain. Therapeutic sacroiliac joint injections have been an appropriate therapeutic intervention for the treatment of sacroiliac joint syndrome. The purpose of this chapter is to discuss and perform an evidence-based analysis of therapeutic intra-articular sacroiliac injections including corticosteroids, viscosupplementation, and prolotherapy.

TECHNIQUE

Fluoroscopically guided intra-articular sacroiliac joint block is a technique that requires a much lower dose of medication to achieve a desired therapeutic effect, thereby minimizing any potential side effects.

The patient should rest in a prone position, and a true lateral image of the joint should be obtained (Figure 15.1). This occurs when the dorsal and ventral aspects of the sacroiliac joint are superimposed. The C-arm is then rotated 0° to 15° in the horizontal plane. Whether the gantry angle moves in a clockwise or counterclockwise direction is dependent upon the patient's anatomy. When the medial joint which represents the dorsal component begins to separate from the lateral joint line, beam rotation is halted (Figure 15.2). The required target point lies along the inferior, posterior aspect of the joint approximately 1 to 2 cm cephalad of its most inferior end (Figure 15.3). This should be the zone of maximum radiolucency in the target region. A puncture point on the skin is selected directly overlying the target. A 22-gauge or narrower, 3.5-inch needle is advanced until the perisoteum of the lateral edge of the sacrum is abutted. The needle is then withdrawn a few millimeters and advanced into the previously identified lucency. Once administration of contrast material demonstrates an arthrographic pattern (Figure 15.4; anteroposterior, lateral, and oblique arthrogram views), a maximum total volume of injection of 2.5 mL is recommended.

At times, a small amount of contrast medium may spread outside the joint space into or onto structures surrounding the sacroiliac joint including the piriformis muscle, the anterior sacral foramina, the posterior sacral foramina, the lumbosacral plexus via a ventral capsular tear, or into the L5 epiradicular sheath. Initially, an anteroposterior view is recommended to show any potential escape of contrast from the superior and inferior ends of the joint. Next an oblique view is necessary to visualize the auricular shape of the joint margins. This view best visualizes ventral capsular tears. Next a lateral view is necessary to provide information about posterior transligamentous escape of contrast medium and ventral tears.

EFFICACY OF INTRA-ARTICULAR STEROID INJECTIONS

In a retrospective chart review, Slipman et al. [6] assessed improvement after intra-articular sacroiliac joint injection of steroid and physical therapy to treat patients experiencing symptoms of sacroiliac joint syndrome diagnosed by a single diagnostic injection with a minimum of an 80% decrease in the pre- and post-sacroiliac joint block visual analog scale (VAS) scores. Patients' symptoms duration before diagnostic injection ranged from a minimum of 1.5 months to a maximum of 84 months (average 20.6 months). Patients received an average of 2.1 injections (1–4 injections). At a mean follow-up of 94.4 weeks (10–160 weeks), VAS scores were reduced by 43% in 31 patients (12 men and 19 women). At follow-up, there was a statistically and clinically significant improvement in Oswestry disability scores (and VAS pain scores). However, there is a key limitation to this study. The study is retrospective with no control group for comparison. Without a control group, the effects of natural history on the observed outcomes are not clear.

In a prospective double-blind randomized control trial, Maugers et al. [7] randomized 13 patients with spondyloarthropathy and low back pain experiencing symptoms of sacroiliac joint pain syndrome based on history and physical examination to a fluoroscopically guided

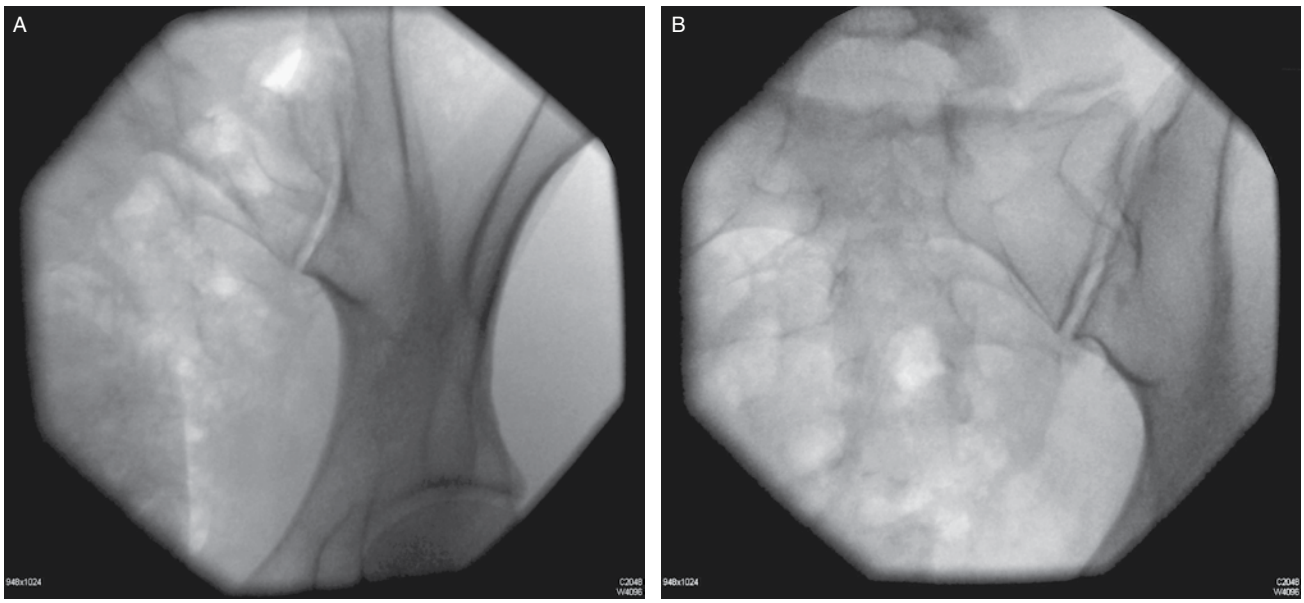

Figure 15.1 Lateral image.

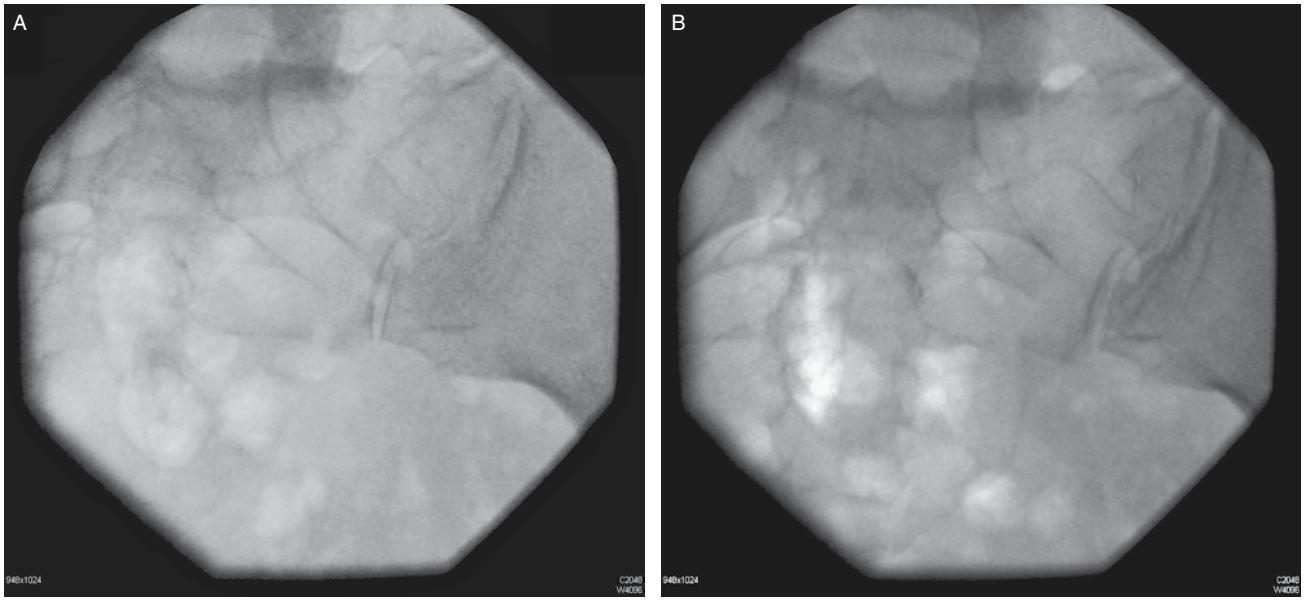

Figure 15.2 The medial joint line here represents the dorsal component.

sacroiliac joint corticosteroid versus saline injection. No diagnostic injections were performed. At 1 month, there was a clinically significant improvement in the corticosteroid group (5/6 vs. 0/7) with very good or good improvement. Dolorimetry, an instrument used to measure pain intensity, at 1 month demonstrated a statistically significant improvement from 6.8 to 1.3 corticosteroid group versus 7.0 to 5.2 placebo group, $P < 0.005$. Although this study further supports Slipman's findings, these findings may not apply to patients experiencing symptoms of sacroiliac joint syndrome without spondyloarthropathy.

In a prospective study, Liliang et al. [8] used intra-articular sacroiliac joint injection of steroid in 39 patients experiencing symptoms of sacroiliac joint syndrome without spondyloarthropathy diagnosed by a dual diagnostic injection paradigm defined at least a 75% pain reduction for 1 to 8 hours on both blocks. The solution consisted of 1 mL 0.5% bupivacaine or 2% lidocaine, mixed with 1 mL (40

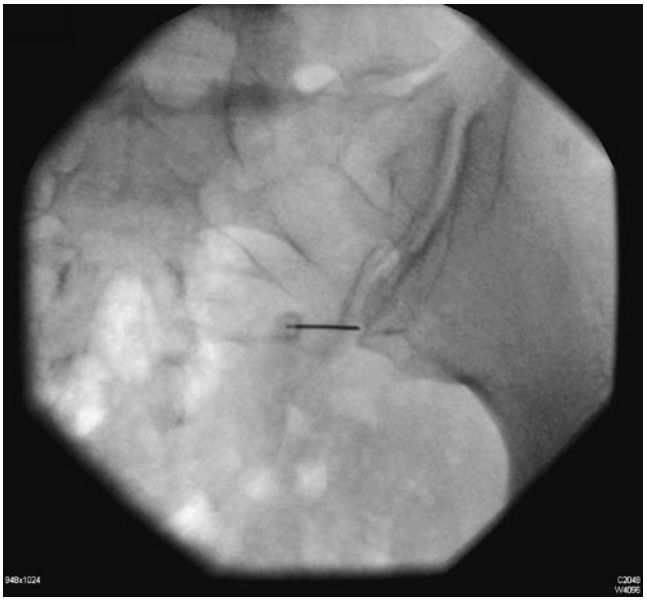

Figure 15.3 Needle positioned into the medial joint line.

mg) triamcinolone acetonide. All 39 patients included in the study had two separate sacroiliac joint injections prior to assessment. Of the 39 patients who underwent dual sacroiliac joint blocks with triamcinolone acetonide, 26 (66.7%) experienced more than 50% pain reduction for more than 6 weeks, which presented a successful response in these patients. The overall mean duration of pain reduction in the 26 responders to the second sacroiliac joint block was 36.8 ± 9.9 weeks (range, 12–60 weeks). Of these 26 responders, 8 showed recurrence of pain and received the third block with triamcinolone acetonide. Thirteen patients responded to sacroiliac joint blocks for a short time, with a mean duration of pain reduction 4.4 ± 1.8 weeks (range, 1–6 weeks). Of the nonresponders, seven had a history of a lumbosacral fusion and/or bilateral symptoms.

EFFICACY OF INTRA-ARTICULAR VISCOSUPPLEMENTATION

The sacroiliac joint is a synovial joint described as an auricular-shaped diarthrodial joint with joint capsule, synovial fluid, and hyaline cartilage on the sacral side and fibrocartilage on the iliac side [9,10]. Therefore, it is susceptible to degeneration like other synovial joints. The fundamental factor in the pathogenesis of osteoarthritis is the loss of viscoelastic properties of the synovial lining during the course of the disease [11,12]. Diminished viscoelasticity, largely its hyaluronan content, makes cartilage susceptible to mechanical damage. This has led to viscosupplementation, where a highly elastoviscous solution is injected into the sacroiliac joint, a potential treatment to restore rheological homeostasis. This provides a significant degree of analgesia.

Srejic et al. [13] reported four cases of sacroiliac joint syndrome (three bilateral and one unilateral) injected with 1 mL (8 mg) of Hylan GF 20 (Synvisc) into the sacroiliac joint diagnosed by two separate diagnostic sacroiliac joint injections using fluoroscopic guidance with a minimum of a 70% decrease in the pre- and post-sacroiliac joint block VAS scores. Hylan injection was repeated three times at 2-week intervals. VAS scores were reduced from 40% to 67% 12 to 16 weeks after the third injection. Since its introduction, Hylan has been used extensively in the treatment of osteoarthritis of the knee. However, its usage remains controversial as a long-term solution to patients with sacroiliac joint pain syndrome.

EFFICACY OF PROLOTHERAPY

Prolotherapy has been defined as "the rehabilitation of an incompetent structure (as a ligament or tendon) by the proliferation of new cells" [14]. The goal of this therapy is to produce dense fibrous tissue to strengthen the attachment of ligaments, tendons, joint capsules, and other fascial structures at their fibro-osseous junctions [15]. Notion of sacroiliac joint mobility and its potential implication for joint pathology has been debated for some time [16,17]. Pain may arise from the deep interosseous ligament which is exquisitely innervated. It has been suggested that when specific exercise programs fail, deficient ligament strength that stabilizes the posterior sacroiliac joint allows for abnormal muscle recruiting strategy [18]. In prospective study, Cusi et al. [19] examined whether prolotherapy injections into the dorsal interosseus ligament of the sacroiliac joint can assist patients with a clinical diagnosis of deficient stability of the sacroiliac joint that fails to respond to specific exercise therapy. Twenty-five patients unresponsive to an exercise program entered the study and underwent three injections of prolotherapy solution 6 weeks apart. The time between injections was based on an assumption that inflammatory reaction and formation of collagen takes up 7 to 8 weeks. The prolotherapy solution was prepared by drawing into a 5-mL syringe 0.8 mL of 50% glucose solution, 2.3 mL of 1% bupivacaine, and 0.8 mL of Isovue (iopamidol), and 0.8 mL was injected into the ligament using computed tomography guidance. Each patient continued there exercise program under the direction of the physiotherapist.

Entry criteria included the diagnosis of persistent suboptimal stability of the sacroiliac joint following a 3-month exercise program. This diagnosis had to be made independently by a sports and exercise medicine physician along with a physiotherapist involved in the study. Clinical history included localized and/or radiating low back pain in the vicinity of posterior superior iliac spine, worse on loading positions such as standing, sitting, or walking for at least 6 months prior to initial assessment. Patients with acute radiculopathy, infection, pregnancy, inflammatory conditions of the sacroiliac joint, and malignancy were excluded from the study. The clinical tests used to assess suboptimal stability of the sacroiliac joint were the sacroiliac joint glide test (anteroposterior and vertical arm with and without self bracing) [20], posterior pelvic pain provocation test [21], active straight leg raise with and without self bracing [22,23], and external manual compression and Gillett test [24].

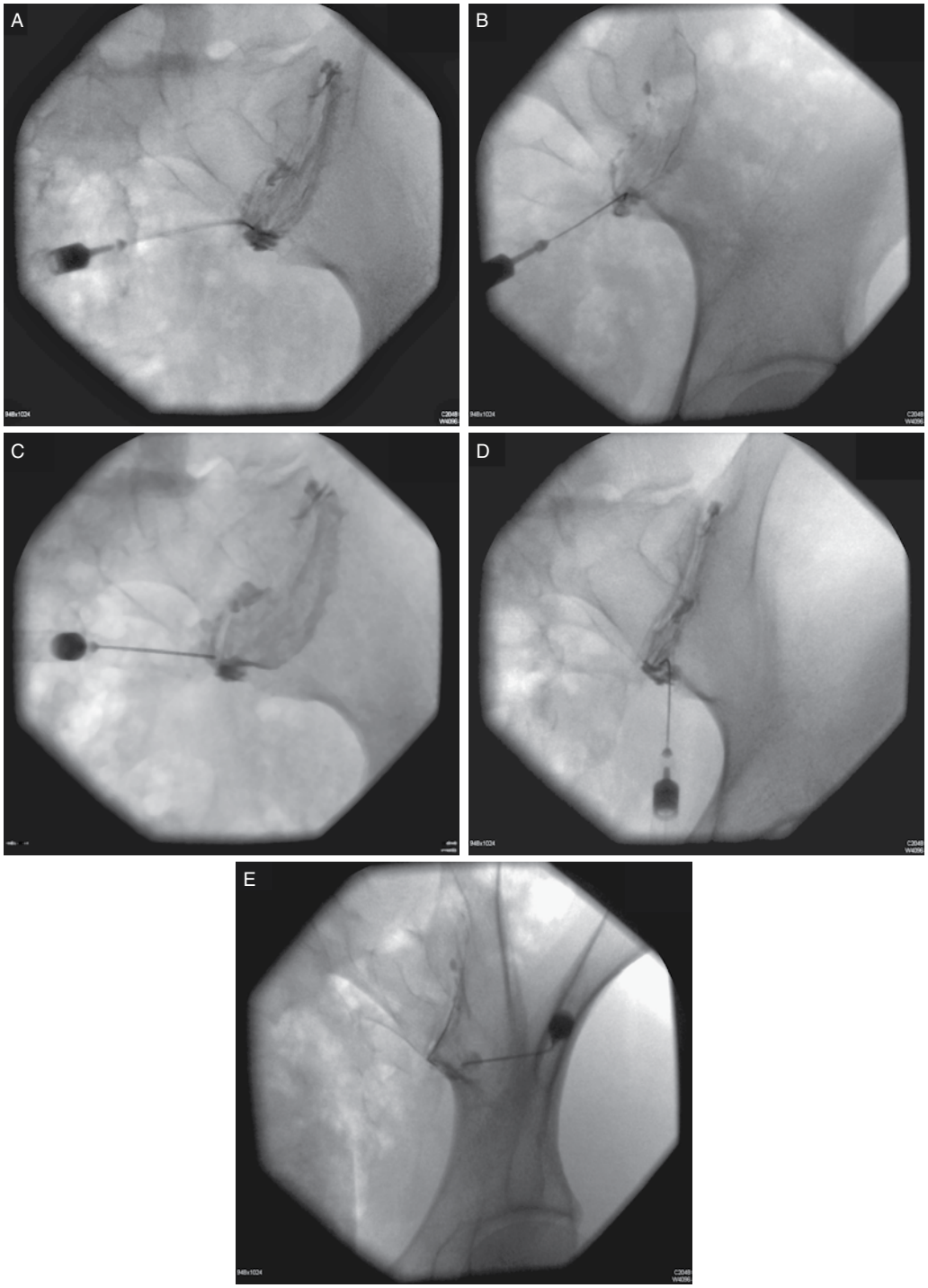

Figure 15.4 Arthogram images. **(A)** and **(B)** Anterior-posterior, **(C)** Oblique, **(D)** and **(E)** Lateral.

They were assessed 24 hours before each injection and 1 week after each injection by the both the sport and exercise medicine physician and physiotherapist. At a mean follow-up of 26 months (6–39 months), 19 patients demonstrated statistical significant reductions of the Quebec Back Pain Disability Scale and Roland Morris 24 Multi-Form Questionnaire at 3 months.

The weakness of this study was the absence of a diagnostic joint injection paradigm not implemented in the diagnosis of sacroiliac joint syndrome and the absence of a nonintervention control group to account for the effects of natural history on the observed outcomes. Prior to this study, the results of the effectiveness of prolotherapy are inconclusive. The diagnosis is generally nonspecific low

back pain with variability seen from author to author in injection technique, substances, volumes, and sites injected. This is the first study that used an injection technique to infiltrate specifically the ligamentous structures of the sacroiliac joint. The results of this trial warrant further research in this area.

CONCLUSION

In well-selected patients with sacroiliac joint pain syndrome, it appears that a significant degree of analgesia can be achieved with an intra-articular injection of steroid. Viscosupplementation and prolotherapy have not been as extensively studied. Long-term prospective, controlled, or comparative studies are currently lacking or are under investigation. Larger well-designed clinical trials are necessary to reach adequate conclusions regarding long-term effectiveness and to confirm or refute preliminary findings.

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16 Neuroablative Techniques for Sacroiliac Joint Pain

Anita Gupta and Steven P. Cohen

ANATOMY AND FUNCTION OF THE SACROILIAC JOINT

The largest axial joint in the body is the sacroiliac (SI) joint. The average surface area is approximately 17.5 cm^2 [1], with variability in size, shape, and surface contour among individuals [2,3]. A complex ligamentous network supports the synovial joint anteriorly and posteriorly [4]. The primary function of this ligamentous system is to bolster stability while allowing for adequate range of motion in multiple planes of movement. The SI joint is further supported by a network of muscles that generate stabilizing forces across the pelvic bones. These muscles include the gluteus maximus, piriformis, and biceps femoris. Their connection to the SI joint ligaments enables effective joint mobility. In 30% of SI joints, there exists a potential for shearing which contributes to the acute angulation of the short horizontal articulating component [5].

The SI joint is perhaps best conceptualized as a complex, with both intra- and extra-articular elements. Injury to either of these components can produce the clinical picture of painful SI joint dysfunction. Animal and human cadaveric studies have demonstrated nociceptors both within the joint capsule and in the surrounding ligaments [6,7]. In addition, clinical studies performed in asymptomatic volunteers and low back pain patients have documented pain provocation with both capsular distension and ligamentous stimulation [8–11].

Understanding the innervation of the SI joint is essential when contemplating denervation procedures. The lateral branches of the S1-S3 dorsal rami comprise the primary innervation to the posterior SI joint in humans, with contribution from the L5 dorsal ramus in most individuals [12–16]. In a cadaveric study, McGrath reported afferent input from S4 to the long posterior SI ligament in more than 50% of SI joints [17]. Although some literature refers to contributions from L4, the obturator nerve, and the superior gluteal nerve, the sources for these references are older and ambiguous.

The posterior lateral branch nerves are inconsistent in their anatomic locations, varying in number and location from patient to patient, side to side, and level to level. The nerves also run their anatomic courses at different depths, with some situated on bone and others embedded in soft tissue. These wide and unpredictable anatomical variations have significant implications when contemplating denervation treatments, because small single plane lesions are unlikely to interrupt all afferent nociceptive information [13,18].

The innervation of the ventral aspect of the SI joint complex has similarly not been well illuminated. However, because the nerve supply is not amenable to denervation, the ambiguity is less relevant clinically. The ventral rami of L4-S2 are most frequently cited as the main innervation to the ventral aspect of the joint [15,19], though some sources report contributions from as cephalad as L2 [1] (Figures 16.1 and 16.2) [12].

SI JOINT PAIN

Prevalence

Based on prevalence studies, SI joint pain accounts for between 15% and 25% of chronic axial low back pain cases [12,20,21]. The diagnosis and treatment of SI joint pain remains a challenge, with often conflicting evidence and complicated treatment algorithms. Most experts maintain low-volume intra-articular anesthetic injections to be the only reliable diagnostic modality [12,20,22].

Diagnosis and Clinical Presentation

Pain generated in the SI joint or surrounding structures can present as low back pain, leg pain, sacral pain, pelvic pain, or gluteal pain. Patterns of somatically referred SI joint pain have been identified and can vary significantly [23]. Numbness, popping, clicking, or groin pain [24] can occur [25]. Unilateral pain is more common than bilateral by a ratio as high as 4:1 [1].

One of the most challenging aspects of treating SI joint pain is the complexity of diagnosis. A variety of physical examination maneuvers have been advocated as diagnostic aids in patients with presumed SI joint pain [26]. Many involve distraction of the SI joints, with two of the most common ones being Patrick's test and Gaenslen's test. Despite the plethora of diagnostic tests, clinical studies have for the most part demonstrated that neither medical history nor any single physical examination finding is consistently capable of identifying a

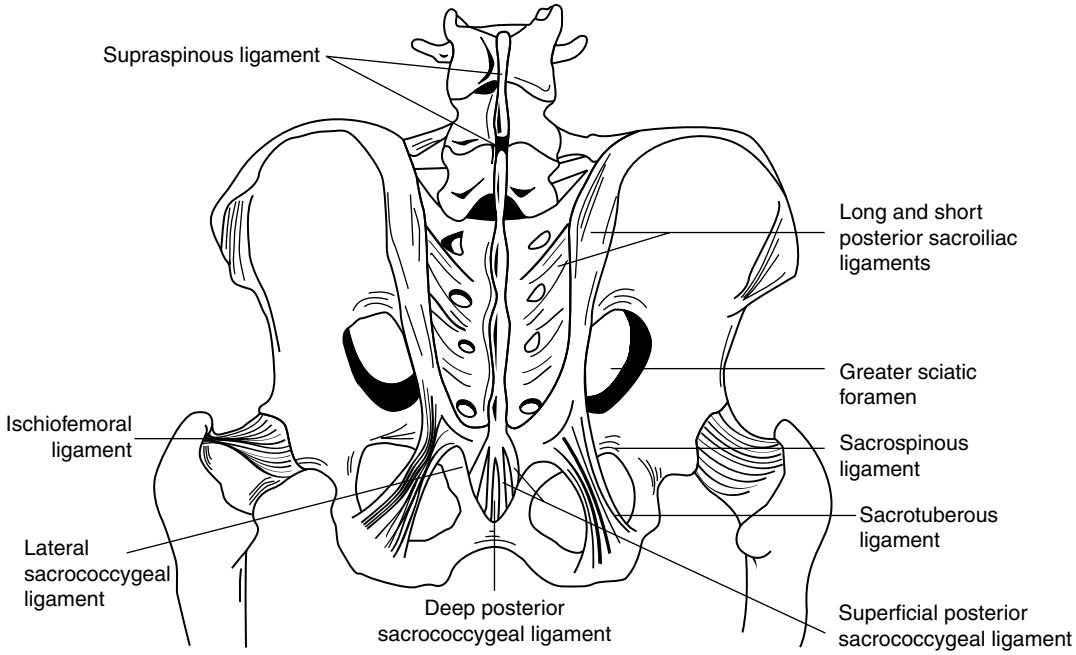

Figure 16.1 Posterior view of the articulations and associated ligaments of the sacroiliac joint and surrounding structures. Drawing by Jee Hyun Kim. [12]

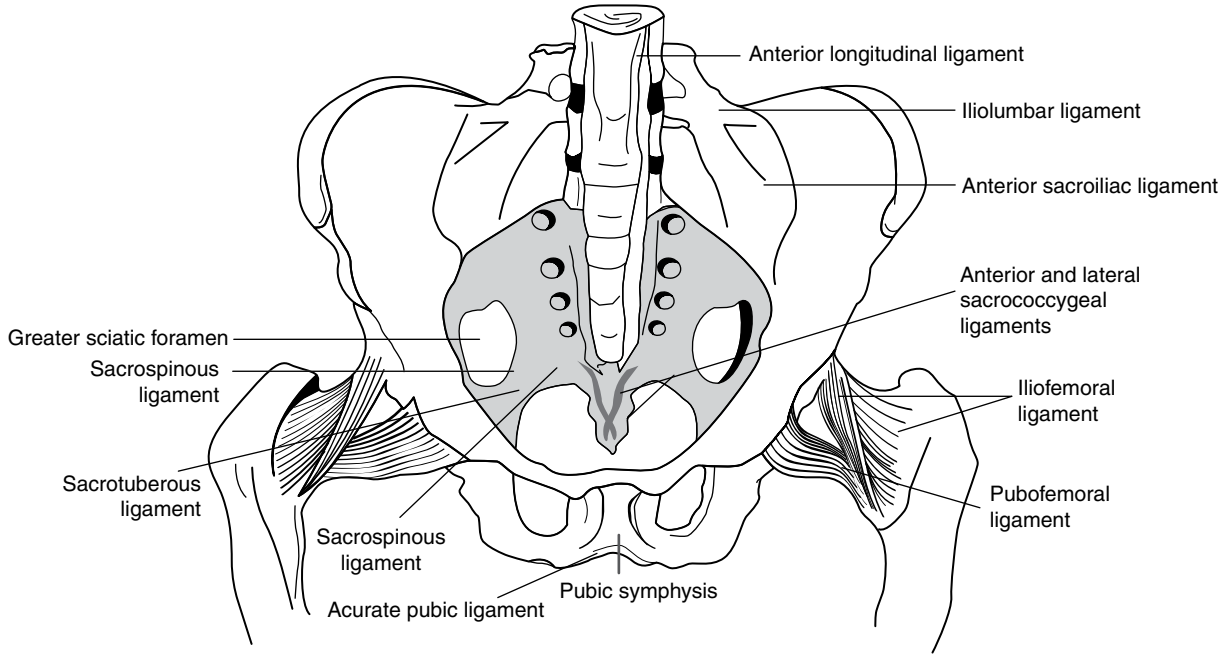

Figure 16.2 Anterior view of the articulations and associated ligaments of the sacroiliac joint and surrounding structures. Drawing by Jee Hyun Kim. [12]

dysfunctional SI joint(s) as the pain generator [11,24,27]. However, most [7,28], but not all [29], systematic reviews have found that batteries [30] of provocative maneuvers may distinguish SI joint from other sources of chronic low back pain.

In addition, Dreyfuss et al. [20] found that 20% of asymptomatic adults had positive findings on three commonly performed SI joint provocation tests. The reliability of provocative SI joint maneuvers and alignment/mobility tests has also been questioned. Whereas some of these studies have found moderate to high interexaminer reliability [30–32], most have not [33–37]. In general, greater reproducibility is found in provocative tests than mobility and alignment assessments.

Results of studies examining radiologic findings in patients with SI joint pain have been similarly disappointing. In studies by Maigne et al. [38] and Slipman et al. [39], the investigators found sensitivities of 46% and 13%, respectively, for the use of radionuclide bone scanning in the identification of SI joint pain. Despite the high specificities in these studies (89.5% for Maigne et al. and 100% for Slipman et al.), the low sensitivities indicate that bone scanning is a poor screening test for SI joint pain. A retrospective analysis by Elgafy et al. found computed tomography (CT) imaging to be 57.5% sensitive and 69% specific in diagnosing a painful SI joint [40].

There have been several attempts to identify pain referral patterns from SI joints. In one of the earliest studies conducted in 10 asymptomatic volunteers, Fortin et al. [9] performed provocative SI joint injections using contrast and lidocaine. Sensory changes were localized to the ipsilateral medial buttock inferior to the posterior superior iliac spine in 6 of the 10 subjects. In two subjects, the area of hyperesthesia extended to the superior aspect of the greater trochanter. The last two subjects experienced sensory changes referring into the upper thigh. In a follow-up study, independent examiners selected 16 individuals among 54 with chronic low back pain whose pain diagrams most closely resembled the pain referral patterns obtained in the first study [8]. These 16 patients proceeded to undergo provocative SI joint injections with contrast and local anesthetic. All 16 experienced concordant pain during the injection, with 14 obtaining pain relief after deposition of local anesthetic. Slipman et al. [41] conducted a retrospective study to determine the pain referral patterns in 50 patients with injection-confirmed SI joint pain. In contrast to the findings by Fortin et al. [9] and Schwarzer et al. [24], Slipman found the most common referral patterns for SI joint pain to be radiation into the buttock (94%), lower lumbar region (72%), lower extremity (50%), groin area (14%), upper lumbar region (6%), and abdomen (2%). Twenty-eight percent of patients experienced pain radiating below their knee, with 12% reporting foot pain. Based on the existing data, the most consistent factor for identifying patients with SI joint pain is unilateral pain (unless both joints are affected) localized predominantly below the L5 spinous process [8,9,11,24,41].

Predisposing Risk Factors

There are multiple predisposing risk factors for individuals to develop painful SI joint dysfunction. Active athletes participating in sports that require unilateral loading such as kicking and throwing are at increased risk [42]. SI joint dysfunction is also commonly found in cross-country skiers and rowers [43]. Patients with SI joint pain are more likely to report a history of trauma (40%–58%) [24,44–46] than those with facetogenic, myofascial, or discogenic pain, with the three most common inciting events being motor vehicle accidents, falls, and cumulative stressors. SI joint pain is more common in pregnant women (possibly as a result of the release of the hormone relaxin, which allows pelvic expansion and increased motion) [42,47]. Other factors, such as the trauma of childbirth, altered posture, increased lordosis, and weight gain, may also predispose parturients to painful SI joint dysfunction.

Asymmetric SI joint laxity measured during pregnancy is predictive of the persistence of moderate-to-severe pregnancy-related pelvic pain extending into the postpartum period [48]. Bleeding into the joint during delivery may predispose one to sacroilitis, which can be seen on CT or bone scintigraphy [49]. Rotation of the hemipelvis in the sagittal plane can produce pelvic torsion (also called pelvic asymmetry). This asymmetry can develop from either anterior or posterior rotation of one innominate bone in relation to the sacrum and the opposite innominate. The association between static pelvic asymmetry and low back pain, however, remains uncertain. Objectively determined asymmetry, when measured in the standing position, may be a risk factor for low back pain and subsequently SI joint pain [25].

RADIOFREQUENCY TREATMENTS

In randomized studies evaluating periarticular and intra-articular corticosteroid injections in patients suspected of having SI joint pain, the results are divided regarding affording any long-term benefit [50–53]. Studies evaluating conservative therapies are flawed by the lack of adequate control subjects and inappropriate diagnostic workups [12]. History of lumbosacral fusion appears to be a risk factor for poor outcomes after intra-articular SI joint steroid injection [54] making SI joint neurotomy a more attractive treatment option of these patients.

Radiofrequency (RF) denervation has emerged as a promising treatment alternative for refractory cases of SI joint pain [55,56]. Because lateral branch RF denervation was first described in the early 2000s, [13,55] numerous uncontrolled [57–60] and controlled [56] studies have since been published on this procedure, which have universally reported positive results. However, these studies are characterized by wide variations in technique, selection criteria, and standards of success.

Patient Selection

Patient selection is critical for any interventional spine procedure, [18,61–65] especially in the application of new innovations as negative results may threaten to undermine the very concept behind treatment. In view of the wide variability in pain referral zones, the ambiguity of innervation, and the overriding controversy surrounding RF denervation in general, one might reasonably argue that proper patient selection criteria are even more critical for SI joint denervation [24,41,66].

There are considerable differences in the reported pain referral patterns from the SI joint. In uncontrolled studies evaluating SI joint denervation, investigators have utilized disparate referral maps in their selection criteria [55,60,67]. Because lateral branch denervation does not interrupt the afferent input from the entire SI joint, and different aspects of the joint most likely possess different referral zones, identifying those referral patterns most amenable to RF lesioning might save some patients from an unnecessary procedure. In view of this dilemma, some

Table 16.1 Summary of Clinical Studies Evaluating Radiofrequency Procedures in the Treatment of Sacroiliac Joint Pain

Author, Year	Study Design	Number of Patients	Treatment	Primary Outcomes	Comments
Cohen et al., 2009 [46]	Retrospective	77	Multiple, 80°C, 90-second lesions of the L4 and L5 dorsal rami and S1–3 lateral branches	Forty patients (52%) obtained a positive outcome. Among the entire study cohort, both NRS and ODI scores declined an average of 40%	Patients included had ≥50% pain relief after at least 1 low-volume (<2 mL) local anesthetic intra-articular SI joint block. Age > 65, high pre-procedure pain score, opioid use and pain below knee were associated with (–) outcome. Cooled RF weakly associated with (+) outcome
Cohen et al., 2008 [56]	Randomized, placebo-controlled	28	Single conventional lesions at L4 and L5. 3 cooled-probe lesions at S1 and S2 and two at S3 and s ometimes S4	In the treatment group, pain scores were reduced by 60, 60, and 57% at 1, 3, and 6 months, respectively	Pain relief of 75% or more calculated from a pain diary after a single SI joint injection required for inclusion
Kapural et al., 2008 [68]	Retrospective, case series	26	One cooled lesion at L5 and 2 to 3 cooled lesions at S1–3	3–4 months after treatment, half the patients had achieved the primary outcome of ≥50% reduction in VAS pain scores	Patients included in study had two diagnostic SI joint blocks with ≥50% of pain relief
Burnham and Yasui, 2007 [58]	Prospective	9	Three conventional lesions at L5 and 3 bipolar strip lesions at S1–3	The percentages of those who indicated that they were very satisfied at 1, 3, 6, 9, and 12 months post-procedure were 78%, 67%, 67%, 89%, and 67%, respectively	Pts included had ≥ 50% relief of index pain on at least one SI joint block and one prognostic lateral branch block
Vallejo et al., 2006 [59]	Prospective	22	Multiple, 39°C to 42°C, 120-second pulsed RF lesions of the L4 and L5 medial branches and S1–2 lateral branches	73% of patients (16 patients) experienced >50% reduction in pain. Duration of pain relief range was 6–9 weeks in four patients, 10–16 weeks in five patients, and 17–32 weeks in seven patients	Confirmation of SI joint pain required 75% or greater pain relief following > 2 SI joint injections
Buijs et al., 2004 [57]	Prospective observational	38 patients, 43 joints	80°C 60-second lesions of the S1–3 dorsal rami in all patients and L4-L5 dorsal rami in about half the patients	At 12-week follow-up, 34.9% of procedures (26.3% of patients) resulted in complete pain relief and another 32.6% (34.2% of patients) reported _50% pain relief	Inclusion criteria included ≥ 50% pain relief with SI joint blocks. Outcomes of patients receiving additional L4–5 dorsal rami lesions no different than those undergoing only S1–3 denervation
Yin et al., 2003 [13]	Retrospective	14 patients, including four who underwent previous spine surgery	80°C, 60-second lesions of the L5 dorsal ramus sensory branch and S1-S3 dorsal rami lateral branches depending on stimulation results. All patients had L5 and S1 branches lesioned. 11 patients had a lateral branch at S2 and 6 at S3 that were lesioned	64% of patients obtained >50% consistent pain relief at 6 months, with 36% obtaining complete relief. 5 patients reported <50% pain relief, and 2 reported no relief whatsoever	Inclusion criteria was >70% pain relief after two separate SI joint deep interosseous ligament injections.
Gevargez et al., 2002 [67]	Prospective observational	38 patients, including 13 who underwent bilateral treatment	Three 90°C, 90-second lesions in the posterior interosseous SI ligaments and one lesion of the L5 dorsal ramus	3 months after treatment, 34.2% were pain-free, 31.6% reported a substantial decrease in pain, 18.4% obtained a slight decrease in pain and 7.9% reported no pain reduction	Did not specify % pain relief required during diagnostic SI joint injections for inclusion
Cohen and Abdi, 2003 [55]	Retrospective	18 patients	80°C, 90-second lesions of the L4 and L5 dorsal rami and S1–3 lateral branches.	13 of 18 patients with SI joint pain obtained 50% pain relief with L4 and L5 dorsal rami and S1–3 lateral branch blocks, with two deriving long-term relief. Eight of nine patients who underwent RF denervation obtained >50% pain relief 9 months post-procedure	Inclusion criteria was >50% pain relief with SI joint blocks. In six patients, empirical lesions were made at the S3 lateral branch because of failure to obtain concordant stimulation
Ferrante et al., 2001 [60]	Retrospective	33 patients, 50 joints	Multiple, 90°C, 90-second lesions made at <1-cm intervals as high in the posteroinferior joint as possible	36.4% of patients obtained >50% pain relief 6 months postprocedure. Average duration of pain relief was 12.0 ± 1.2 months	Did not specify % pain relief required during diagnostic SI joint injections for inclusion. Only posteroinferior joint denervated

Abbreviation: RF, radiofrequency

investigators have used "prognostic" lateral branch blocks done with local anesthetic to screen RF candidates [55,58], whereas others have used confirmatory SI joint injections [13,59,68] because of the high false-positive rate associated with uncontrolled blocks [30,69–71]. Yet most studies have not used any confirmatory or prognostic procedure before proceeding to definitive treatment [46,57,60,67].

Cohen et al. [46] recently described outcome predictors for SI joint denervation procedures, which currently is the sole study evaluating selection criterion. The results of this study demonstrated that although certain demographic and clinical variables may influence outcome, no single factor strongly and reliably predicted treatment results. This preliminary data did not support the routine use of more stringent selection criteria, such as multiple SI joint local anesthetic blocks, near-complete pain relief from diagnostic blocks, or prognostic left bundle-branch block [46].

Dreyfuss et al. [10] demonstrated that adequately performed lateral branch blocks effectively block ligamentous probing (extra-articular stimulation), but not capsular distention (intra-articular stimulation). This limits their use in patients with suspected intra-articular pathology, such as arthritis and spondyloarthropathy. However, there may be considerable overlap between intra- and extra-articular etiologies. In a randomized, placebo-controlled, double-blind study, Luukkainnen et al. [50] demonstrated efficacy for periarticular injections, which should theoretically block extra-articular pathology, in patients with spondyloarthropathy, which predominantly affects the joint cavity. Cadaveric studies have also shown that single-site and depth lateral branch injections are not effective in anesthetizing most lateral branch nerves as they converge into the foramen [10]. This finding may explain why the use of single-site lateral branch blocks in the Cohen study failed to improve RF outcomes [46].

Alternative Denervation Techniques

Intra-Articular Phenol Injection

There are currently no controlled studies demonstrating the benefit of intra-articular phenol. The conceptual appeal of intra-articular phenol is that unlike RF ablation which targets only dorsal SI joint pathology, the injectate may spread throughout the entire joint, encompassing both the cephalad and ventral portions [72]. In a small (n = 10) retrospective study, Ward et al. described the use of fluoroscopy-guided 6% phenol injections (2.5 mL) in patients with injection-confirmed SI joint pain. Twenty percent of patients experienced greater than 70% pain improvement, with the average duration lasting 24 weeks. Sixty percent obtained between 50% and 70% improvement. The obvious downside of this procedure is that spread to the sacral nerve roots is unpredictable and not uncommon with intra-articular injections [69]. This in turn can lead to serious neurological sequelae.

Cyroanalgesia

Cryoneuroablation, also known as cryoanalgesia or cryoneurolysis, is a specialized technique for providing intermediate-term pain relief in interventional pain management settings. The application of cold to tissues creates a conduction block, similar to the effect of local anesthetics. Intermediate-term pain relief from nerve freezing occurs because ice crystals create vascular damage to the vasonervorum, which produces severe endoneurial edema. The clinical applications of cryoanalgesia include a wide range of both neuropathic and nociceptive pain conditions. Cryoanalgesia for SI joint pain was reported by Trescot et al. [73] to be an alternative treatment to conventional RF ablation. The two main advantages of cryoanalgesia are that it is a non-neurolytic procedure and it creates a very large lesion size which may provide similar efficacy to cooled RF ablation. However, the former benefit is mitigated by the fact that conventional RF is not generally associated with worsening neuropathic pain when used to treat nociceptive conditions. The drawbacks include the shorter duration of benefit and higher risk of bleeding and nerve injury, which are functions of the large probes usually employed.

Pulsed RF

Pulsed RF is a novel technique in which a relatively high voltage is applied near neural tissue in short pulses, which avoids a significant rise in temperature to neurolytic thresholds (45ºC). Hence, pulsed RF ablation is essentially a non-neurolytic procedure. Because of the large electromagnetic field created, the affected target area may be greater in scope than that associated with conventional RF. However, there is limited evidence supporting the efficacy of this procedure, and the evidence that does exist suggests that the benefit may be shorter in duration than that obtained with conventional RF. In the only published study on pulsed RF for SI joint pain, Vallejo and colleagues reported the results of a prospective case series conducted in patients with intractable SI joint dysfunction who were treated with pulsed RF denervation of the lateral branches from L4-S2. One hundred twenty-six patients with presumptive SI joint dysfunction based on history and physical examination underwent fluoroscopically guided intra-articular SI joint injections. Among the 52 patients (41.3%) with a positive response, 22 failed to obtain long-term relief and proceeded to pulsed RF. Sixteen (73%) of these individuals experienced either "good" or "excellent" pain relief lasting at least 6 months. In positive responders, the mean duration of analgesia was 20 weeks. In addition to pain scores, quality of life scores also improved in all measured categories [59].

APPROACHES TO RF ABLATION AND TECHNIQUE

The principal purpose for RF denervation procedures is to provide prolonged pain relief compared with more conservative measures in patients suffering from injection-confirmed SI joint pain. For SI joint pain, the most common indication is significant but transient relief with diagnostic SI joint injections. The RF techniques used have ranged from ablating the nerves supplying

the SI joint [13,55,57], creating lesions in the joint itself [60], and using a combination of ligamentous and neural RF ablation [67]. Few previous studies have described RF lesion within the SI (or other) joint(s) itself. Among these, the results have been inconsistent and mostly disappointing [60,74].

In contrast, the success rates of studies targeting the nerve supply are higher than those focusing on the joint itself, with approximately two thirds of patients reporting significant pain relief. Following is a review of the various approaches to RF ablation including the following: intra-articular approach, extra-articular approach, cooled RF ablation, bipolar RF ablation, and a combination of ligamentous and neural RF ablation.

Intra-Articular Approach

In 2001, Ferrante et al. described the intra-articular approach for RF ablation for SI joint pain (Figure 16.3) [60]. The authors reported the results of a series of 50 SI joint RF denervations performed in 33 patients with SI joint pain. All patients underwent diagnostic SI joint injections with local anesthetic before denervation. Changes in visual analog pain scores (VAS), pain diagrams, physical examination (palpation tenderness over the joint, myofascial trigger points overlying the joint, SI joint pain provocation tests, and range of motion of the lumbar spine), and opioid use were assessed pre- and postdenervation. The criterion for a successful procedure was 50% or a greater decrease in visual analog scale for at least 6 months, and 36.4% of patients (12 of 33) met this criterion. This study demonstrated that intra-articular RF lesioning of the SI joint can significantly reduce pain in only a minority of patients with SI syndrome. The main disadvantage of this approach is that it only denervates the posterior inferior one third of the joint.

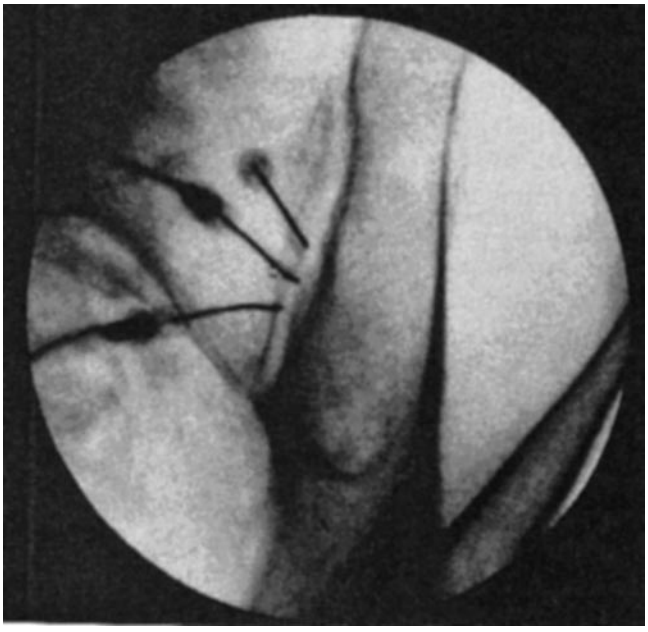

Figure 16.3 Leapfrog technique of SI joint RF denervation. [60]

Extra-Articular Approach (Lateral Branch Denervation)

As with all interventional techniques, lateral branch RF ablation must be performed under sterile conditions, with the patient positioned prone and a C-arm present to optimize visualization of the target sites. For lesioning of the L4 medial branch and L5 dorsal ramus (if targeted), the RF cannula with active tips are inserted parallel to the course of the nerve until bone is contacted just superior and medial to the junction between the superior border of the transverse and superior articular processes for procedures done at L4, and at the junction of the ala and articular process of the sacrum for L5 procedures, similar to previously published studies (Figure 16.4) [18,75]. Inserting the electrode parallel to the course of the nerve has been shown to increase lesion size, and hence minimize the chance of inadvertently missing the target the nerve [76].

Because it is not possible to discern electrostimulation between the various (e.g., medial, lateral, and intermediate) branches of the primary dorsal rami, the targeted nerve at this level is referred to as the parent branch. At each level, placement of the electrode in close proximity to the nerve is confirmed using electrostimulation at 50 Hz, with concordant sensation achieved at 0.5 V or less. Before lesioning, the absence of leg contractions is verified with stimulation at 2 Hz up to 2 V typically causing multifidi contractions only. After satisfactory electrode placement, 0.5 mL lidocaine, 2% mixed with corticosteroid, can be injected through each cannula to reduce procedure-related pain and the subsequent risk of neuritis [77]. By enhancing electrical conductivity, the preablation injection of local anesthetic may also increase lesion size. Once sufficient time has elapsed for the local anesthetic to take effect, the RF probe is reinserted, and a 90-second, 80°C lesion is made using a RF generator.

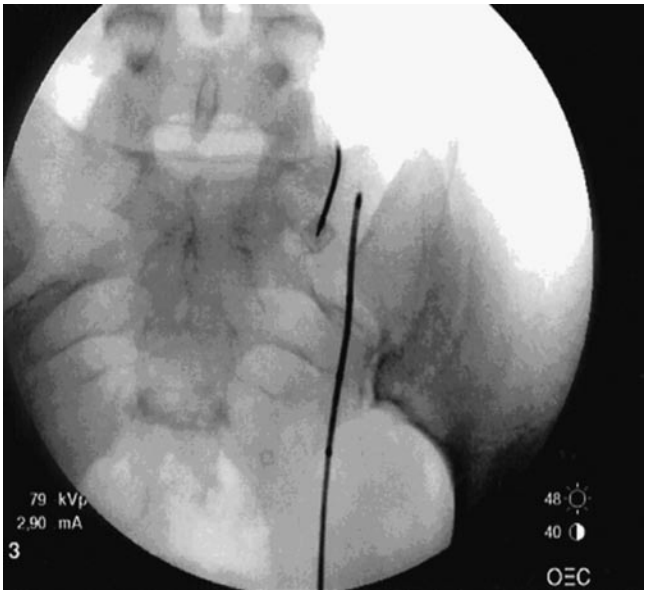

Figure 16.4 AP radiograph demonstrating single electrode placement for S1-3 lateral branch radiofrequency denervation. Also pictured is a separate electrode for L5 dorsal ramus lesioning.

For S1-S3 lateral branch procedures, conventional electrodes may be inserted between 3 and 5 mm from the lateral border of the foramina at predesignated positions (Figure 16.5).

Generally a pure anteroposterior view is used to optimize visualization of S2-S4, though occasionally the image intensifier must be angled cephalad to properly visualize the posterior opening. For S1, either slight cephalad or ipsilateral oblique angulation is often needed to discern the foramen. In certain patients, it may not be possible to definitely visualize all of the foramina, in which case inserting a 25-gauge needle into the obscured ones may be helpful to conceptualize the anatomy. In obese patients on high-dose opioids, a bowel prep can be used to maximize visualization [46].

For right-sided S1 and S2 procedures, the electrode target sites correspond approximately to the 1:00, 3:00, and 5:30 positions on the face of a clock; on the left, the target sites were at 7:00, 9:00, and 10:30 (Figures 16.5–16.7). At S3, needles are placed at 1:30 and 4:30 on the right side, and 7:30 and 10:30 on the left side. We currently target S4 only when the foramen is at a level parallel or cephalad to the inferior border of the SI joint.

It is our practice to perform sensory stimulation at each level only for the first needle placement, provided concordant sensation is elicited at 0.5 V or less. Before lesioning, 0.5 mL of 2% lidocaine is administered per spinal level. To ensure that anesthetic spread to adjacent foramina does not impede sensory testing, electrodes are generally placed and stimulated at contiguous levels before denervation is commenced. When all needles are properly positioned, monopolar electrodes are sequentially inserted into the cannulae, and 90-second lesions are created [13].

Cooled RF Ablation

Cooled-probe RF ablation is a new modality of treatment for painful SI joints. Using cooling-probe technology, the tissue temperature immediately adjacent to the cooled electrode is maintained at 60°C, while the target tissue is heated to 75°C, resulting in a lesion diameter ranging between 8 and 10 mm (Figures 16.8–16.10) [56]. The main advantages of cooled-tip probes are the larger heating distance (up to 3 cm from the active tip), and greater depth

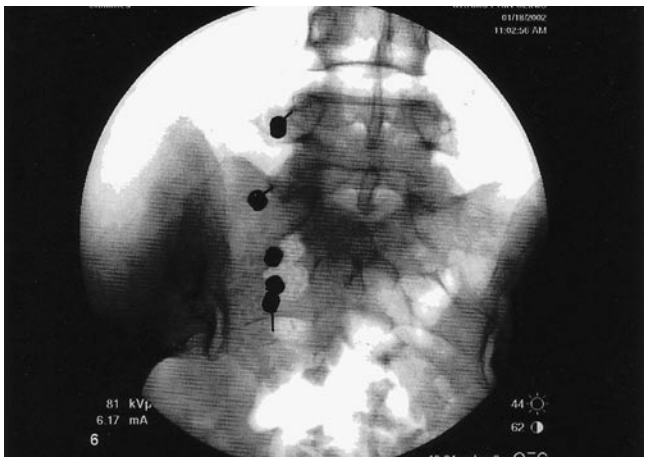

Figure 16.5 AP radiograph depicting electrode placement for L4 and L5 primary dorsal ramus, and S1-S3 lateral branch blocks.

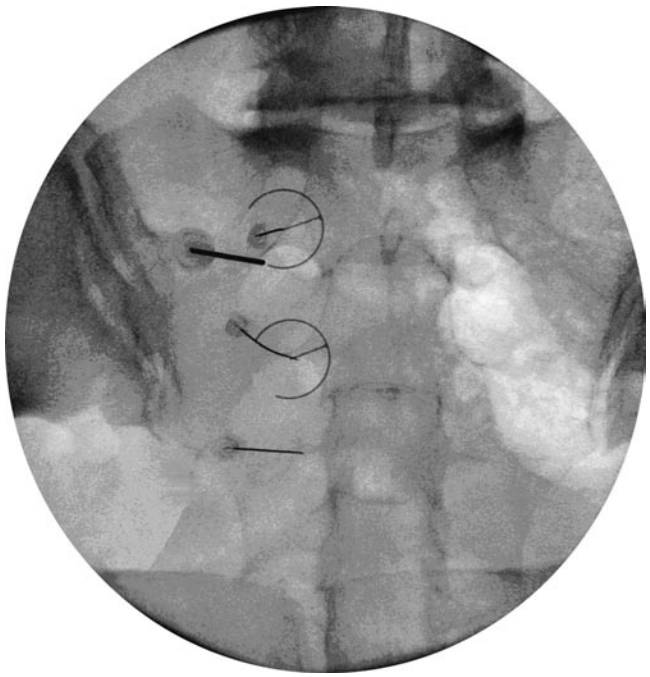

Figure 16.6 AP radiograph demonstrating S1 electrode placement at 7:00 on the face of a clock. The smaller needles delineate the location of the foramina.

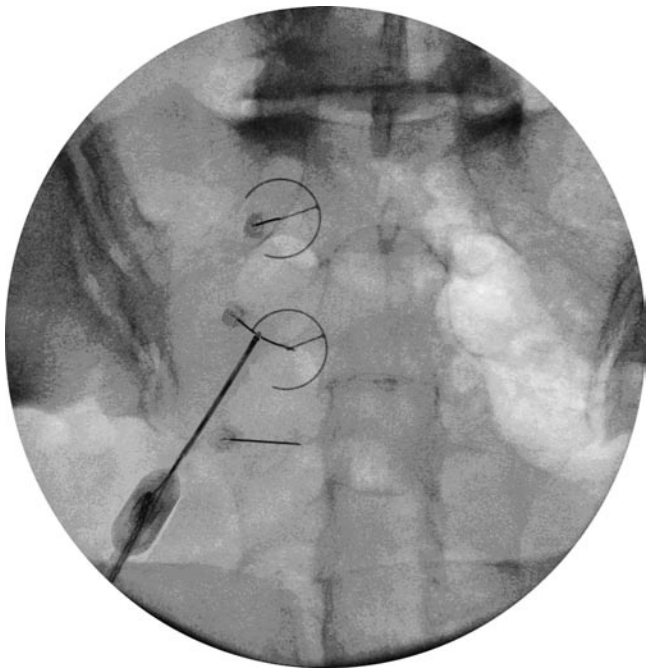

Figure 16.7 AP radiograph demonstrating S2 electrode placement at 10:00 on the face of a clock. The smaller needles delineate the location of the foramina.

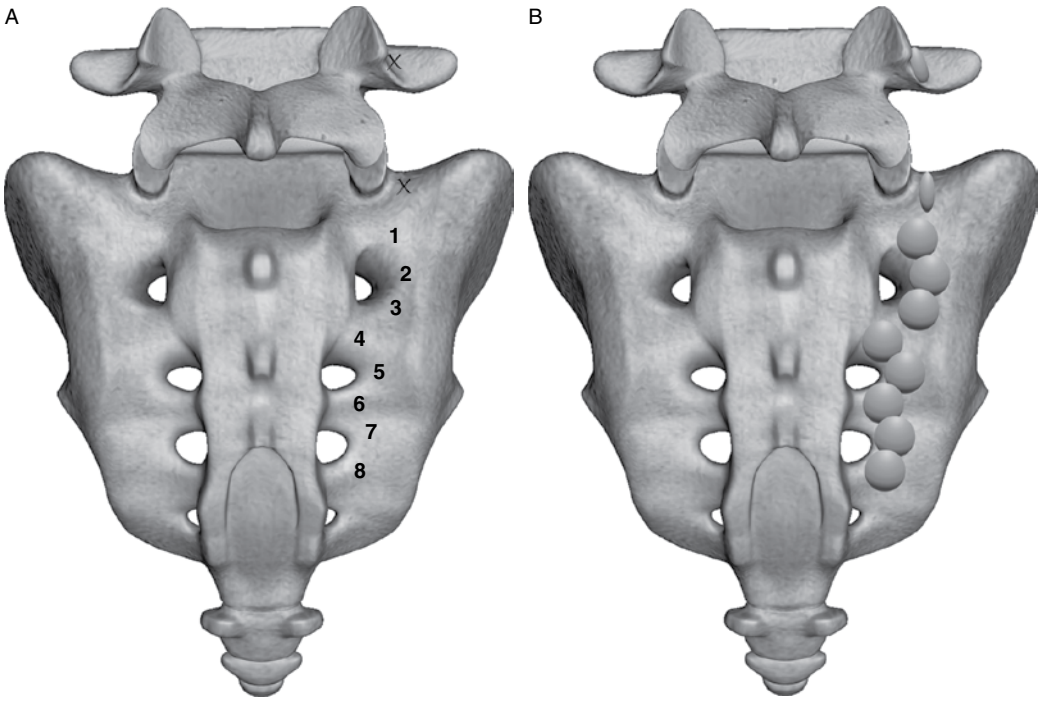

Figure 16.8 Schematic diagram illustrating: **(A)** Target points for right-sided conventional (L4 and L5) and cooled (S1-S3) radiofrequency denervation at the junction of the L5 superior articular and transverse processes (L4 primary dorsal ramus), the sacral ala (L5 primary dorsal ramus), and S1-S3 foramina (lateral branches). **(B)** Anticipated lesions at each of the target points. [56]

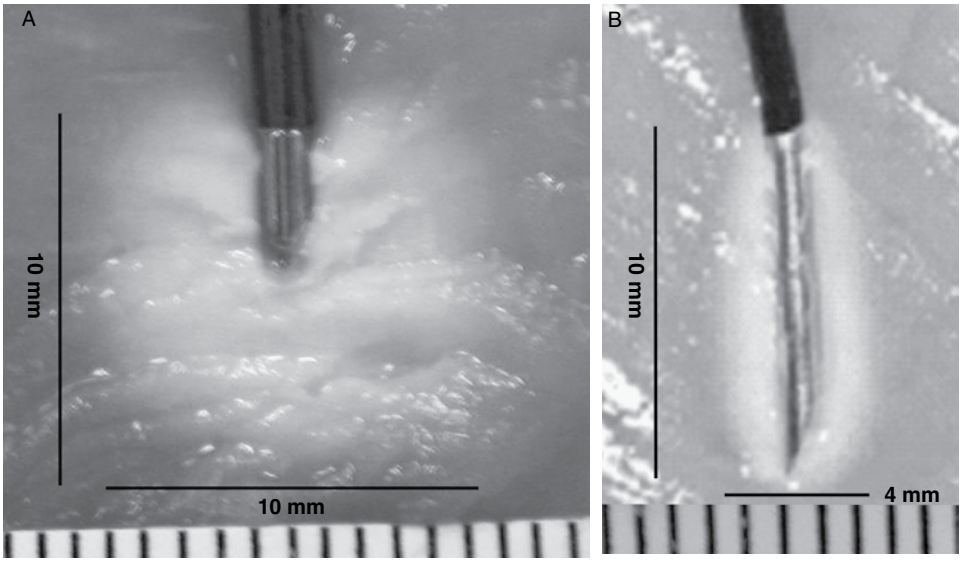

Figure 16.9 Adjacent photographs demonstrating the difference in lesion size between cooled **(A)** and conventional **(B)** radiofrequency probes in chicken meat. Each *small line* represents a distance of 1 mm. [56]

of lesioning, which should theoretically improve success rates. In contrast, conventional RF ablation creates lesions approximately 3 to 4 mm in diameter, which is less likely to interrupt all afferent nociceptive input from the SI joint. This is an important consideration in light of the individual anatomic variations in location and quantity of nerves. When using cooled-probe technology, inserting the electrodes at least 5 mm from the foramen is necessary to ensure that the temperature within the foramen does not exceed 45ºC. Because of the eightfold increase in lesion volume, most clinicians choose not to perform sensory stimulation provided the locations of the foramina are clearly demarcated. An additional advantage of cooled RF is that the needles are placed using a perpendicular rather than parallel trajectory, which is technically easier and traumatizes less collateral tissue.

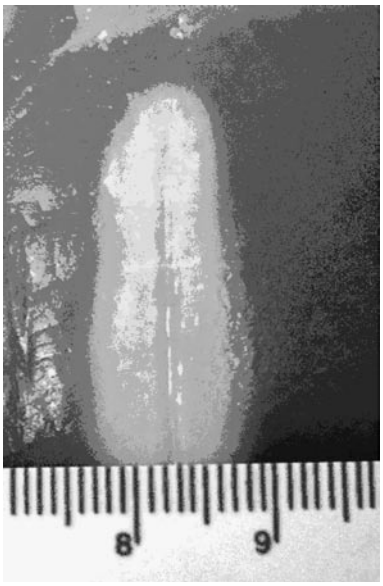

Figure 16.10 Depiction of lesion dimensions for single electrode sacral lateral branch radiofrequency technique. A 10-minute, 90°C lesion measures 13 mm in diameter and 52 mm in length.

In the only placebo-controlled study evaluating SI joint denervation, Cohen et al. [56] compared sham and cooled RF denervation of the L4-S3 lateral branches in 28 subjects with SI joint injection-confirmed pathology. For 6 months post-procedure, the treatment group obtained significant improvement in pain scores, functional capacity, and medication usage compared with the control group. Fifty-seven percent of patients in the RF group continued to report pain relief 6 months after treatment, compared with 0% in the placebo group. In those patients with a successful procedure, the median duration of relief was 7.9 months.

The main disadvantages of cooled RF are the greater electrode diameter, which may increase the risk of bleeding, and the longer lesioning time (2.5 minutes vs. 60–90 seconds for conventional RF). For safety reasons, the aggressive lesion size may increase the risk of motor nerve injury when targeting lumbar dorsal rami, and at present cannot be recommended at these levels (Figures 16.8–16.10) [56].

Bipolar RF Ablation

In 2007, Burnham and Yasui [58] performed a prospective, open-label study evaluating the effectiveness of bipolar RF ablation on pain, analgesic usage, and disability in nine patients with injection-confirmed chronic, mechanical SI joint pain. Subjects were treated with a series of bipolar RF strip lesions performed adjacent to the lateral dorsal foraminal aperture plus conventional monopolar lesioning at the L5 dorsal ramus. Overall, eight of nine subjects were satisfied with the procedure, with two thirds experiencing significant pain relief 1 year after treatment. The primary concern with this approach is that tissue impedances are highly variable in the SI joint complex, which may result in asymmetrical, nonconfluent lesions, or even technical failure.

Combination of Ligamentous and Neural RF Ablation

Gevargez et al. [67] performed a study to evaluate CT-guided percutaneous RF denervation of the SI joint in patients with low back pain. The procedure was performed on 38 patients who only temporarily responded to CT-guided SI joint blocks. The RF lesioning was performed in three locations in the posterior interosseous SI ligaments, and once on the dorsal ramus of the fifth spinal nerve. Three months after the therapy, 13 patients (34.2%) were completely free of pain. Twelve patients (31.6%) also reported substantial pain reduction, seven patients (18.4%) obtained slight benefit, and three patients (7.9%) no pain relief. The principal drawback of this procedure is that it targets only a small portion of the ligamentous connections of the SI joint, and leaves most of the nerve supply intact [67].

COMPLICATIONS

Postdenervation neuritis can occur after SI joint neurotomy and usually resolves within 6 to 8 weeks. Preemptive injection of low-volume corticosteroids via the RF introducer cannula just after ablation may help reduce the onset of neuritis [77]. Theoretically, bleeding and infection can occur with any percutaneous procedure, though RF denervation may exert a protective effect against bacterial growth [78]. Less frequent complications include nerve damage and allergic reaction to local anesthetic. In the event the electrode is misplaced, muscle weakness or incontinence can result from injury to the spinal nerve or ventral ramus. For patient comfort and to avoid any patient movement, intravenous conscious sedation may be prudent and may allow more patient comfort.

CONCLUSIONS AND FUTURE RESEARCH

SI joint pain is a common cause of axial low back pain, affecting between 15% and 25% of people [79]. In patients who obtain significant but short-term benefit from diagnostic blocks, RF denervation may provide a reasonable treatment alternative. Based on preclinical and clinical studies, the ideal candidates for RF denervation may be younger patients with suspected extra-articular pathology. Several techniques have thus far been described, but current evidence favors lateral branch RF lesioning as the most effective treatment option. When selecting patients, neither double comparative blocks nor prognostic lateral branch blocks have been properly studied. Studies conducted in cadavers have demonstrated that the L5-S3 levels should be targeted in most people, though some individuals may benefit from lesioning L4 and S4 as well. Indirect evidence has shown that cooled-probe technology can enhance lesion size, and may thus improve treatment outcomes. However, randomized comparative trials are needed to definitively establish superiority [80–82].

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17 Midline Posterior Element Disorders

Sarah Knievel and Tim Lamer

BAASTRUP'S DISEASE AND INTERSPINOUS LIGAMENT PAIN

Commonly identified pain generators of the low back include the intervertebral discs, zygapophysial joints, myofascial structures, and sacroiliac joints. Less commonly appreciated generators include the posterior ligaments (interspinous and supraspinous ligaments), spinous processes, and pseudoarthroses involving adjacent spinous processes or Baastrup's disease. This chapter will review the normal anatomy, physiology, and pathophysiology relating to the lumbar spinous processes and associated ligamentous structures before discussing current treatment and diagnosis.

Normal Anatomy

Spinous Process

The spinous processes of the lumbar spine project dorsally and caudally from the junction of the laminae. The spinous process of L5 is the least substantial and the tip is often blunted. In the sacrum, the continuation of rudimentary spinous processes is represented by the spinous tubercles. The spinous processes act as substantial levers by providing areas for lumbar muscle and ligament attachments. All of the muscles that are involved in spinal movement and stabilization are attached solely to the posterior elements of the vertebral column. As a result, the spinous processes are subjected to significant forces with movement or stabilization of the spine [1]. T2 and T1 sagittal magnetic resonance imaging (MRI) of normal lumbar spinous processes are moderately bright because of the marrow component with lower signal representing the cortical margins [2].

Interspinous Ligament

The interspinous ligament spans between adjacent spinous processes and consists of connective tissue formed of collagen fibers. Anteriorly, it is continuous with the ligamentum flavum and posteriorly it merges with the supraspinous ligament. Between L5 and S1, the outermost layer of the interspinous ligament contains fibers from the aponeuroses of the longissimus muscle that contributes to the stability at that level. Within the ligament, there are three symmetric layers orientated in different planes to resist motion from different directions. This ligament functions primarily to prevent excessive spinal flexion and separation of the adjacent spinous processes. In addition, cadaveric studies suggest that certain layers of the interspinous ligament are oriented in an oblique direction, allowing it to aid with controlling vertebral rotation during flexion helping the facet joints remain in contact while gliding [3]. Microscopic evaluation demonstrates dense innervation of the posterior portion of the interspinous and supraspinous ligaments with Ruffini and Pacini corpuscles originating from the dorsal rami. The role of the sensory nerve endings is thought to provide proprioceptive information and protection against excessive stretch or compression [4]. T1 and T2 MRI sagittal views of the interspinous ligament demonstrate a nonhomogeneous intensity with alternating areas of moderate and high signals as a result of the different orientations of the layered fibers.

Supraspinous Ligament

The supraspinous ligament is a fibrous cord attached to the posterior tips of the spinous processes starting at approximately C7 and extending distally to L4-L5. It does not extend to the sacrum but there is a substitution of fibers from adjacent muscles and fascia that contribute to the lumbosacral junction. Although it is not as substantial as the interspinous ligament, it contributes to limit forward flexion of the lumbar spine and resist separation of the adjacent spinous processes. On both T1 and T2 sagittal MRI, the supraspinous ligament is seen as a linear band of low intensity signal across the tips of the spinous processes. Normally, it is taut between the adjacent spinous processes and curves with the lordosis of the lumbar spine [2].

Pathoanatomy and Pathophysiology

Degenerative Changes

Degenerative changes in the lumbar spine do not occur in isolation and are often associated with other structural changes. Hypertrophy of the tips of the spinous processes may occur in elderly persons especially those with an occupational history of long periods of back flexion or previous

trauma. This condition is usually associated with chronic postural hyperlordosis and regional loss of disc spacing [5]. In the setting of compromised disc height and facet arthropathy, the interspinous ligament becomes redundant and can lead to near or true collision of the vertebral spinous processes ("kissing spine"), osteophytosis, eventual neoarthrosis formation, and possible bursal cavities between adjacent spinous processes (see Figure 17.1). This potentially painful pseudoarticulation was first described by Dr Christian Baastrup in the 1930s and is often referred to as Baastrup's syndrome or Baastrup's disease. The redundancy and hyperplasia of the interspinous ligament may extend into the posterior aspect of the spinal canal leading to central canal stenosis [6]. In addition, there are case reports documenting the development of intraspinal posterior epidural cysts associated with Baastrup's disease that resulted in posterior central compression of the thecal sac [7]. Epidemiologic evaluation of 539 patients undergoing MRI for lumbar or leg pain revealed the prevalence of lumbar interspinous bursae in 44 patients or 8.2%, noting the presence was infrequent but not rare [8]. Unfortunately, this study did not correlate the clinical diagnosis of the patients with the radiologic findings. In patients with low back pain undergoing diagnostic injections, DePalma et al. found a prevalence of 1.8% (95% confidence interval [CI]: 0.6–5.1) [9].

The interspinous ligament also has characteristic degenerative changes that can contribute to the generation of low back pain. As early as the 1950s there was surgical evidence of interspinous ligamentous rupture in patients undergoing disc surgery [10]. With age, thickening of collagen fibers and calcification of the posterior ligaments at the attachment to the spinous processes can cause progressive stiffening and potential rupture of the interspinous and supraspinous ligaments which can lead to decreased lumbar mobility and dysfunction (see Figure 17.2). Although the interspinous and supraspinous ligaments are innervated, the extent to which they are responsible for pain generation has not been determined.

Traumatic Changes

Fractures to the spinous processes and associated ligamentous structures primarily occur as a result of direct trauma or severe muscular contractions. The integrity of the interspinous and supraspinous ligaments can only be overcome with violent traumas exceeding their limits of extension [4]. Treatment consists of symptomatic bracing for immobilization and usually do not require surgical intervention as they do not compromise the structural stability of the vertebral column. Severe distraction or flexion injuries of the lumbar spine that disrupts the interspinous, supraspinous ligaments and possibly disc space with or without concurrent bony disruption often heal slowly and necessitate surgical stabilization [11].

Diagnostic Evaluation

Clinical Features

Degenerative changes of the lumbar spinous processes and surrounding ligamentous structures can manifest clinically as localized midline low back pain. The pain

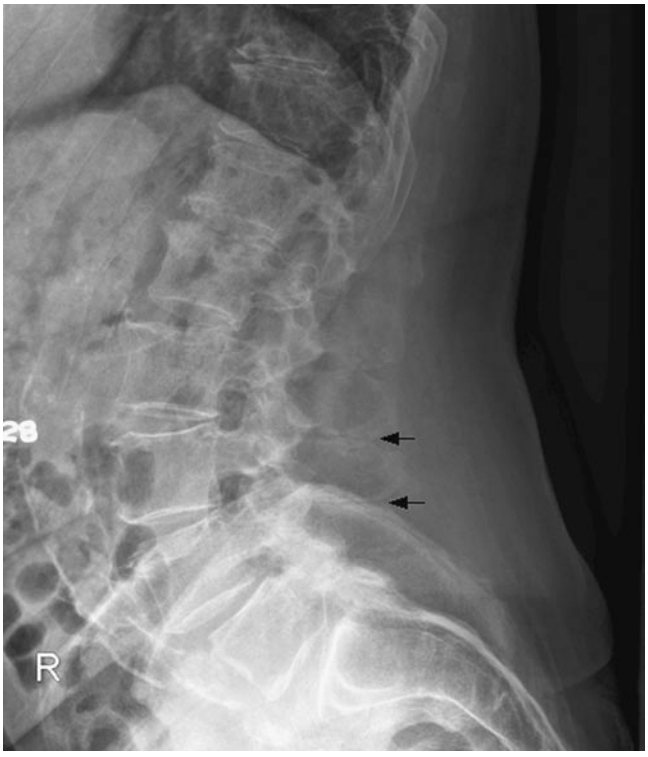

Figure 17.1 Lateral x-ray demonstrating direct contact between adjacent lumbar spinous processes.

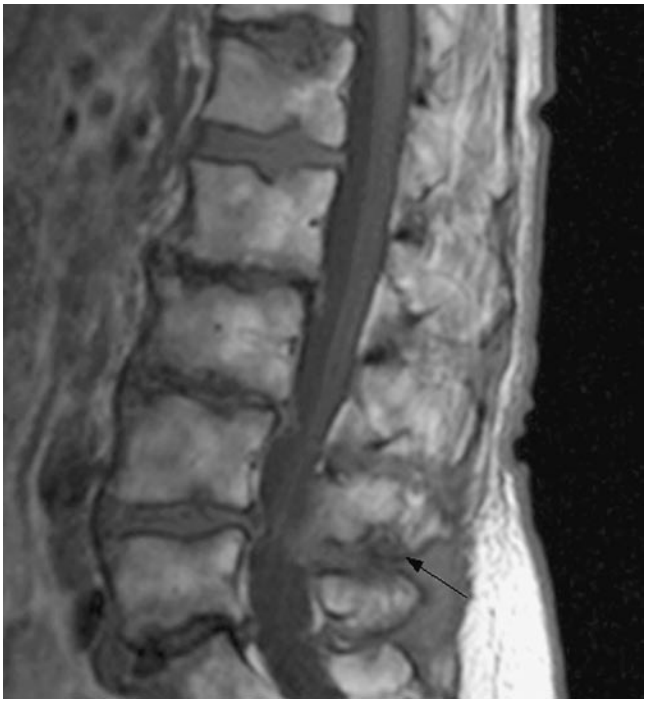

Figure 17.2 Sagittal T1 weighted MRI demonstrates opposing spinous processes and associated sclerosis with flattening of the appositional surfaces.

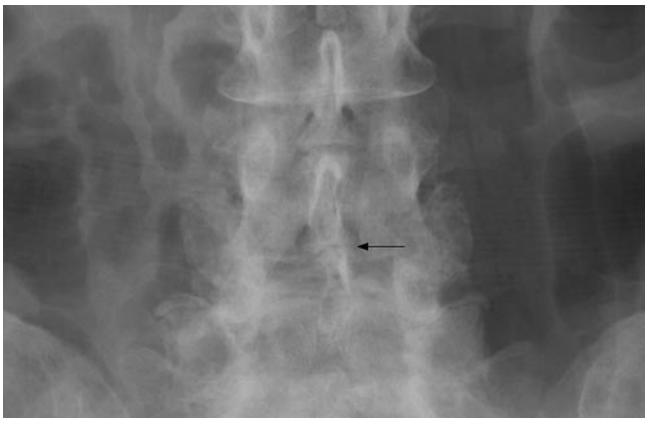

Figure 17.3 Posterior-anterior radiograph of the lumbar spine demonstrating very closely approximated L4 and L5 spinous processes.

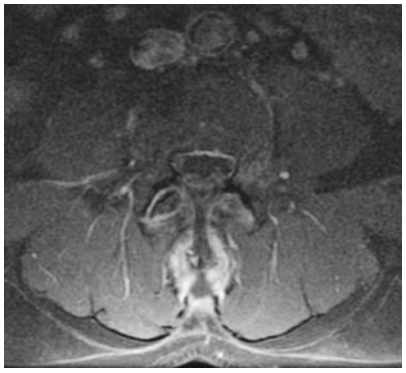

Figure 17.4 Axial post-contrast fat-suppressed T1-weighted image demonstrates intense enhancement within and around the dorsal portion of the interspinous ligament and geode formation in the spinous process.

may be described as sharp or deep ache, often worse with activities that increase lumbar lordosis or compression of these structures. History often reveals an insidious onset without associated trauma [12]. Palpation of the midline back and spinous processes may reproduce their symptoms [12]. Physical examination maneuvers like stork test (standing on one leg with passive extension of the lumbar spine) or active spinal extension [12] can reproduce their symptoms.

Radiographic Features

Radiographic features of spinous processes pathology can be recognized on MRI or x-ray imaging. Baastrup's disease is characterized by direct contact between adjacent lumbar spinous processes (see Figure 17.3). Often there is associated sclerosis, enlargement, and flattening of the appositional surfaces. MRI findings may include edema [12], inflammation, and possible geode formation between the spinous processes. This is demonstrated in fat-suppressed T2 images as regions of nonenhancing signal and in T1 contrast-enhanced fat-suppressed sequences this is seen as areas of enhancement (see Figure 17.4). In addition, the development of adventitious bursa with possible creation of synovial articulations can be appreciated on MRI [6]. This can be seen as a fluid-like signal intervening between consecutive spinous processes in fat-suppression sequences. Imaging of pathologic changes in the interspinous and supraspinous ligaments is seen only on MRI and often demonstrates tears, redundancy, and calcification at the insertion site on the spinous processes.

Diagnosis

Identification of the spinous processes or ligamentous structures as the primary pain generator requires close correlation between the physical examination and imaging. Physical examination alone is notoriously unreliable, and degenerative changes occur commonly in imaging studies of patients older than 65 years. Often, other

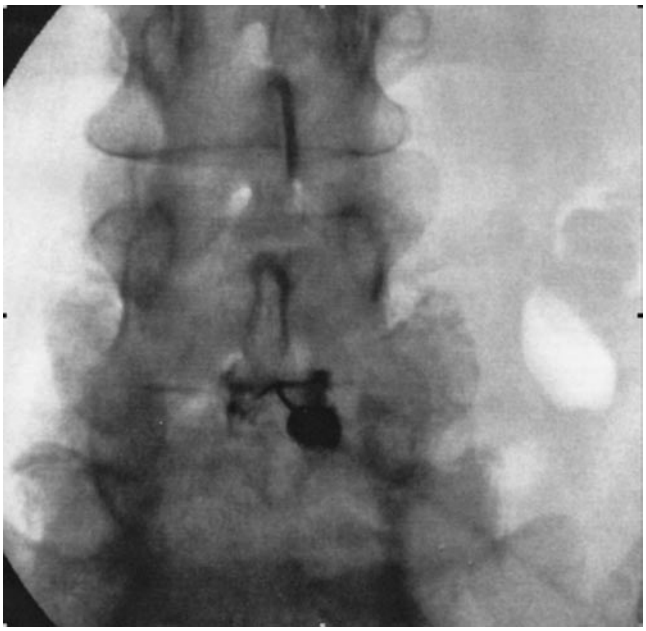

Figure 17.5 Posteroanterior image of interspinous injection with contrast infiltrating the narrowed space between the L4 and L5 spinous processes.

structural sources of pain need to be ruled out, and, if possible, fluoroscopic palpation for confirmation of the area of tenderness should correspond to the affected spinous processes or structures. Isolated tenderness to palpation of the posterior elements and corresponding changes on imaging can be suggestive of underlying spinous process or ligamentous disease. Fluoroscopically guided, contrast-confirmed intraligamentous injection of local anesthetic can be utilized to confirm a clinical impression of lumbar interspinous symptomatology [12]. Although it has not been studied, it would seem logical that temporary pain relief following image-guided local anesthetic injections into the suspected symptomatic structure would help diagnostically.

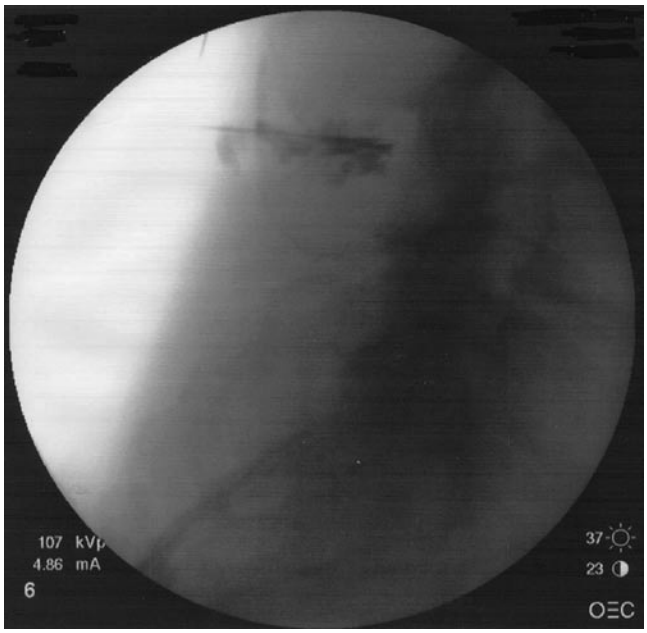

Figure 17.6 Contrast injected through the needle shown in this figure can be seen to infiltrate a majority of the space between the L3 and L4 spinous processes.

Treatment

Conservative

First-line conservative treatment should consist of local modalities, over-the-counter analgesics, and physical therapy focused on neuromuscular education of the core muscles and posture in a flexion bias and stretching of the hip flexor groups.

Interventional

Literature is lacking regarding definitive interventional treatment of spinous process and associated ligamentous disease. Some published interventional options discuss injection of the interspinous ligament and adjacent spinous processes for diagnostic [12] and therapeutic purposes [13]. The injection technique involves placing the patient prone, sterile preparation of the region, and infiltration of the area with local anesthetic. A 22-gauge styletted needle is advanced under posterior-anterior fluoroscopic guidance directly between the affected spinous processes [12,13]. Lateral imaging should visualize the needle tip approximately midway along the dorsal-ventral axis of the spinous processes (see Figure 17.6). Injection of 1 mL of radiopaque contrast should spread between the target spinous processes. This will be followed by injection of 1 to 3 mL of local anesthetic mixed with steroid [12,13]. For acute to subacute pain, an injection may result in long-term improvement. Persistently symptomatic Baastrup's disease or interspinous bursitis can be treated with bilateral medial branch neurotomies at the affected segmental level [14]. Refractory cases may require surgical referral for removal of the involved spinous processes is an option.

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18 Fusion Hardware Mediated Low Back Pain

Amit Bhargava

INTRODUCTION

The first successful fusion procedures were described by Hibbs and Albee in 1911 for prevention of progressive deformity from Pott disease [1–3]. In recent decades, spinal fusion surgeries have increased markedly in frequency [1]. The majority of spinal fusion cases are being performed for degenerative spine conditions, such as disc degeneration, spinal stenosis, and degenerative spondylolisthesis [4].

The effects of these surgical interventions to the biomechanical and biologic integrity of the spine are still not completely understood. Nevertheless, lumbar spine fusions are the gold standard in operative treatment for advanced stages of degenerative changes or higher grades of spondylolisthesis [5]. Despite developments in fusion technologies, the incidence of residual or recurrent postoperative back pain (so-called failed back syndrome) remains high because of the influence of multiple factors [1,6].

Pain after fusion surgery may be due to many conditions such as pseudoarthrosis, infection, flat back, adjacent level disc or facet joint degeneration, and painful implants [1–37]. Cook et al. [12] introduced the concept of "late operative site pain" of unknown etiology as the most frequent indication for reoperation. Late operative site pain, defined as pain around the site of operation beginning at least 6 months following implantation, is a relatively common problem associated with the use of large spinal implants. The exact cause has been suggested to be due to metal allergy, tissue reaction to particulate debris, or low-grade infection in connection with debris [12–16].

PATHOLOGY

Corrosion has been observed in the majority of the types of spinal instrumentation [12,17–19]. Alanay et al. [7] reported no corrosion in their patients. Cook et al. [12] found corrosion to be a common finding but was not present in all of their patients. The prevalence of corrosion cannot be determined in asymptomatic patients [12].

Corrosion of orthopedic implants is generally a slow but progressive phenomenon with undesirable effects. It can lead to mechanical failure, and the release and dissemination of corrosion particles can produce adverse biological reactions in the host [17,20]. The clinical importance of degradation of metal implants is evidenced by particulate corrosion and wear products in tissue surrounding the implant. Wear and/or corrosion were observed even before one full year of implantation [21].

There are multiple mechanisms of corrosion which alter the surface of the fusion hardware. Fretting corrosion is due to mechanical damage from interfascial micromotion and can result in wear debris. Fretting is defined as the deterioration of the interface between contacting surfaces as a result of corrosion and slight oscillatory slip between the two surfaces [22,23]. Crevice corrosion results from rigid interconnections in tissue fluid environments. It is defined as a localized corrosion of metal surface at, or immediately adjacent to, an area that is shielded from full exposure to the environment because of close proximity between the metal and the surface of another material [22,23]. Rigid interconnections provide the opportunity for oxygenated biologic fluids to enter into the internal spaces between the component parts, causing corrosion due to variation in oxygen tension and pH. Galvanic corrosion is defined as accelerated corrosion of a metal because of its contact with a more noble metal or nonmetallic conductor in a corrosive environment. These three forms of corrosion can occur simultaneously [22–24]. Corrosion is considered a mechanical (wear) or electrochemical phenomenon, but the importance of biological agents is now being considered [20]. Biocorrosion—or microbially influenced corrosion—is a concept to be taken into account [17,25]. Also, synergistic interactions may exist between the metal surface, corrosion products, and bacterial cells and their metabolites, which increase the rate of corrosion of the metal [17].

Many authors have reported increased concentrations of local and systemic trace metal in association with metal implants [20,26–56]. Senaran et al. [13] reported that the site of implant degradation products were found abundantly around transverse rod connector-rod connection areas (metal-metal interface) and in lesser amounts around rods (metal-muscle interface) and pedicle screws (metal-bone interface). The diameters of particles around transverse rod connectors measured greater than those of the particles in the other regions [13].

The metals in contact with biologic systems corrode [20,57,58], and the released ions, while not sensitizers on their own, may activate the immune system by forming complexes with native proteins [58–61].These metal-protein complexes are considered to be candidate antigens (or more

loosely termed, allergens) for eliciting hypersensitivity responses [61]. Metals known as sensitizers (haptenic moieties in antigens) are beryllium [62], nickel [62–65], cobalt [62], and chromium [62], although occasional responses have been reported for tantalum [58,66], titanium [67,68], and vanadium [66]. Nickel is the most common metal sensitizer in humans, followed by cobalt and chromium [63–65,69]. The incidence of metal sensitivity among the general population is approximately 10% to 15%, with nickel having the highest sensitivity (approximately 15%) [69]. Cross reactivity between nickel and cobalt is purportedly most common [58,69]. Senaran et al. [13] identified two different types of debris: one rich in iron (Fe) and the other Chromium (Cr).

During reoperation after fusion surgery, observations made were corrosion [12], metal debris [12,70,71], tissue discoloration[12], and bursae tissue formation around the implants [7,12]. Histopathology revealed an acute and chronic inflammation with granuloma formation [72–74]. Intracellular metallic debris [12] and macrophage cellular response [71] has been observed. Any signs suggesting allergic reaction or infection were not found on histologic examination [13]. Spinal implant particulate debris elicits a macrophage-mediated response leading to increased levels of local proinflammatory cytokine tumor necrosis factor production, subsequent osteoclastogenesis, and cellular apoptosis [58,70].

CLINICAL PRESENTATION

It is of utmost importance to find the source of recurrent low back pain and treat it appropriately. Repeat fusions have higher risk of perioperative mortality [75]. Patients will usually present with low back pain postoperatively with tenderness in the surgical area. Patients usually have paramidline low back pain which can be reproduced by direct digital palpation. Delayed neurologic symptoms caused by intraspinal metallosis, radiculopathy, and paraparesis have been reported [17,76,77]. Patients may present a fluctuant mass or, more commonly, drainage, months or years after surgery. Late drainage is observed in some patients in relation to corroded implants. It is believed to be caused by bacterial infection [14], whereas others believe it is caused by corrosion and the action of metal degradation products [17,19]. Fluctuant mass and drainage is evaluated and treated as infection, until ruled out otherwise. No signs of infection have been identified in this group of patients. [7,13,17].

Investigations

Patients usually have routine hematologic investigations (complete blood count with differential, erythrocyte sedimention rate, C-reactive protein) within the normal range [7,17]. Cultures positive to low-virulence skin organisms, such as *Propionibacterium acnes*, may be obtained, but most patients have negative cultures [17]. Under investigation is the role of metal levels in urine or blood, which may be useful in the future, to monitor spinal implants and in early diagnosis of corrosion [17,78].

Radiography is the standard follow-up imaging method, and it provides a great deal of useful information. Radiologic imaging studies can identify postoperative complications such as incomplete fusion, hardware failure, suboptimal positioning of instrumentation, infection, and hematoma. By accurately identifying complications of spinal instrumentation, the radiologic investigations can play an important role in evaluation of persistent postoperative pain [1]. However, they do not provide information regarding possible soft tissue inflammation related to the fusion hardware. Because of the artifacts observed on magnetic resonance imaging films from the metal, it is difficult to observe any soft tissue reaction around the instrumentation.

To further delineate the source of pain, diagnostic injections may be performed (Figure 18.1). Alanay et al. [7] noted that the most consistent predictor of pain relief after implant removal is the percent pain relief after the diagnostic injection of the painful regions on and around the operative site. In a prone position, the most tender area over the skin is marked. The skin over the lumbar region is prepared in a sterile fashion. Skin is anesthetized at the tender area, and 3.5-inch, 22-gauge needle is advanced to under fluoroscopic guidance onto the posterior margin of the hardware. The needle position is confirmed in anteroposterior and lateral views or oblique views. Aspiration is done to rule out any fluid collection. Injection should not be done if aspirated fluid is purulent or there is any draining site. A small volume of radioopaque contrast dye is injected to confirm the position of the needle and observe the flow of the dye. The contrast dye will localize at the site of injection and not flow anywhere else (Figure 18.2). Needle position may be adjusted if any vascular flow is observed. About 0.5 mL of local anesthetic (0.5% Marcaine or 2% lidocaine) is injected

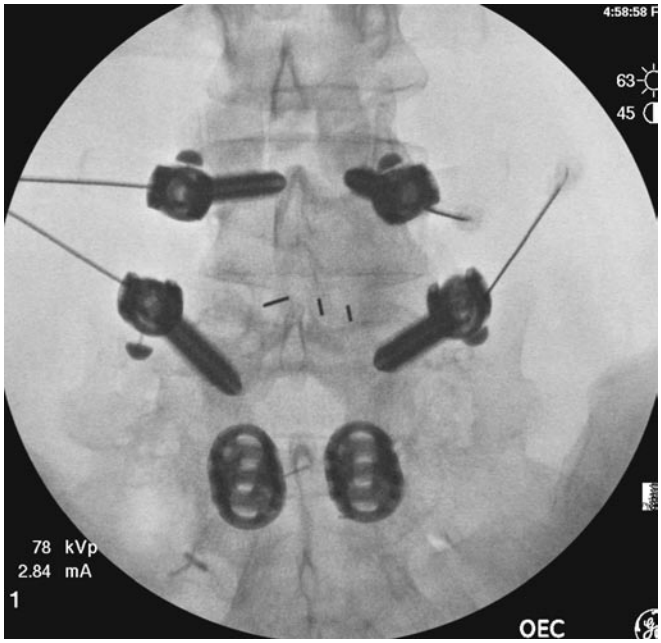

Figure 18.1 Anteroposterior fluoroscopic image of needle placement over the posterior margin of the bilateral L4 and L5 pedicle screws.

into the soft tissue around the hardware. A similar procedure can be performed at other tender regions.

TREATMENT

All patients with recurrent back pain should be offered a regimen of pain management for chronic and breakthrough pain plus physical therapy [79]. Analgesic medication should be prescribed judiciously as analgesic-related deaths are reported to be responsible for more deaths and more potential life lost among workers who underwent lumbar fusion than any other cause [75]. Physical therapy has been shown to be effective at decreasing pain and improving function in adults with chronic low back pain in several meta-analyses or randomized controlled trials [1,79–81]

Implants tender to palpation have been removed if there is unexplained pain in the region of underlying implant in the absence of infection and has been considered an appropriate procedure for patients [72,82–85]. Diagnostic injections may be done before implant removal as they were noted to be the most consistent predictor of pain relief after implant removal [7].

Authors' Preferred Method of Treatment

If the diagnostic block is positive, the patient undergoes a therapeutic injection with steroid and local anesthetic at each of the positive block sites. On the basis of the operative findings, we are treating this as a bursitis and inflammation around an irritant. Compared to reoperation, which may or may not include refusion, this is a simpler and easier procedure for the physician and patient. We re-examine the patient in 2 weeks after the therapeutic injection. On the basis of the duration and type response, we may recommend further injections up to a maximum of three in a year. If the injections do not successfully reduce the patient's low back pain, implant removal may provide a significant pain relief for carefully selected patients with implant-related pain [7].

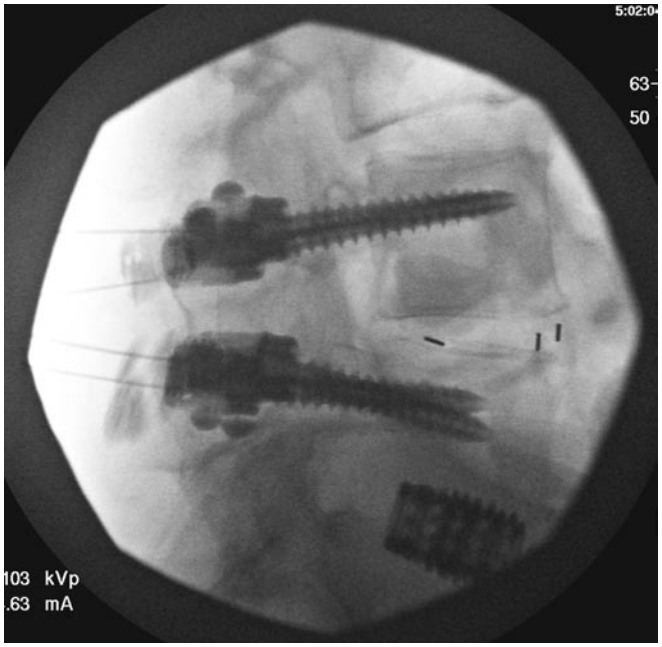

Figure 18.2 Lateral fluoroscopic image of focal collection of injected contrast dye over the posterior margin the L4 and L5 pedicle screws.

CONCLUSION

There are multiple causes of persisting low back pain. It may be appropriate to perform diagnostic injections to rule out the lumbar fusion instrumentation causing the pain. Corrosion cannot be source of pain in all patients as one study did not show any corrosion and had positive diagnostic injections [7], whereas another showed corrosion in some patients and not in other patients [12].

The tissue surrounding modern implants may include areas of osseointegration and fibrous encapsulation, and there may be a variable foreign-body response. Tender areas are usually typical clinical findings of patients having fusion with instrumentation. As the magnetic resonance imaging and x-rays are unable to assist with the evaluation of soft tissue around the instrumentation at the present time, the role of ultrasound evaluation of the tissue around the instrumentation may further be studied. After a diagnostic injection of the painful regions on and around the operative site, therapeutic injections may be performed. Further studies are needed to evaluate the long-term efficacy of therapeutic injections. Removal of the implant may provide significant pain relief and is a safe procedure for carefully selected patients with implant-related pain [7]. The patients should be informed that the likelihood of reoperation following a lumbar spine operation is substantial [86].

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19 Lumbosacral Spine Disorders in the Young Athlete

Joshua A. Thomas, Jared S. Greenberg, and Larry H. Chou

INTRODUCTION

Treating low back pain is a challenging endeavor. Back pain is virtually ubiquitous and is the second leading cause for primary care office visits. Treating back pain in athletes can be even more difficult. Athletes often have higher pain thresholds, fear of potential loss of playing time, the need to rapidly return to play, and loss of identity if unable to play. Treating the young athlete with low back pain requires not only a comprehensive knowledge of the spine but also an understanding of the external and psychological factors influencing their recovery.

The spine's structure and physiology are well adapted to generate, tolerate, transfer, and dissipate internal and external forces. However, the composition and design do have finite endpoints for failure, resulting in injury and loss of function. The objective of this chapter is to review the anatomy, physiology, presentation, diagnostic work-up, and management of spondylolysis, spondylolisthesis, disc and endplate injury, and paraspinal compartment syndrome in the young athlete.

SPONDYLOLYSIS

Anatomy

Spondylolysis is defined as an acquired or congenital defect in the pars interarticularis of the lumbar spine. The pars interarticularis is the part of vertebra located between the inferior and superior articular processes of the zygapophyseal joint (or facet joint). It runs obliquely from the lateral to superior border of the lamina [101]. In spondylolysis, there is a bony defect in the pars interarticularis on one or both sides of the neural arch. Figure 19.1 shows a unilateral defect. When this occurs bilaterally, there can be displacement of one vertebra on another. This is called spondylolisthesis and is discussed later.

Etiology/Pathophysiology

The exact etiology of spondylolysis is uncertain, but genetic predisposition and repetitive traumas have been implicated [1]. The acute variety is more common in young athletes. The congenital variant can be associated with spina bifida occulta. Regarding acute spondylolysis, it is almost uniformly thought to represent a fatigue fracture in the pars in the setting of repetitive motion especially flexion, extension, and rotational forces [2]. There is controversy whether the fatigue fracture is caused by repetitive loading and stress or there is a single microfracture which progresses as a result of repetitive overload. Wiltse et al. [3] support the fatigue fracture theory in spondylolysis. They hypothesized that these fractures develop early in life, have a hereditary basis, and may be incidental findings on x-rays. Farfan et al. [4] theorized that lysis of the pars may result from sudden, increased forces in multiple directions and hypothesized that torsional forces are the most disruptive. Some authors have looked at mechanical loading on the pars. Cyron and Hutton studied forces on cadaveric vertebrae to cause fractures of the pars interarticularis in vivo. They placed repetitive forces on the inferior articular facets with a rig utilizing cyclic loading and showed that after a certain number of cycles the pars would fracture

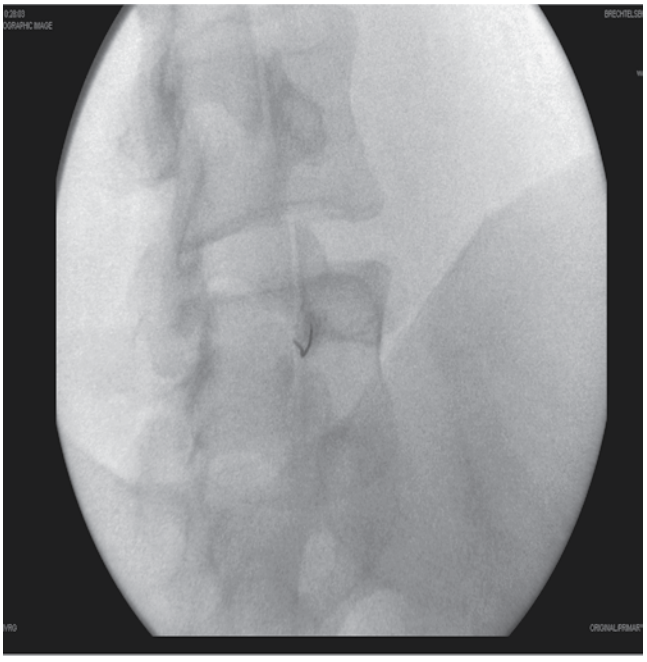

Figure 19.1 Oblique x-ray L5 spondylolysis. Note needle tip at fracture line.

in 53 of 74 vertebrae. They also showed that the neural arch tended to strengthen after the fourth or fifth decade and the possibility of developing a spondylolytic fracture decreases after age 50. In addition, they concluded that female spines were more susceptible to fracture in vivo [5]. However, males have been shown to be twice as likely to develop spondylolysis as females in longitudinal population studies [6].

Incidence/Prevalence

The overall incidence of spondylolysis has been reported to be about 3% to 6% [7,8]. Spondylolysis is prevalent in the general population but is often asymptomatic and detected incidentally on plain x-rays. When spondylolysis is of a congenital or chronic nature (due to a remote fracture), it is more often asymptomatic. Acute spondylolysis can be more often painful. Unfortunately, both can lead to substantial disability and pain. Spondylolysis is the most common cause of persistent low back pain in young athletes and should be considered in every adolescent with functionally limiting low back pain [9]. In a study of 100 adolescents with persistent back pain versus 100 adults, spondylolysis was found in 47% of the adolescents and only 5% of the adults [10]. Fredrickson et al. studied 500 first-grade children and 500 newborns. They found no incidence of spondylolysis in healthy newborns with plain film imaging. They demonstrated the incidence in children to adults to be 4.4% at age 6 with an increase to 5.2% by age 12 and 6% by adulthood [8].

Regarding young athletes, spondylolysis has been reported in almost every sport but has been disproportionately higher in sports such as gymnastics and swimming, which involve repetitive lumbar extension. Jackson et al. [11] found an 11% incidence in female gymnasts which was four times higher than there nonathletic female peers. Rossi and Dragoni found 390 cases in 3132 competitive athletes. The incidence in diving (43.13%), wrestling (29.8%), and weight lifting (22.86%) were the highest [12]. Both studies used plain radiographs for diagnosis, which are less sensitive than other imaging techniques. Consequently, the incidence of spondylolysis may be even higher.

Clinical Presentation

Symptomatic spondylolysis most commonly presents with axial back pain, which may radiate to the buttock, thigh, or hamstrings [1,7,13]. Patient may describe pain with hyperextension or rotational movements. Neurologic complaints are not infrequent in the adult athlete. Night pain and constitutional symptoms are not consistent with this diagnosis and should alert the practitioner of other etiologies of the patient's pain. Other elements of the sports-specific history are important to gather such as specific demands for the individual sport, training regimen, and competitiveness of the patient.

Physical examination should include a detailed neurologic examination. Pertinent examination findings include postural changes, increased lumbar lordosis, scoliosis, flattening of the back, or shortening of the waistline [1,13]. There are no validated physical examination findings for spondylolysis. The only reported consistent finding is reproduction of pain with single-leg hyperextension test, or stork test (Figure 19.2). Jackson et al. [14] studied seven young athletes with a stress reaction involving the pars interarticularis and determined that the single-leg hyperextension test consistently elicited pain on the ipsilateral side. Kraft presented data at a national conference that this test was very sensitive but only 50% specific for pars fracture [15]. This maneuver may help aid in the diagnosis of spondylolysis but has not been studied extensively. Some authors have suggested that extension and rotation maneuvers are reliable in provoking pain in patients with spondylolysis [7,16].

Diagnostic Evaluation

Any athlete complaining of persistent pain should undergo initial radiographic evaluation. Views include anteroposterior, lateral, and oblique plain films. Lateral radiographs are the most sensitive projection and demonstrate a linear lucency in the pars. This lucency can also be seen on oblique radiographs, the most specific projection [17]. Flexion/extension views are useful in assessing stability of the involved segment. Plain films are, however, less sensitive and specific than other imaging techniques to adequately diagnose pars pathology relative to other imaging modalities. In addition, acute spondylolysis

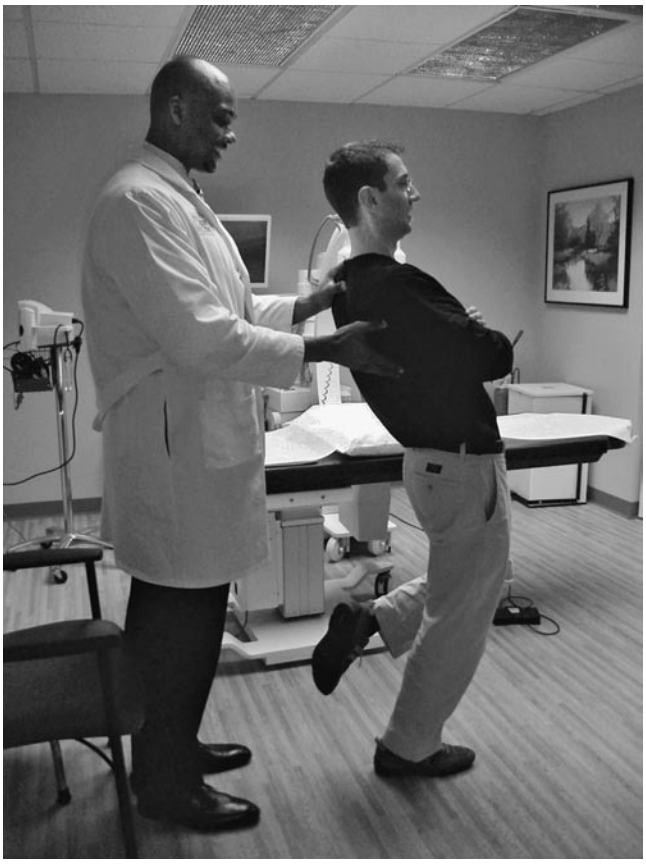

Figure 19.2 Standing stork test.

versus chronic spondylolysis is impossible to differentiate on plain x-ray. Only recent studies have used advanced imaging to diagnose spondylolysis. Older studies which used only plain radiography likely missed many cases of spondylolysis and assumed incorrectly that a fracture was acute rather than chronic [7].

Plain radiography was the original tool to diagnose spondylolysis. The defect in spondylolysis is commonly described as the broken neck of the Scotty dog. For optimum visualization, the defect should be aligned tangentially to the radiographic beam. Unfortunately, the pars is situated oblique to all three orthogonal planes so is not seen clearly on standard radiographic views [18]. Blanda et al. supported this notion in a series of 82 patients of whom they discovered spondylolysis in 66 of them. In eight of the patients plain x-rays were normal, but these same patients had positive bone scans. This demonstrated the importance of utilizing advanced imaging in spondylolysis [19].

Magnetic resonance imaging (MRI) (Figure 19.3), computed tomography (CT) (Figure 19.4), and bone scintigraphy with single photon emission computed tomography (SPECT) (Figure 19.5) are often used to better visualize spondylolysis and spondylolisthesis. These have been used when x-rays were negative and clinical suspicion remains high. Masci studied 71 young, active subjects with low back pain and compared the aforementioned advanced imaging techniques. The authors also looked at clinical assessment using the one-legged hyperextension test (stork test). Of these subjects, 50 pars interarticularis demonstrated acute spondylolysis as defined by bone scintigraphy, the gold standard. MRI revealed bones stress in only 40 of these. In the aforementioned group, CT scan revealed 19 fractures, of which MRI identified 18 showing agreement between these modalities for detecting fracture. The authors concluded that bone scintigraphy with SPECT should be the first-line advanced imaging investigation in athletes with low back pain followed by CT when it is positive. They also concluded that the stork test should not be relied on to exclude the diagnosis [9]. The algorithm of using SPECT as a first-line diagnostic modality followed by a thin-cut CT (1 mm axial cuts) through the abnormal segment to both confirm the diagnosis and stage the lesion for treatment is also agreed on by other authors [2]. There is a growing tendency to use MRI as a first-line diagnostic imaging tool. MRI has the added advantage of evaluating the disc and endplates. Marrow edema in the pars may signify a relatively recent injury in contrast to a congenital pars defect. Moreover, the lack of any ionizing radiation should also be considered in the young patient. However, CT scan is still recommended for evaluation of indeterminate cases and if assessment of healing is necessary [20].

Bone scan without SPECT lacks the specificity but is still very sensitive for increased uptake in the posterior elements. Bone scan with SPECT has higher sensitivity and specificity and is useful in distinguishing a lesion capable of healing with one that will likely not fuse. Increased metabolic activity at the pars interarticularis is associated with an acute injury whereas chronic or congenital pars defects do not typically show increased radiotracer uptake. CT scan is the preferred study to evaluate bony detail and evidence of active bone healing [1]. Campbell et al. considers MRI an effective first-line imaging modality for identifying acute stress fractures in the diagnosis of early spondylolysis. They compared MRI versus SPECT and CT as well as versus SPECT and CT in combination in 72 juvenile patients with likely spondylolysis. They

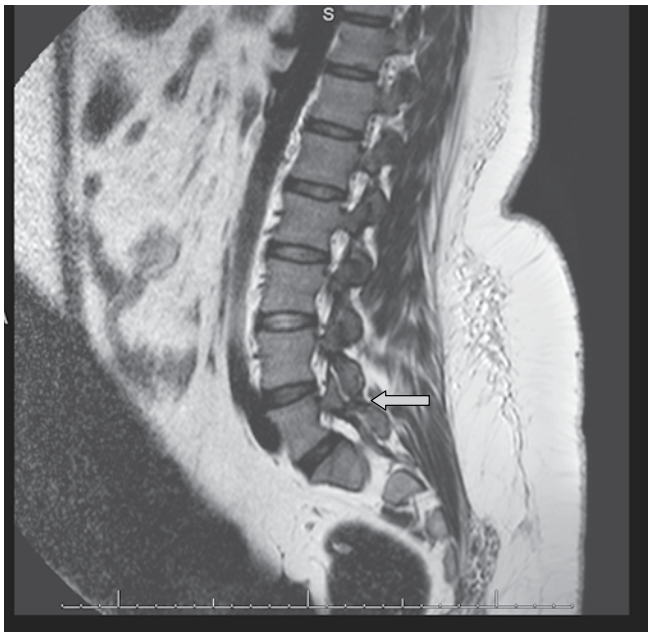

Figure 19.3 Sagittal short-tau inversion recovery (STIR) MRI L5 pars defect.

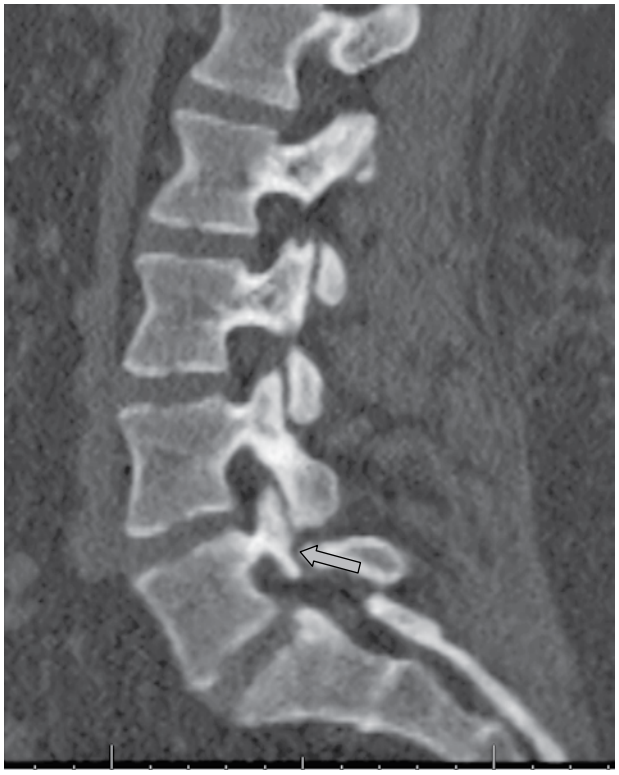

Figure 19.4 Sagittal CT—L5 pars defect.

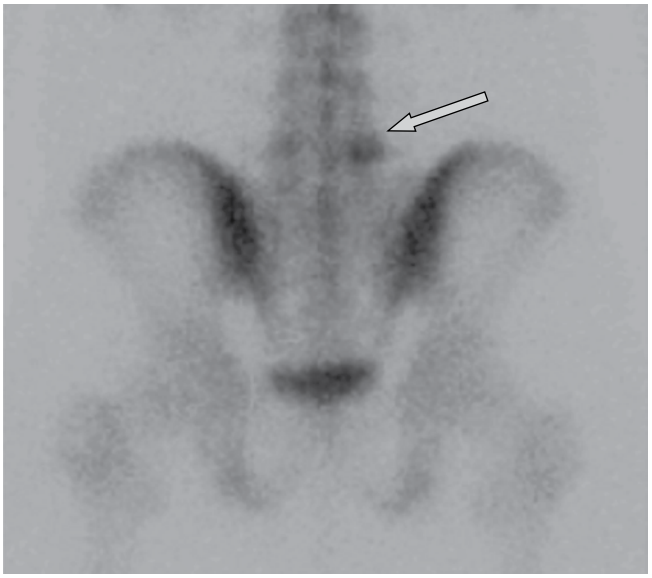

Figure 19.5 Bone scan with SPECT—L5 pars defect.

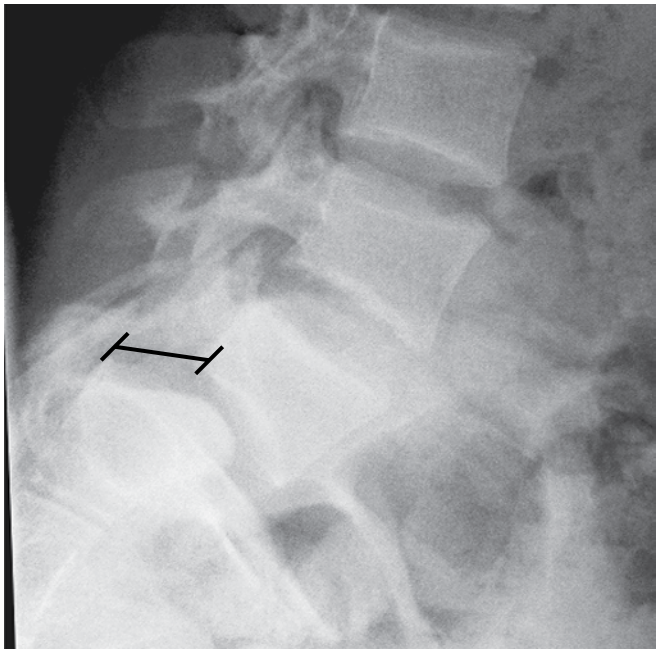

Figure 19.6 Lateral x-ray—grade III L5-S1 spondylolisthesis.

determined that the MRI, SPECT, and CT demonstrated a high level of agreement in identifying pars defects (κ = 0.794, 0.829, 0.786, respectively). MRI and SPECT differed in their ability to distinguish stress reaction in the absence of overt fractures, and partial versus complete fractures. CT was better at diagnosing indeterminate cases [20]. Because of the lack of ionizing radiation and the ability to image the neural elements, disc, and soft tissue abnormalities, MRI is an attractive secondary imaging option. SPECT has the disadvantage of not reliably being able to differentiate between spondylolysis and other pathology at the pars such as osteoid osteoma, facet arthritis, infection, and neoplasms [13,21].

Progression

With the acute or chronic fracture in spondylolysis, there can be displacement of one vertebra on another. Wiltse et al. described five subtypes of spondylolisthesis. Types I to V include dysplastic, isthmic, degenerative, traumatic, and pathologic. The isthmic type is the most common and important in athletes [3]. The amount of slippage is defined as a percentage of the slip of the vertebral body width between segments. Grade 1 is defined as 0–25% slip, grade 2 as 25–50%, grade 3 as 50-75%, and grade 4 as 75–100%. Pictured is a grade III spondylolisthesis (Figure 19.6). The most severe degree of spondylolithesis has been described as spondyloptosis. This is defined as the complete anterior dislocation of an adjacent vertebra secondary to a pars defect or abnormality. Almost 90% of spondylolytic defects occur at L5, with L4 being the next most common. L5 lesions typically result in L5 slippage on S1 [1,7].

Overall, the risk of progression of spondylolysis with or without a small grade spondylolisthesis is small, especially after adolescence [6]. Beutler et al. conducted a 45-year follow-up on the study of Fredrickson's school children mentioned earlier. Of the 30 patients diagnosed with spondylolysis, none with unilateral defects experienced slippage and there was no association with the degree of slip and back pain. In bilateral defects, 81% had some degree of slippage but progression was slow occurring mostly at a younger age. The average slip progression was only 11% of the baseline spondylolisthesis. The incidence of back pain increased in the entire population including those with and without spondylolysis over the 45-year study length. There was no significant difference in the pain experienced between groups [6]. Frennered et al. studied 47 patients younger than 16 years for a mean of 7 years with an initial slip of 9% to 14% and found that only 4% of these patients progressed to a 20% or greater slip [21].

Unilateral versus bilateral defects differ in their propensity to slip. In a study by Miller et al., [22] 44 young athletes with early spondylolysis (negative x-rays, positive bone scan) with bilateral or unilateral defects were studied after undergoing conservative treatment including bracing and rest. Radiographic follow-up of 11 subjects (of the 44 original patients) showed that none of the seven bilateral defects healed, with three progressing to spondylolisthesis. All four unilateral defects healed. The authors concluded that unilateral defects tend to heal, whereas bilateral defects may undergo slippage over time. They also assessed this group's functional outcome 7 to 11 years later and determined that 91% had good or excellent outcome scores and none required surgery. Early recognition and treatment of spondylolysis has been generally thought to prevent increased slippage and is associated with improved fracture healing and prevention of nonunion. However, in a study by Muschik et al., it was shown that in 86 asymptomatic young athletes with spondylolisthesis or spondylolysis who continued in competitive sports had similar rates of progression over a mean of 4.8 years when compared with other studies of similar populations

who did not return to competitive sports. This study did not have a control group. They also found no increase in symptoms despite daily, intense training. The authors concluded that prohibiting athletes with asymptomatic spondylolysis or spondylolisthesis from competitive sports may not be warranted [23].

Therapeutic Options

The treatment options for acute spondylolysis vary greatly, and there are no double-blind randomized controlled trials comparing treatment strategies [2]. There is also controversy on the duration or utility of bracing, restriction of activity, and utilization of surgical options. In the adolescent population, treatment may also depend on the skeletal maturity of the patient.

The physician must decide if the fracture is acute or chronic and treat accordingly. In an acute fracture, the goal is to promote healing and avoid nonunion. The mainstay of treatment is rest and activity restriction in the form of curtailing competition and training. Some authors recommended removal from athletics for a minimum of 3 months [1]. This is the minimum time pars defects have demonstrated healing on imaging studies [24]. If the fracture is chronic, rest can be instituted but bracing is rarely indicated for symptomatic relief.

Immobilization with a spinal orthosis has been used traditionally as an adjunct to activity restriction for acute fractures. There have been no controlled trials to support or refute this treatment approach. However, some authors recommend bracing in the setting of an acute pars fracture, especially for the young athlete [1]. Medicolegal factors may also influence the decision as to whether a brace is recommended or not. In general, studies have shown bony healing with the use of a rigid brace, soft brace, or no brace [7]. Steiner and Micheli studied 67 patients with a mean age of 16 years who had symptomatic spondylolysis or grade I spondylolisthesis treated with a modified Boston brace. The follow-up was 2.5 years. The duration of brace use was 6 months for 23 hours per day. Physical therapy was allowed when symptoms abated. They showed that 78% had excellent or good results, 13% continued to have mild symptoms, 18% showed bony healing, and 9% required surgery [25]. Bracing may be more useful as a reminder for the patient to limit activity than stabilizing fractures. It is rarely indicated in the older athlete and is used only when the older athlete is highly symptomatic. Bony union is more likely in the skeletally immature, particularly in those with young bone age. For this reason, bracing is often recommended in this population [26]. Bracing can be discontinued once there is evidence of bony healing as evidenced by decreased symptoms and bridging trabeculae across fracture sites visualized by CT. If the fracture has gone to nonunion, bracing can be discontinued once the patient is asymptomatic. There has been surprisingly little correlation between bony healing and outcome [7].

Bracing is not always recommended by physicians. Jackson et al. treated patients with activity restriction only without bracing in patients with early spondylolysis and were able to return 100% of patients with unilateral lesions and 50% of patients with bilateral lesions to their sport. It should be noted that their sample size was only seven young athletes [14].

D'Hemecourt et al. developed a treatment protocol for spondylolysis that is often cited. They reviewed 73 athletes treated with a Boston brace for 23 to 24 hours per day. The patients returned to sports in 4 to 6 weeks with bracing if pain-free. At 4 months, the brace is weaned if bony healing or nonunion and symptomatic. If there is no healing and patient is symptomatic, electrical stimulation is considered. At 9 to 12 months, nonunion and persistent pain are indications for surgery [27].

In the athletes who remain symptomatic for greater than 6 months, have neurologic deficits, or are skeletally immature with high-grade slips, surgical treatment can be considered. The gold standard for treatment in spondylolysis is surgical fusion, with success approaching 90% [1]. Direct repair of the pars may be considered with spondylolysis without spondylolisthesis [100]. Raby and Mathews performed spinal fusion in 12 of 27 patients diagnosed with spondylolysis by clinical evaluation, SPECT, and CT. They found that those with positive SPECT scans responded well to surgery. They concluded that patients with positive bone do better with spinal fusion than those with negative SPECT scans [1]. Minimally invasive techniques have been described for treating a bilateral pars interarticularis fracture with screws by utilizing intraoperative 3D imaging. This is a novel approach as the previous options ranged from conservative care to open fusion [28]. Return of athletes to sport after surgery is very individual. Recommendations range from return to sport when patient is asymptomatic to not allowing return to collision sports at any time [2].

In general, when the patient begins to experience diminished symptoms, rehabilitation can ensue. This should focus on low impact aerobic conditioning with core stabilization and flexibility training. Tight hamstrings often coexist in this population. Athletes are progressed through more advanced stabilization techniques with addressing of their kinetic chain deficits. Activities such as loading the facet joint with high impact activities and hyperextension exercises should be avoided. Eventual progression to sports-specific exercises should be undertaken depending on the athlete's symptoms. Training deficiencies and biomechanical deficits should be addressed. There is a paucity of literature defining the timeline to return to sports. Athletes can typically be returned to sports activities without restrictions when they are no longer symptomatic. When they demonstrate full pain-free range of motion, appropriate conditioning, and sports-specific skills in a controlled environment they may return to sports. Generally, rehabilitation requires 2 to 4 months to complete, resulting in a return to sports approximately 5 to 7 months after diagnosis [2].

It is generally thought that follow-up studies in a nonsurgical patient are not necessary for older adolescents of skeletal maturity who have gone through appropriate rehabilitation and remain asymptomatic. The presence of spondylolisthesis, bilateral, or a unilateral pars fracture in a very young athlete should be followed with routine lateral radiographs to monitor for slip or slip progression every 6 to 12 months [2]. Further work-up is only necessary if symptoms fail to improve or worsen.

DISC AND ENDPLATE INJURY

Back injuries in the young athlete are estimated to occur in 10% to 15% of participants, with high variability among sports activities [29,30]. Furthermore, the overall prevalence of low back pain in adolescent athletes is 46%, compared with 18% in nonathletes [31]. However, acute disc injury in this population is not common, and clinical presentation may be different than in adults [32]. Approximately 5% of lumbar disc injury occurs in patients younger than 18 years, although disc herniation in the first decade of life is extremely rare, with only a few documented cases in the literature [33,34]. There is a genetic component. Individuals younger than 21 years, with a positive family history of lumbar disc injury, report a five times greater rate of disc herniation [35]. Cervical and thoracic disc herniations are extremely rare in this population. Furthermore, there does not appear to be a clear gender predominance. Pediatric disc herniation is more likely to be acute, related to a specific trauma or sports-related injury, as compared with adults in which the etiology is more likely related to be a chronic, degenerative process [36]. Of note, it is also important to consider atraumatic etiologies of back pain in the pediatric athlete, such as tumor, congenital, infection, and rheumatologic disease [37–39].

Pathophysiology

Sports involving flexion, rotation, and axial loading are most commonly associated with disc herniation [40–42]. These include, but are not limited to, football, wrestling, tennis, hockey, gymnastics, dance, skiing, biking, and weight lifting [36,43–46]. Similar to that seen in adults, the L4-5 and L5-S1 levels represent greater than 50% of all lumbar disc herniations in the pediatric population. Multilevel disc herniations are uncommon, and evaluation of herniated disc material often reveals preserved elasticity and water content [47]. Less often does the young athlete with an acute disc injury present with radicular pain, but rather complains of back spasms, hamstring tightness, or buttock pain [37]. This is due to the fact that most herniations are central rather than posterolateral in this population [48]. It has been suggested that children with a disc herniation tend to have more limited movement than adults with a similar herniation [36]. Discogenic pain is exacerbated when intradiscal pressure increases, such as with prolonged sitting, leaning forward, coughing, and sneezing [49]. Furthermore, on clinical examination, straight leg raise may reproduce the patient's pain because of irritation of the dura or posterior longitudinal ligament in the setting of a central disc herniation [50].

Degenerative discs as a result of repetitive microtrauma, mainly seen in the adult population, have also been studied in the pediatric population, particularly preadolescent gymnasts. The process of disc degeneration has been studied extensively by numerous researchers, including Michael A. Adams, PhD, who has used animal models to confirm that cellular changes occur in the disc following structural damage due to trauma. He states that excessive mechanical loading disrupts the disc's architecture, resulting in a cascade of cell-mediated responses that leads to further disruption, a process closely related to genetic factors, age, and loading history [51].

Goldstein et al. [44] showed an 11% incidence in the pre-elite, 43% incidence in the elite, and 63% incidence of degenerative disc changes in the Olympic gymnast irrespective of back pain or injury. Dimar et al. retrospectively identified 76 cases of juvenile degenerative disc disease (JDDD) using MRI, out of 1877 pediatric patients younger than 21 years, or 4% incidence, referred for persistent low back pain. Twenty patients (26%) were active participants in sports including gymnastics, football, soccer, golf, track and field, and skiing. A total of 41% of the JDDD cases identified had radicular symptoms on presentation, with L4–5 and L5-S1 being the most common levels affected. Furthermore, there was a high incidence of associated congenital spinal stenosis in these patients, suggesting that there may be a relationship between these two distinct pathologic processes [52]. However, although disc degeneration is found more commonly in children with low back pain compared with controls, 20% of asymptomatic children have also been found to have radiographic evidence of JDDD on MRI [53,54].

Similar to in the adult population, discogenic pain can result from internal derangement or frank herniation. Annular tears can develop from excessive annular stresses, which may coalesce to radial tears extending to the periphery of the disc [49]. Further stress on the disc may then result in a herniation of nuclear disc material through the radial tear, triggering an inflammatory response in the local tissues and exiting nerve roots [55]. Unique to the skeletally immature spine, vertical disc herniation through the vertebral endplate or apophyseal ring may occur in the setting of a substantial axial load with lumbar forward flexion [56]. This type of herniation is most likely to occur at the thoracolumbar junction [57].

Reactive, rather than idiopathic, scoliosis tends to occur more frequently in children with a disc herniation than in adults [58,59]. Also known as lumbar or sciatic shift, this represents the body's attempt to relieve pressure on the compressed, irritated nerve root, affected disc or apophyseal ring, by bending to the contralateral side, thereby opening the neural foramen and relieving the pressure. With successful treatment, the reactive scoliosis typically corrects [36].

Soft tissue imbalances also contribute to spine pathology in this population. Growth of musculotendinous tissues tends to lag behind the bony elements of the lumbosacral spine, resulting in an imbalance of stresses applied across susceptible growth areas [49]. This is consistent with reports in the literature that demonstrate children with disc herniations have grown more in height and build than their age-matched peers without herniation [60,61]. Furthermore, increased lumbar lordosis results in increased shear forces on the intervertebral disc, predisposing to injury; this can be due to tightness of the iliopsoas and thoracolumbar fascia, weak abdominal musculature, genu recurvatum, excessive femoral anteversion, and thoracic kyphosis [37].

Patterns of injury and pathology in the adolescent spine are a reflection of growth-related risk factors, including immature ossification centers and areas of growth

cartilage [37]. During periods of rapid growth, these areas become the weakest link of force transfer, and are therefore more susceptible to compression, distraction, and torsional forces [37,62]. In addition, composition of the nucleus pulposus is different in the developing intervertebral disc [63]. It is more hydrophilic than in adults, and this results in a central distribution of force transfer to adjacent vertebrae. The highly hydrophilic adolescent disc is relatively stronger than its adjacent bone structures and likely accounts for the lower incidence of discogenic injuries in the young athlete [64–66].

In the skeletally immature spine, disc herniations may also be associated with an apophyseal ring fracture. The junction of the posterior vertebral body and ring apophysis is not completely ossified in this population, making this anatomic region more susceptible to injury [49,67]. It begins to ossify at 7 to 8 years of age, begins to fuse with the vertebral body at 14 to 15 years of age, and undergoes complete physeal closure at 21 to 25 years of age [64,65]. With either acute trauma or repetitive microtrauma, disc herniation may avulse the fibrocartilaginous ring apophysis from the vertebral body posteriorly into the spinal canal. Furthermore, significant compressive axial loads during lumbar flexion may result in a posterior rim vertebral endplate avulsion fracture [37]. Compression studies on young pig spines demonstrate similar findings, with posterior edge fractures being most common, running from the endplate to the cartilaginous growth plate [68]. The most common site for this pathology is the inferior endplate of L4, and tends to occur in young athletes participating in weight lifting or sports that involve repetitive lumbar hyperflexion [48,69,70].

Clinical Presentation

Signs and symptoms of an apophyseal ring injury are similar to a central focal disc herniation, with axial back pain the most common feature [48]. Radicular symptoms due to nerve root injury are more common with an avulsion of the superior vertebral body/endplate as compared with the inferior [49]. Interestingly, contralateral (crossed) straight leg raise test is more commonly positive in patients with disc herniations associated with an apophyseal ring fracture [48].

Repetitive microtrauma to the functional spinal unit occurs with participation in multiple sports. For example, approximately 6000 N of compressive force is applied to the lumbar spine during a golf swing, rowing, and blocking maneuvers performed by football linemen [71,72]. In addition, shear stresses in the sagittal plane tend to disrupt disc annular fibers at significantly lighter loads, resulting in structural disc injury [73]. As a reference, the adult lumbar spine can withstand compressive forces between 3000 and 10,000 N before sustaining vertebral endplate fracture [74]. Furthermore, endplate failure can occur at significantly lighter compressive loads with increased repetitive movements [49]. The increase in centrally directed forces against a relatively weak vertebral endplate likely explains the higher incidence of vertical disc/endplate herniations (Schmorl's nodes) in this population [63]. Swärd et al. [75] studied the adolescent male gymnast population, and reported that 71% had evidence of Schmorl's nodes on MRI, compared with 44% of control subjects, and 17% had evidence of apophyseal ring injury, compared with none of the controls. A study of 120 young elite skiers, younger than 17 years of age, demonstrated a significantly higher rate of anterior endplate lesions in this population compared with the control group. On average, the elite skiers had 3.9 endplate lesions per person, compared with 1.9 lesions in the control subjects, with the majority located in the lower thoracic and upper lumbar spine [76].

Diagnostic Evaluation

Evaluation of the young athlete with back pain requires synthesizing information from the history, physical examination, and radiologic studies. As no single physical examination provocative maneuver is pathognomonic for lumbar disc herniation [77], it is important to correlate all information obtained from the history and physical with the imaging findings when formulating a diagnosis [49]. A comprehensive musculoskeletal and neurologic examination should be performed, consisting of palpation along the spinous processes to identify pain and/or instability, assessment of lumbopelvic motion, specific provocative maneuvers, and evaluation of gait and balance, lower extremity alignment, and components of the relevant kinetic chain of motion for the athlete [63].

Lumbar spine radiographs may demonstrate narrowing of the intervertebral space in 50% to 70% of children with herniated discs, but it is difficult to establish the diagnosis with radiographs alone [36,78]. Radiographs are also important for ruling out other lumbar spine pathology on the differential, including spondylolysis, spondylolisthesis, and fracture. MRI is typically indicated to complement the radiographic images when disc herniation is suspected (Figure 19.7), given its high resolution of soft tissues and neural elements [36]. With apophyseal ring involvement, plain films may reveal a small avulsion fracture from the vertebral body [49]. Similarly, vertebral endplate fractures are typically identified on standing posterior-anterior and lateral lumbar spine radiographs, with flexion-extension views identifying spinal instability when present [56]. CT scan is recommended in these instances to better define the extent of osseous injury, as MRI may not differentiate between bone and disc material [48,49] (Figure 19.8). Given that children often provide a less detailed description of their pain compared with adults, compounded by the overall low incidence of disc herniation in this population, time to diagnosis is often delayed [36]. Mean duration of symptoms in children prior to diagnosis is 10 months, compared with 4.7 months in the adult population [79,80].

Therapeutic Options

The average age at which young athletes seek medical treatment for low back pain is 15 to 16 years [81,82]. The initial treatment for a suspected discogenic injury is conservative therapy with rest and analgesics, progressing to physical therapy addressing core stabilization and neuromuscular control exercises [49,63]. However, this does not apply to patients who present with significant neurologic

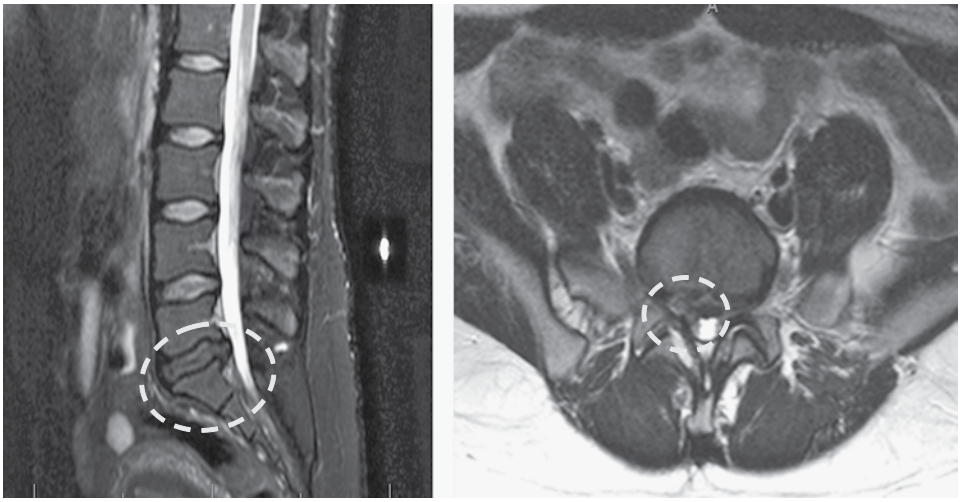

Figure 19.7 Lumbar disc herniation—sagittal and axial images.

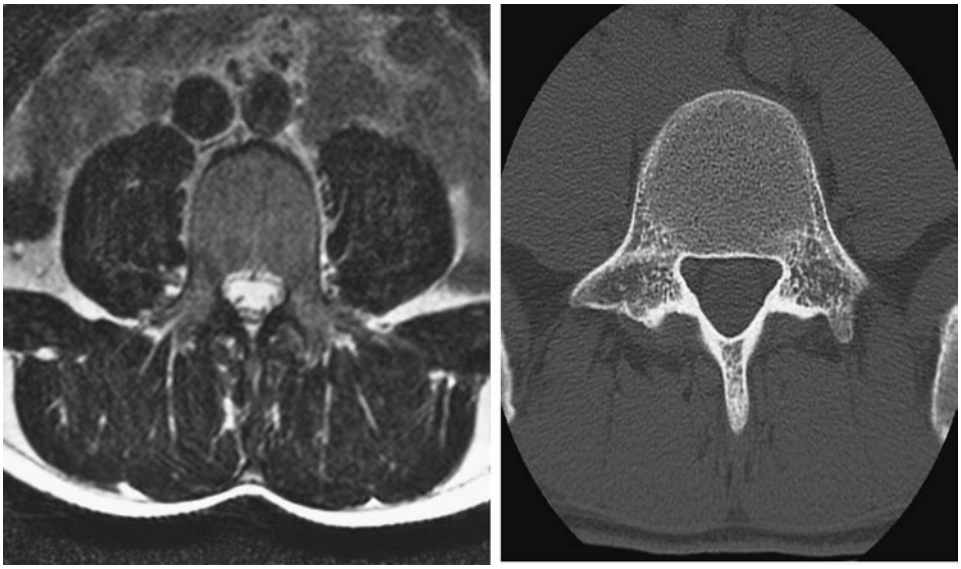

Figure 19.8 MRI (left) versus CT (right) of the lumbar spine.

deficits, including bowel and bladder dysfunction; in these instances, which are fortunately rare in the pediatric population, urgent surgical decompression of the neural structures is indicated without a course of conservative therapy [36]. There are several factors that need to be considered in caring for the young athlete with a low back injury. First, the physical training regimen prescribed should not interfere with the athlete's physiologic growth and development. Second, the psychosocial environment of an injured athlete may pose a challenge for treatment, and ultimately hinder complete recovery. In addition, care must be taken when prescribing medications, to ensure proper dosing (may be different than adult dose), and to be aware of substance use policies that may apply to the athlete's sport and level of competition [63]. Moreover, with the unfortunate rise of performance-enhancing supplement use in this population, there is an increased risk of significant interactions with prescribed medications [83].

Nonsteroidal anti-inflammatory medications may be used for analgesia and to decrease the inflammatory reaction that begins with disc herniation. For instances, where pain is not well controlled or sleep is interrupted on nonsteroidal anti-inflammatory medications alone, opioid medications may be added cautiously as well [49]. Should the patient fail to improve using these modalities, epidural steroid injections may be indicated to place a corticosteroid and local anesthetic mixture directly at the site of herniation and inflammation [55,79,84]. Following this algorithm, less than 0.5% of adolescent athletes require surgical disc decompression for persistent symptoms [56]. However, there are reports that conservative, nonoperative treatment of lumbar disc herniation is less effective in children than in adults [79,85]. This may be due to the highly viscous, nondegenerative nature of herniated discs in the pediatric population, resulting in less resorption than with degenerative discs [36,86]. The same treatment algorithm applies when an apophyseal ring avulsion fracture is identified as well [49]. In these cases, discectomy with excision of the avulsed bone fragment is indicated for persistent or progressive symptoms [37,48].

Return-to-play guidelines for the young athlete with a disc herniation are lacking, and little has been formally studied on this issue [63]. It is important that the treating physician provide adequate education to the patient and their family on the potential risk of further injury and/or pain. Without a significant neurologic deficit, it is generally accepted that continued participation in sports will not result in serious injury. However, it is logical that the same excessive mechanical stresses that caused the disc herniation from sport participation could cause additional herniation and/or flare of pain symptoms with return to that sport. Many physicians, as documented in the literature and from clinical experience, advocate limiting sport participation during the acute treatment period, and gradually advancing activities as tolerated [36]. In the case of vertebral fractures, range-of-motion and strengthening exercises along with return to noncontact sports can begin when the young athlete is pain-free; full return may be possible when complete fracture healing has occurred, as evidenced on imaging, which is typically 6 to 8 weeks postfracture [56]. Rehabilitation should progress to a sport-specific training program prior to the athlete returning to play, similar to a work hardening program, to best optimize performance and theoretically minimize the risk of future injury [63].

PARASPINAL COMPARTMENT SYNDROME

Acute compartment syndrome generally describes a condition of increased pressure within a confined fascial space, compromising perfusion to the enclosed tissues and resulting in ischemia, eventual necrosis, and impaired function [87]. Although this syndrome most commonly occurs in the leg after significant exertion or trauma, it has also been documented to rarely occur in the lumbar paraspinal compartment [88–91]. It was first described as a possible etiology for lumbago by Peck in 1981 though the true incidence and prevalence remains unclear [92]. Cadaveric studies demonstrate a well-defined paraspinal compartment, with the erector spinae muscle complex bounded by thoracolumbar fascia anteriorly, posteriorly, and laterally, and by the spinous processes, interspinous ligaments, and attachments of the thoracolumbar fascia medially [89,93]. Normal physiologic pressure within this compartment is 3.1 to 10.8 mm Hg, and muscle fatigue with exercise may occur at pressures greater than 14 mm Hg [89,94,95]. Studies suggest that lumbar paraspinal compartment pressure is highest in the "skiing stance," standing with knees flexed [88].

Clinical Scenario

Acute lumbar paraspinal compartment syndrome appears to be less common than chronic, and of the few cases reported, none were due to acute trauma, but rather repetitive intense muscle activity [88,90,91]. Minnema et al. analyzed data presented in several cases of acute paraspinal compartment syndrome. All documented cases occurred in males, 24 to 35 years of age, who presented with severe low back pain and had physical examination findings consisting of loss of lumbar lordosis, pain exacerbated by straight leg raise, and sensory loss over the affected muscles. Laboratory findings demonstrated significantly increased serum creatinine phosphokinase and aspartate aminotransferase values. Creatinine phosphokinase values reported in the literature for lumbar paraspinal compartment syndrome range from approximately 5000 to 70,000 [90]. In most cases described, there was also focal paraspinal muscle rigidity and edema, with point tenderness over this area [88,91,92]. Diagnosis is confirmed using MRI data and intracompartmental pressure measurements using a pressure transducer. MRI scans may show increased signal in the paraspinal musculature on T2-weighted images, representing edema, with a resultant asymmetric increase in muscle size within the compartment [88]. MRI signal abnormalities will occur on both T1- and T2-weighted images in cases of hemorrhagic myonecrosis [92]. Intracompartmental pressures in these patients that are documented in the literature range from 14 to 108 mm Hg [91]. There are also a few reports documenting the utility of technetium Tc^{99m} bone scan imaging to assess the extent and location of muscle damage [96–98].

Treatment

Similar to the standard treatment of rhabdomyolysis, intravenous hydration should be initiated to minimize myoglobinuria [91,92]. Pain control is typically managed with opioids, and upright activity is restricted (bed rest) until symptoms start to dissipate [97]. From the limited cases reported in the literature, there is a debate as to whether surgical or conservative treatment of lumbar paraspinal compartment syndrome is the standard. Styf and Lysell showed that fasciotomy normalized the intramuscular pressure and relieved the pain in a patient who had a chronic lumbar paravertebral compartment syndrome [99]. In acute cases, there is a report of a patient who recovered well at 4 months having received only conservative therapy [92], whereas other authors favor fasciotomy in this population to facilitate a quicker and more complete recovery [88,90,91]. In the patient treated conservatively, he returned to regular activity at 6 weeks and sport at 4 months [92]. In the patients treated with fasciotomy, they returned to sport at 1 to 2 months symptom free [88,90].

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20 An Algorithmic Approach to Lumbosacral Radiculopathy

Philip Tasca

INTRODUCTION

Low back pain and associated symptoms commonly lead the patient population in the United States to seek medical evaluation. Low back pain lasting at least 2 weeks has a lifetime prevalence of 13.8% in this country, with 1.5% of these episodes also associated with symptoms of lumbar radiculopathy [1,2]. In a comprehensive analysis of the general state of health of Finnish adults, sciatica was diagnosed in 5.1% of those evaluated [3]. In response to the seeming ubiquity of low back pain and related disorders, a variety of treatment options have evolved, from short courses of over-the-counter oral medications and physical therapy to interventional treatments to surgical correction. It is logical that, in any given case, efficient utilization of this arsenal would follow the localization of the site of clinical pathology, the pain generator. Sensitive diagnostic techniques, able to correlate the most subtle findings with the correct tissue source of symptomatology, could aid in this efficiency. On the other hand, specific localization of the tissue injury would potentially allow for the most specific treatment, whereas exclusion of structures not causing pain would minimize treatments that would be less helpful.

Here lies the challenge to the interventional spine practitioner: the differentiation and localization of lumbosacral radiculopathy from a myriad of other potentially similar conditions. The anatomic site of pathophysiology in radiculopathy is, generally speaking, the spinal nerve root and associated dorsal root ganglion. Considering that there are 12 spinal nerve roots from L1 to S1, but also 20 facet joints, five intervertebral discs, multiple pelvic and appendicular joints, and an even larger number of low back, pelvic, and lower limb soft tissue structures—all potential pain generators—it is easy to see that the spine specialist's task of localizing the source of a patient's signs and symptoms can be quite challenging.

Optimizing the success of diagnosis-specific treatment may help not only the specific patient being treated but also society in general by properly directing healthcare expenditures in an evidence and/or knowledge-based spine medicine approach. In the literature, meta-analyses have evaluated outcomes of various interventional spine procedures. One must consider that some of these analyzed studies include patient subjects triaged to potentially suboptimal interventions due to inaccurate diagnostic appraisal. It is certainly plausible that employment of rigorous diagnostic examinations will improve triage of patients, achieving globally improved outcomes.

HISTORICAL FEATURES

The nerve root and dorsal root ganglion are the primary sites of pathology in lumbosacral radiculopathy. In one animal study, chronic compression of the dorsal root ganglion was found to be associated with prolonged, spontaneous neural discharges postulated to be the physiologic substrate of neuropathic radicular pain [4]. Given this theoretical pain generator localization, a variety of potential symptoms may be deduced. Many of these potential clinical-pathologic correlations have been observed empirically.

Symptom Location

Nerve pathology is well known to be painful, and the distribution of this pain is often along the sensory—or dermatomal—distribution of the nerve in question. Lumbosacral nerve root sensory maps have been constructed. A common dermatomal mapping scheme is that constructed by Keegan [5,6] (Figure 20.1). Lower limb radicular pain may not always follow discrete patterns in a contiguous fashion [7].

Based on the aforementioned characteristic of nerve-based, or neuropathic, pain to localize to the sensory distribution of the nerve in question, it is, theoretically, simply a matter of understanding the symptom distribution of each nerve root to anticipate the area of pain that would be associated with pathology at each nerve root level. However, multiple empirically observed caveats limit the predicted specificity of such a postulate. First is the fact that given a specific nerve root distribution, pain may be felt throughout the entirety of the distribution or only in certain locations within [7]. For example, we classically think of S1 radiculopathy as, according to the dermatomal scheme of Keegan (see Figure 20.1), associated with pain from the posterior buttock to the posterior thigh, posterior or lateral calf, and lateral foot. However, certain areas within this overall distribution may be nonpainful. Pain may exclude all areas below the knee, for

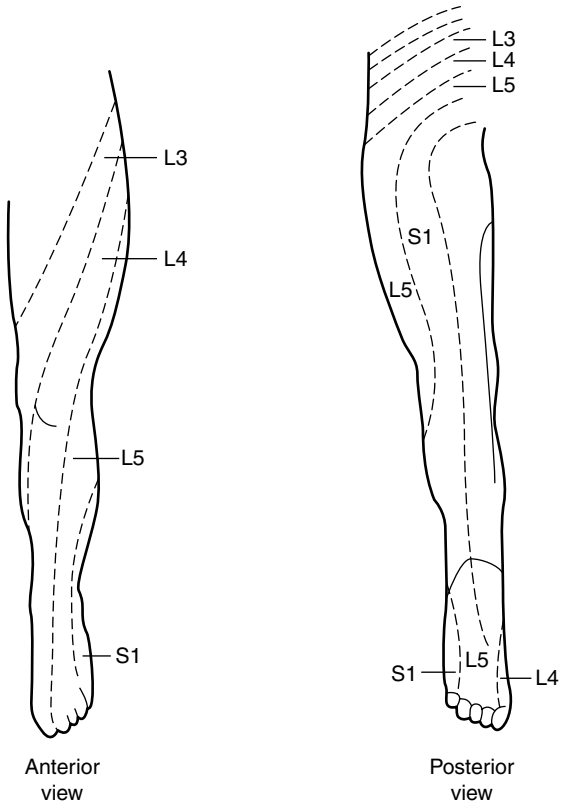

Figure 20.1 Dermatomal map.

instance. Or, multiple, noncontiguous areas may be painful, such as the posterior thigh and lateral foot, with the intervening calf nonpainful. Observed also has been the possibility of changes of location of pain within a given dermatome with position, activity, or simply over time. This latter observation, with respect to time, is postulated by van den Hoogen et al., in their literature review, to potentially partially account for the lack of any single test to have a high sensitivity and specificity in the diagnosis of radiculopathy [8]. According to Keegan's (see Figure 20.1) scheme, the L5 dermatomal distribution includes the lateral thigh, pretibial region, and dorsal foot. The L4 distribution includes the anterior thigh, medial leg below the knee, and medial foot. The same variations in clinical findings may affect these distributions as in our S1 example mentioned earlier.

A second, more predictable limit to specificity of correlating level of nerve root injury to pain location is the fact that there is substantial dermatomal overlap between adjacent nerve roots. A given radicular dermatome, which generally speaking extends as a thin patch of skin from the low back into the lower limb in a ribbon-shaped distribution, shares roughly 50% of its innervation on either side with the adjacent nerve root (Figure 20.1). Thus, any given patch of skin is typically innervated by two nerve roots. Even a whole strip of skin, from low back to distal lower limb, following a well-described dermatomal path, by definition, exists within the distributions of two nerve roots. Therefore, even in the case of classic "sciatica," pain distribution alone does not completely localize the pain generator. Limitations in the clinical usefulness of pain location have been noted in the literature, as in one comparison between pain location as a historical feature with the presence of a positive straight leg raise (SLR) (discussed later) on examination. Haldeman et al. found no correlation between lower limb pain or numbness location and the presence of a positive SLR [9]. Other studies have explored variations in dermatomal distribution corresponding to transitional lumbosacral anatomy—anatomic configurations where the fifth lumbar vertebra is sacralized, or the first sacral segment is lumbarized. Seyfert discovered dermatomal variation in a significant number of patients with a transitional lumbosacral or thoracolumbar anatomy: the "dermatomal gap" usually present between areas of innervation of L1, L2, and L3; and S2 and S3 was positioned atypically [10]. Following this study, Kim et al., using selective nerve root blocks (SNRBs) with electrical stimulation, found that in patients with a lumbarized S1 segment, the distribution of motor and sensory symptoms in the "L6" distribution corresponded to the typical S1 distribution of nontransitional anatomy. Further, in the setting of a sacralized L5 vertebral body, the L4 root supplies the typical distribution on the L5 root [11].

Symptoms—Nonradicular Pain

Further along these lines is the limited specificity of pain location due to factors other than dual innervation. Recalling the aforementioned list of potential low back and lower limb pain generators, one can see that there are a variety of nonradicular causes of pain potentially experienced in any given location in the low back and lower limb. For example, it has been suggested that sacroiliac joint-based pain may be referred into the groin. These painful distributions may resemble partial dermatomal distributions (here, L3), further diminishing the specificity of pain location in localizing the pain generator. As an aid to the diagnosis of radicular versus nonradicular pain, the work of Udén and Landin may be considered. Pain diagrams of 81 patients with radicular pain due to a corroborative disc herniation were analyzed as were subsequent myelograms. Of nine patients with groin pain, only one was found to have a disc herniation. All patients with disc herniation had pain below the knee [12], a finding in alignment with subsequent observations by Haldeman et al., in which pain below the knee had a 40% chance of being diagnosed as radiculopathy on subsequent physical examination or electrodiagnostic testing. The likelihood of pain below the knee was further noted to correlate with the size of anatomic pathology on subsequent computed tomography [9]. Mann et al.'s subsequent computerized analysis of pain drawings showed this historical modality to be 51.7% accurate in the detection of herniated disc, and 32.2% accurate in the detection of spinal stenosis. The authors' positive conclusions regarding these drawings, at least in helping shape an "initial impression," echoes previous reports [13–17] in that pain referred below the knee should raise the clinical suspicion of radicular pain.

Symptoms—Pain Quality

The anatomic relationship between the nerve root and distal sensory nerves and skin provides us a framework for understanding potential location of radicular pain. Similarly, the fact that the nerve root is composed of neural tissue allows us to theorize that lumbosacral radicular pain will have the qualitative characteristics of neuropathic, as opposed to nociceptive, pain. Empirically, this is commonly the case, with radicular pain often described as "lancinating," "sharp," or "electric" in feeling. However, these descriptions are not fully specific, as "dull," "aching," and "sore" may also be descriptors. Because other potential pain generators, although expected to be associated with nociceptive pain, may be described in ways that mimic neuropathic pain (e.g., knee-based pain described as "shooting" down the pretibial region, even with tingling noted), specificity is further limited. A diversity of patient descriptors in low back pain and sciatica has been noted in the literature [18]. As noted by Udén, pain character as communicated via pain diagrams was not reliably associated with subsequent diagnosis of disc herniation except that the combination of "numbness" and "pins and needles" was more common in those without a herniated disc [12].

Symptoms—Positionality and Walking

Another common feature of nerve root-based pain is its often positional nature: certain conformations of the lumbosacral spine, such as lumbosacral extension or postures that put tension on the nerve root, may exacerbate or cause symptoms. One typical activity that is well known to exacerbate radicular pain via lumbar extension is walking. Because lower limb pain with walking has been classically associated with claudication, this nonvascular connection between walking and lower limb pain is known as pseudoclaudication. In one study, pseudoclaudication was deemed the most common symptom of lumbar central stenosis, occurring in 94% of myelographically and surgically confirmed cases. Lower limb symptoms included pain, numbness, and weakness, and were bilateral 68% of the time [19]. Findings of decreased walking tolerance in terms of both distance and time, due to claudicatory symptoms, may be present [20]. More specifically, pain with walking up to 0.5 km has been reported to occur in 66% of patients with central stenosis, with which pseudoclaudication is classically associated, and in 26% of patients with lateral stenosis [21]. The anatomic cause for this finding is generally thought to be narrowing of the central canal with extension, and expansion of the canal with lumbar flexion—both changes relative to neutral posture. In this paradigm, walking is thought to be associated with neutrality or extension. Lumbar flexion, classically as with walking while pushing a shopping cart, is commonly thought to be associated with decreased radicular pain in central stenosis. Such anatomic changes have been well studied for decades and observed in cadaveric [22–24] as well as clinical studies [25]. Similarly, changes in foraminal dimensions have been shown to correlate with nerve root compression [24,26]. Lumbar flexion, as well as other movements, including extension, has been shown to sometimes increase incidence of nerve root abutment [27], however. This limits the sensitivity of historical findings in foraminal stenosis, because in two different patients the same activity can increase foraminal nerve root encroachment in one, and decrease it in the other. This also perhaps helps explain the absence of a commonly held connection between standing and walking and foraminal (as opposed to central) stenosis. The fact that fixed—at least in the short term—geometric properties of the lumbar spine lead to exacerbation of radicular symptoms helps explain the test-retest reliability of the exercise treadmill examination, especially in lumbar central stenosis [28].

Other typical sensory characteristics of neuropathic pathology are, predictably, often present. These include numbness and tingling in a dermatomal distribution, with the same caveats noted earlier. Similarly located allodynia, the subjective sense of pain with nonpainful stimulation, and hyperpathia, a heightened sense of pain associated with typically painful stimulation, may be present. In a prospective study of 300 patients with radiographically and surgically confirmed lumbar nerve root compression syndromes, Jőnsson and Strőmqvist reported a history of sensory disturbances in 60% of patients with herniated discs and 48% of patients with lateral stenosis [21]. A recent study of 65 surgical cases diagnosed as having foraminal stenosis reported 100% prevalence of back and leg pain and 45% prevalence of paresthesias. Also noted was a 31% prevalence of the motor counterpart to these sensory findings—subjective weakness [29].

Signs—Weakness

The complement to the sensory function subserved by the lumbosacral nerve root is the motor component. Neural signals, originating in the brain and causing volitional muscle contraction in the lower limbs, are transmitted through the nerve roots. Root pathology is therefore a possible cause of impairment in ability to contract, or maintain contraction of, target muscles in the lower limb. As the patient relates the history of his or her present illness, or perhaps during the review of systems, weakness may be noted. A subjective sense of weakness in and of itself is nonspecific. Often, pain, discomfort, or other sensory neurologic symptoms are experienced or described as "weakness." Such a potential misnomer is more likely to exist in the case of weakness noted by the patient to be present in the entire limb, with subsequent questioning revealing lack of any focal deficits, full functional ability, and subsequent physical examination without findings of weakness. Specifically, review of systems may check not only for a subjective sense of weakness but also focal deficits, including knee buckling, foot slapping, toe catching, and foot dropping. Functional deficits may be inquired about, including tripping, falling, and any specific deficits with activities of daily living, vocational activities, and avocational activities, including sports.

PHYSICAL EXAMINATION FINDINGS

Given that radiculopathy by definition is pathology of the nerve root and associated neural structures, neurologic examination is helpful toward diagnosis. Given that the mechanism of this pathology commonly stems from mechanical impingement, or inflammation often related to mechanical derangement, orthopedic evaluation forms the second pillar of the physical examination.

The Neurologic Sensory Examination

Sensory examination involves the areas subserved by the lumbosacral dermatomes. Optimally, this examination can localize the level of lesion to the nerve root, excluding pathology at the level of the peripheral nerve or specific nerve fiber type. Each radicular dermatome is tested, in multiple locations corresponding to different peripheral nerves within the dermatome. Abnormalities spanning multiple peripheral nerve territories but all within one radicular dermatome are more specific for radiculopathy than a single abnormal area. In the latter case, a solitary area of abnormality—as an isolated examination component—could be referable to the nerve root or the peripheral nerve innervating the area. Thus, specificity is diminished.

To exclude a more general neuropathology affecting a certain fiber type, multiple sensory modalities may be tested in each area. These may include light touch, pin prick, and vibratory sense. Abnormalities may include hypesthesia, paresthesia, hyperpathia, and allodynia. Weise et al. noted an ability to localize the correct level of nerve root pathology with 88% accuracy utilizing the pressure and vibratory thresholds at points within the symptomatic radicular pain pattern, but not light touch or pinprick testing [6].

The sensory examination requires patient perception, cognitive processing, and verbal feedback to the examiner, thus representing a relatively large neurologic feedback loop. Any disruption to or modification of this circuit at a neurologic level rostral to the nerve root could distort the outcome of the examination from the examiner's viewpoint. The sensitivity and specificity or the sensory examination are limited. This may help account for the high reported variability, 21% to 84%, in reported sensory disturbances in patients with herniated lumbar discs [4,30,31]. Further limiting specificity is the fact, noted earlier, that any particular area of skin is dually innervated by adjacent nerve roots. Once the lesion has been localized the level of the nerve root, interdermatomal specificity remains limited. Extradermatomal sensory findings may be found that are not due to multiroot pathology, but of unknown cause. Bogduk has categorized various nerve root anastomotic patterns, in which nerve roots or constituent fibers may emerge from an atypical foramen (i.e., a foramen above or below the expected location), travel an unusual course within or near the foramen, or simply be supernumerary [32]. In such cases, pathology of known location relative to the vertebrae could produce radicular symptoms outside the expected dermatomal distribution based on atypical root anatomy (e.g., a lesion in the L3-4 foramen causing symptoms referable to the L5 root that is, contrary to the usual path of this nerve, exiting this foramen due to an anastomosis). Falconer noted "spreading hypoalgesia," in which this finding was present outside of the confines of the one or two root distributions theoretically affected by the solitary disc protrusion found at operation [23].

The Neurologic Motor Examination

This dual innervation has a second ramification to the physical examination: most muscles are innervated by two adjacent nerve roots, limiting specificity of lesion localization if weakness is detected. A useful corollary is the fact that muscles innervated by adjacent but overlapping pairs of nerve roots, say the vastus medialis (L3 and L4) and the tibialis anterior (L4 and L5), if both weak, point to the single overlapping root level (in our example, L4) as the level of pathology. Such a deduction is strengthened if muscles more exclusively innervated by root levels above and below the suspected pathologic level (in our example, the iliopsoas—L3; and the peroneus longus—L5) are not weak.

Again, caveats regarding specificity exist, and one contributing factor is variability in specific nerve root contributions to motor strength of particular muscles. The aforementioned localization scheme is based on an assumption of only one pathologic root level. Multilevel root pathology could exist and yield the same examination findings, if there were different relative contributions of the nerve roots to the muscles examined. In the example mentioned earlier, if the iliopsoas were more predominantly innervated via the L2 root, it would be possible that root pathology existed at L4 and also L3. In a study of 100 patients with diagnosed radicular symptoms, Kerr found weakness in various lower limb muscles, but myelographic findings did not always align: for instance, hip flexion strength was reduced in 10% of patients—six patients demonstrated protrusion of the L4-5 intervertebral disc and four patients with less correlative L5-S1 protrusions on myelography [33]. Notable is the fact that 4% of patients with symptoms of radiculopathy but not protrusion on myelography showed similar weakness—hinting at decreased sensitivity of myelography compared with motor examination.

Another factor limiting sensitivity is the potential for extraneous causes of detected weakness. A stiff joint or decreased volitional contraction due to pain are two not uncommon examples. Malingering, for instance in the case of a patient eager to demonstrate a neurologic deficit, could lead to decreased volitional contraction masquerading as weakness. Yet, nonpathologic weakness is typically different than physiologic weakness. The former is "ratchety" (gegenhalten) or give-way and not smooth and gradual as in the later.

Sensitivity is an issue as well, especially regarding manual muscle testing of the lower limb. It must be remembered that manual muscles testing is but one modality within the physical examination used to test

strength. A six-point scale, from 0 to 5, is commonly used and has been described previously [34]. Specifically, the ankle and thigh musculature could, even if reduced in strength, be strong enough to overpower the examiner's hand and arm strength—the examiner's hands and arms being, by definition, the tools of manual muscle strength testing. In these instances, it may be useful to place the examined muscle in a position of mechanical disadvantage and to perform repetitive assessments (e.g., repeated calf raising) to detect subtle weakness or an endurance deficit.

Muscle Stretch Reflexes

Lower limb-targeted motor signals that do not originate in the brain, but instead originate from the spinal cord in response to proprioceptive stimulation from the limb, are muscle stretch reflexes. Both the afferent and efferent arc pass through the nerve root at the same level, so pathology at the root has in effect two avenues by which to disrupt the reflex arc. In a prospective study of 300 patients, Jőnsson and Strőmqvist found patellar and ankle reflex reductions at a frequency of 42% and 65%, respectively, in patients with central stenosis. With lateral stenosis, patellar and ankle reflex reductions occurred in 13% and 37% of patients, respectively [21]. The specificity of muscle stretch reflex elicitation with respect to putative nerve root involvement is limited, as shown by Kerr's findings in 48 patients with symptoms of radiculopathy, altered ankle reflex, and myelographic findings of disc protrusion: 41 of these patients showed L5-S1 findings on myelography, whereas 7 showed L4–5 findings [33]. Possibly accounting for such decreased specificity is the fact that the ankle reflex is mediated by the S1 nerve root, which passes by all lumbar discs, including L5-S1, and L4–5. The findings of Peyton, that 11 patients with diminished quadriceps reflexes were found to have disc lesions at L5-S1 [35], are less explicable, but only if anatomic impingement is the sole pathology considered. If other pathology, such as inflammatory insult to the nerve root, is allowed for, then Peyton's findings may be explained. It is this inconsistency, noted relatively early in the literature, that points us toward the limitations in later studies that rely on surgical findings as a gold standard, and also hints at the potential utility of other means of testing for radicular pain, means unrelated to anatomic impingement.

The Orthopedic Examination

The presence of neurologic findings on examination is not completely specific: in one study, only 75% to 80% of patients with such findings were found to have positive findings at operation. Conversely, it has been demonstrated that 20% to 30% of patients with herniated discs have no neurologic abnormalities [36,37]. Orthopedic examination of the lumbosacral spine and, indirectly, the nerve roots thus forms the second main element of examination. Inspection of the spine for deformity, including scoliosis, is an initial step. Palpation may further elicit deformity, as well as discover areas of tenderness, a finding associated with 71% of cases of foraminal stenosis in one surgical study [30]. In a study of 100 patients with symptoms of radiculopathy with correlative myelographic findings, Kerr found that 63% demonstrated scoliosis on physical examination [33]. Lumbar range of motion may serve to demonstrate instability, muscle tone with its secondary effect on spine conformation, and pain reproduction. In Jőnsson and Strőmqvist's study, lumbar hyperextension was noted to cause radiating lower limb pain in 43% of patients with lateral stenosis, and 51% of patients with central stenosis [21]. In a subsequent study of 105 patients, lumbar flexion and side-bending range of motion, as well as pain distribution related to lumbar flexion while standing, were found to be of value, correlating with axial imaging findings of herniated nucleus pulposus [38]. These findings are consistent with earlier myelographic and surgical observations that many disc prolapses may become more prominent with lumbar extension [39]. Such dynamic radiographic findings continue to be observed in the setting of axial imaging [40]. Further surgical analysis has been corroborative as well [41].

The SLR Test

A relatively well-studied physical examination finding relates to the association of tension on an injured or otherwise pain-producing nerve root with reproduction or increase in lower limb pain. The classic such test is the Lasègue test (Figure 20.2), whose namesake published his observations on sciatica in 1864, and whose student Forst depicted one version of the test in his doctoral dissertation in 1881 [42,43]. Details of the test vary in the literature [42–44], but the common element includes passive hip flexion in a supine patient, with the knee in full extension. The "well leg" or crossed SLR test, first described in 1901 by Fajersztajn, is a variant in which passive hip flexion in the unaffected (i.e., "well") lower limb causes pain in the

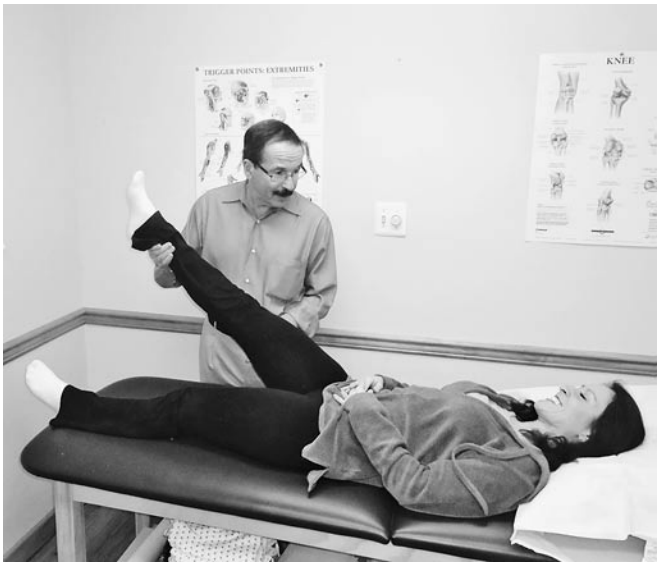

Figure 20.2 The straight leg raise.

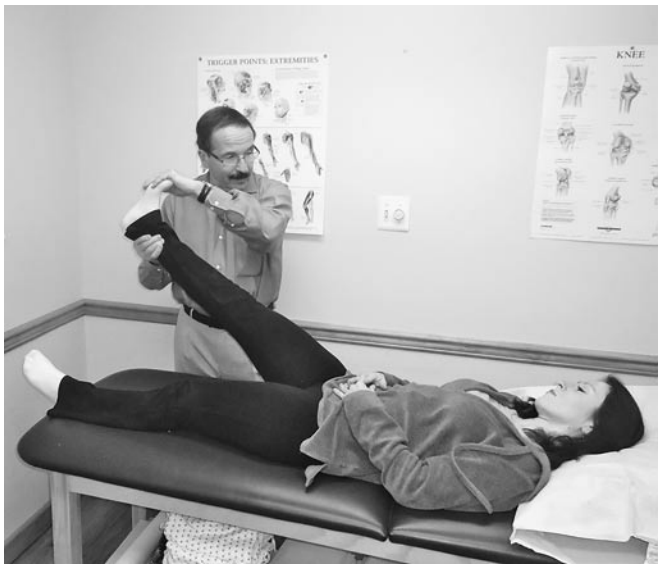

Figure 20.3 Braggard's maneuver.

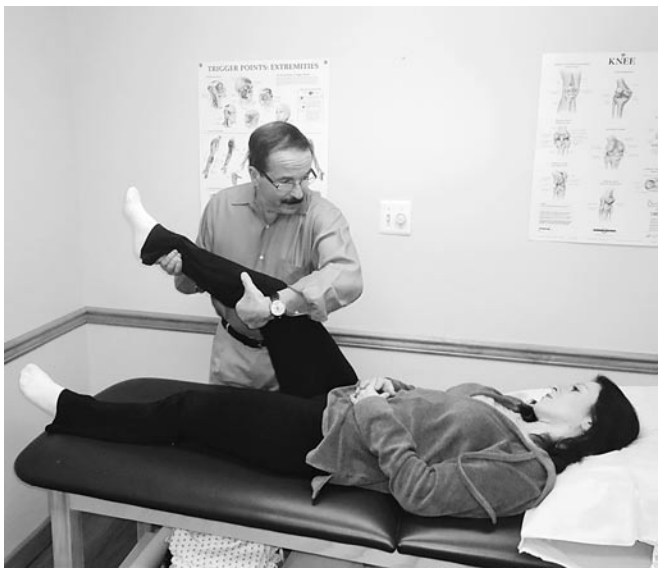

Figure 20.4 The tibial nerve tension test.

contralateral, affected limb. Almost 80 years later, this version of root tension testing was concluded to be associated with lumbar disc herniation in 97% of patients, with such specificity that herniation was noted at surgery in 90% of patients with this finding but negative myelography [45]. Subsequent to this, Kosteljanetz found that of 20 patients with unilateral sciatica, 19 of those with a positive crossed SLR were found to have a disc prolapse at surgery [46].

Variations on the theme abound. Fajersztajn elaborated the SLR test, noting pain elicitation with passive ankle dorsiflexion when the patient is positioned so that the SLR test has just become positive (Figure 20.3). A neck flexion component was noted as well [43,47]. Another variant includes inducement of a positive SLR (ipsilateral or crossed), with subsequent slight lowering of the lower limb, followed by knee flexion of 20°, and then pressure on the posterior tibial nerve in the popliteal fossa. This is known as the posterior tibial nerve sign, described by Cram in [48] (Figure 20.4).

Other neural tension tests continue to be described, from the relatively well-studied slump test [49] to the knee flexion test [50]. The slump test combines, in the sitting patient, knee extension and foot dorsiflexion, but also trunkal flexion (i.e., a "slumping posture") and cervical flexion [49]. In one study of 75 patients, Majlesi et al. found that this test is more sensitive, but slightly less specific, than the SLR test [51]. High interrater reliability has also been observed in this test [52]. A reversal of the typical SLR test is the femoral nerve stretch test (Figure 20.5). In his 1989 analysis, Chistodoulides found that this test, in which the prone patient's knee is passively flexed with the hip at neutral, is pathognomonic of a lateral L4–5 disc protrusion [53]. In a subsequent surgical series, Kunogi and Hasue noted correlation between femoral nerve stretch test and L4 root involvement [41].

Although such clinical variations continue to proliferate, the unifying anatomic mechanisms of such tests have also been studied [54–56]. In one cadaveric study, Goddard and Reid called attention to the mobility of neural structures in the neural foramen and pelvis during lower limb motion. Discovered were multiple fibrous adhesions causing the root within the foramen to be a relatively fixed structure, with the amount of these adhesions being proportional to subject age and observed pathoanatomic signs of "wear and tear." Increased amounts of adhesions were associated with decreased neural mobility. It was concluded that in subjects with such decreased mobility, distal motion would produce proportionally increased nerve pressure over fixed proximal anatomic structures, including the neural foramina, sacral ala, and sciatic notch. Also induced was increased proximal neural tension [56]. Roughly three decades later, the concept of linear spinal nerve motion and strain continues to be the conclusion of cadaveric studies [57]. This picture complements the earlier work of Sunderland and Bradley, in which it was concluded that under tension, the elastic properties of proximal neural structures were such that lumbar nerve roots are more vulnerable to deformation than are nerve trunks [58]. Based on physical principles of stress and strain, this finding is consistent with Haldeman et al.'s observation that leg pain with straight leg raising at less than or equal to 60° is more likely to be associated with radiculopathy than pain experienced at greater angles. The kinematic correlate of the slump test has also been studied in situ, with baboon cadaveric analysis demonstrating cephalad translation of the lumbar cord with passive cervical flexion [59].

The SLR test has been studied both generally and in more detail. One more general evaluation of the test observed that specificity was such that of 49 patients with positive SLR, 43 had a prolapsed disc at surgery [46]. In a more detailed analysis of SLR parameters involving 50 patients, the central versus lateral location of disc protrusion was correlated with pain location on straight leg and

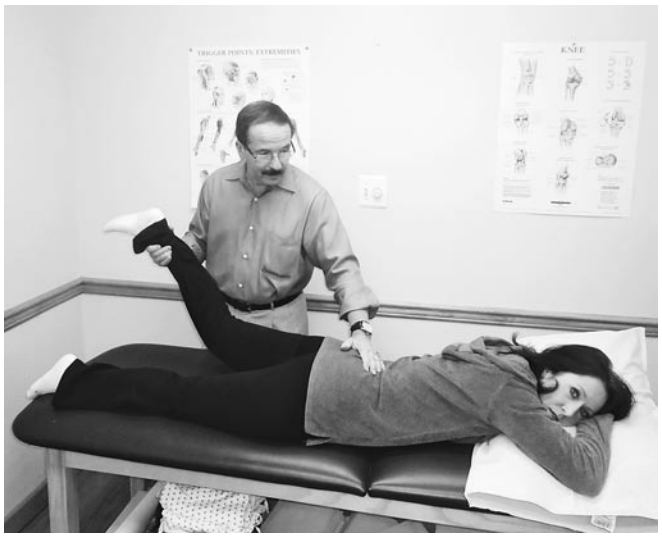

Figure 20.5 The reverse straight leg raise.

crossed straight leg raising. Protrusions were surgically categorized as central, lateral, and intermediate. The dura alone, nerve root sheath alone, or both, are contacted by disc material, respectively. Pain location with straight leg raise between 30° and 70° was found to be correlative with this lesion location scheme: pure leg pain correlated with lateral protrusion, pure back pain with central protrusion, and mixed leg and back pain with intermediate protrusion. This was observed for both (ipsilateral) SLR and crossed SLR testing. In the ipsilateral SLR, pain location was found to predict protrusion location in this manner 80% of the time. This exceeded the myelography's predictive ability of 79%. In contrast, actual nerve root level of involvement could be predicted by pain location 50% of the time [44]. The ability of straight leg raise-induced pain location, as just described, to predict central, intermediate, or lateral disc protrusion was verified by Shiqing et al., with a demonstrated accuracy of 88.5%.

Furthermore, the angle at which the test became positive was found to correlate to the size of disc protrusion found at surgery [60] and presence of inflammatory mediators at the nerve root-disc interface. These findings echo Troup's earlier conclusion that the probability that a disc herniation is causing radicular symptoms is inversely proportional to the angle at which the SLR become positive [61,62]. Interrater accuracy of angle of elicitation of symptoms in the SLR has been noted by Kosteljanetz et al. to be 66% to 75%, with interrater variability to be less than 10° [47]. Further fleshing out the properties of the SLR, Sprangfort noted that although the test was positive in more than 98% of surgical patients with disc herniation, incidence of a positive SLR sign decreases as disc level moves cephalad. Also, with increasing age, specificity seems to increase, whereas sensitivity decreases [63].

Conclusion—History and Physical Examination

Our understanding of lumbosacral radiculopathy has evolved over the past century. Appreciation of anatomic, both static and dynamic, principles, and later appreciation of other physiologic variables such as inflammation, has progressed significantly. The diagnostic abilities of history-taking and physical examination are limited however. Evaluations of the diagnostic accuracy of combined history and examination findings hint at improved diagnostic ability with such multimodality testing. But they also indicate that there is room for improvement [64,65]. A high variability in sensitivity and specificity of various historical and physical examination findings—evaluated individually or in various combinations—is the state of the current literature [66]. Even the commonly used SLR test is of questionable reproducibility [67] and overall clinical utility [68]. However, many studies that attempt to correlate such clinical—historical or physical examination—findings with the true presence of lumbar radiculopathy as a pathologic entity fail to do so. They correlate clinical finding with radiographic findings or anatomic abnormalities observed at surgery. Theoretically, these "confirmatory" modalities would fail to detect radiculopathy due to inflammation—a mechanism gaining improved understanding within the medical community. Perhaps in response to this, other diagnostic modalities maintain prominence. Electrodiagnostic evaluation (Chapter 22) can more directly assess the physiologic health of the nerve roots of interest, including those subject to inflammatory insult. Alternatively, diagnostic techniques more specifically directed toward biochemical processes causative of clinical radiculopathy have evolved.

DIAGNOSTIC SNRBS

The theoretical mechanism underlying the usefulness of SNRBs is best summarized by Schutz et al. in their early exploration of the subject: "We postulated that if the nerve root mediating the symptoms could be blocked selectively with a local anesthetic agent, the symptoms would be temporarily abolished. Conversely, if an asymptomatic nerve root were blocked, the patient's symptoms would be unrelieved" [69]. The technical features of SNRB have been discussed in detail elsewhere [70]. Key principles include the use of real-time fluoroscopy to introduce a needle immediately adjacent to the nerve root via a transforaminal approach. Needle tip placement is then confirmed with instillation of a radiopaque contrast agent, again with real-time fluoroscopic visualization. Many studies, such as Schutz's, also rely on concordant symptom reproduction at the final moment of needle placement, theoretically due to irritation of the nerve root in question. In Schutz's study, post-SNRB operative findings were in agreement with the SNRB-diagnosed root level of pathology in 13 of 15 patients [69]. The usefulness of SNRB concluded by Schutz et al. mirror that noted by Macnab in his wide-ranging analysis of nerve root pathology a few years earlier [71]. Another early study in the surgical literature is that of Krempen and Smith, in which "appropriate surgical treatment" is provided in 16 of 22 patients based on SNRB [72].

Similar accuracy was noted in a later retrospective surgical study by Dooley et al. Here, radicular pain

reproduction with needle entry into the epiradicular sheath, followed by pain relief with SNRB, was 85% accurate in indentifying a single symptomatic nerve root [73]. Patients with arachnoiditis were excluded from this calculation. Comparing the accuracy of SNRB with that of myelography, Haueisen et al. found that of 55 patients who underwent surgery, 93% had had accurately diagnosed level of pathology via SNRB, compared with 24% via myelography [74]. A favorable comparison was subsequently made alongside other diagnostic modalities. In a prospective surgical study involving 50 patients, it was noted that 20 cases demonstrated pain reproduction with transforaminal needle placement at the nerve root, with subsequent abolition of pain with anesthetic instillation. In patients with this pattern, defined as a positive diagnostic result, it was subsequently concluded at surgery that in 19 of the patients, the correct level of painful root had indeed been correctly discovered. This was in contrast to correct root level diagnosis in 14 patients achieved by computed tomography, and 12 patients by radiculography (just provide a quick, concise definition of radiculography). Noteworthy is the fact that lack of pain reproduction with needle placement essentially terminated analysis—evaluation of pain relief with anesthetic instillation, as a sole modality, was not truly performed [75]. The paradigm of SNRB positivity being defined as symptom reproduction with needle placement and subsequent relief with anesthetic instillation was subsequently utilized by van Akkerveeken. In 37 patients with disc protrusions and 9 with metastases, surgical confirmation ascribed to this protocol 88% to 100% specificity, and 90% sensitivity. The protocol's ability to predict a good result from a partial medial undermining facetectomy was at least 50% [76]. Of note, studies that utilize surgical findings as a gold standard inherently skew patient selection toward those with surgically treatable, and thus anatomic, lesions. It is theoretically possible that given SNRBs' treatment of inflammation, not anatomic compression, analysis of patients with purely inflammatory (i.e., noncompressive and thus nonsurgically treatable) lesions would demonstrate higher predictive abilities for this test.

Because SNRB is an invasive procedure, with instillation of contrast as well as anesthetic and possibly other materials (corticosteroids?) into the perineural space, complications are a possibility. Two large studies of more than 100 patients have demonstrated an absence of major complications, with a roughly 10% incidence of minor, transient, complications, such as increased back pain, increased leg pain, flushing, vasovagal reaction, nausea, headache, vomiting, and increased blood sugar [77,78]. It should be noted that the understood physiologic mechanisms responsible for flushing and elevated blood sugar implicate injected corticosteroids, implying that these reactions are much less likely to occur after injection of solely local anesthetic.

CONCLUSIONS

For the common problem of lumbosacral radicular pain, even as technologic advances enable ever more accurate anatomic evaluation, and as improvements in electrodiagnostic study permit enhanced physiologic analysis, a single perfect diagnostic protocol remains elusive. The lack of a diagnostic gold standard that would, without fail, allow researchers to compare their method of evaluation to the actual pain generator is likely the greatest obstacle. Perhaps this is why, despite longstanding evolution built upon more than 100 years of peer reviewed study, the oldest diagnostic method—that of obtaining a patient's history and performing a physical examination—provides useful but imprecise information. Conversely, the latest diagnostic technique, that of SNRB, based on the limited "gold standard" techniques that we presently possess, is shown to require further evaluation and refinement after initial promise. As our study of these diagnostic techniques, both old and new, continues in parallel with advances in other diagnostic methods, the overall optimal method of clinical patient evaluation, that set of tests that is most sensitive, specific, precise, and accurate, may yet come to light.

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21 Diagnostic Imaging of Lumbosacral Radiculopathy

Massimo Gallucci, Nicola Limbucci, and Fabrizio Fasano

INTRODUCTION

Nerve root compression can be a common cause of low back pain (LBP) and related lower limb pain syndromes. LBP is extremely common: 80% of the population have at least one episode in their lifetimes and it is the fifth leading reason for medical office visits in the United States. LBP is reported to involve 26% of the working population each year and disables 2% to 8% of affected individuals. LBP also has major economic consequences in industrial societies: its compensation accounts for 33% of all workers' compensation costs (one third for medical treatment, two third for indemnity), 75% of compensation payments go to LBP patients, although they constitute only 3% of total compensation patients [1,2].

LBP has several different causes and can occur in isolation or be associated with lower limb referred pain, weakness, numbness, and bladder or bowel symptoms. The irradiation to the lower limb along specific dermatomes is typically called cruralgic or sciatic. In such cases, specific nerve roots are involved. There are, however, several pain conditions which can mimic radicular pain such as sacroiliac joint pain (Chapter 3). Therefore, it is imperative that diagnostic imaging findings be critically evaluated and correlated with the clinical scenario. In all cases in which an interventional approach is needed, the correct assessment of the diagnosis is mandatory. The main discrimination is addressed to recognize radicular signs and symptoms from nonradicular referred somatic symptoms. In the following pages, attention will be paid to define and recognize the typical clinical and instrumental patterns of nerve root involvement to best evaluate radiologic findings.

MAIN SYNDROMES

The most common symptom of lumbar radiculopathy is lower limb radicular pain. This is a pain that refers from the back into the buttocks, thigh, legs, and feet. Cruralgia is a painful syndrome of the lower limbs, involving the anterior and medial aspect of the thigh. The sciatic and crural pain correspond to the areas covering the L4-S1 roots and the L2-L4 roots, respectively. Usually, the compromission involves one specific nerve root, more rarely two. In case of big lesions, a multiradicular involvement can determine a cauda equine syndrome. In that case, depending from the level of compromise, multiple lumbar and sacral roots are compressed and cause radicular symptoms in both legs and may impair sphincter and sexual function. In painful radiculopathies and mononeuropathies, the area of pain and sensory abnormality may extend beyond the known sensory distribution of the affected peripheral nerve or beyond the dermatome of the affected root or dorsal root ganglion, as in postherpetic neuralgia. This phenomenon has been attributed to central nervous system plasticity. In most nerve root syndromes, however, a precise description of referred pain will help localize to a nerve root level. Diagnostic imaging scans must then be critically analyzed to detect a structural abnormality that would explain the clinical picture.

RADICULAR PAIN AND INNERVATION

Radicular pain is usually described as burning, tingling (pins and needles), or "asleep" or numb in quality. The behavior of the radicular pain can help discriminate its source. Pain worse at night and with standing and walking suggests stenosis. Whereas, radicular pain that is worse with prolonged sitting and better with off-loading positions suggests a corroborative disc herniation. In most nerve root syndromes, a precise description of the referral pattern will help localize to a specific nerve root level. In painful radiculopathies and mononeuropathies, the area of pain and sensory abnormality may extend beyond the known sensory distribution of the affected peripheral nerve or beyond the dermatome of the affected root or dorsal root ganglion, as in postherpetic neuralgia (Figure 21.1). As the sensory fibers are peripheral to the ganglion and the spinal nerve, pain is the earlier symptom, associated or not with paresthesias along the dermatome. Muscle weakness is usually a sign of nerve root compression if occurring in combination with radicular numbness without radicular pain. Muscle involvement is selectively referred to the compromised root (Table 21.1). Although the majority of subjects seem to share the same dermatomal distributions, the neural anatomy of the lumbar spine has some degree of variation. Approximately, 20% of the patients have atypical nerve root dermatomal distributions. Nitta et al. [3] mapped the sensory-dermatomal distribution variations of the L4 (Figure 21.2A), L5 (Figure 21.2B), and S1 (Figure 21.2C) nerve root. In L3 and L4 nerve root syndromes, LBP can be a prominent symptom

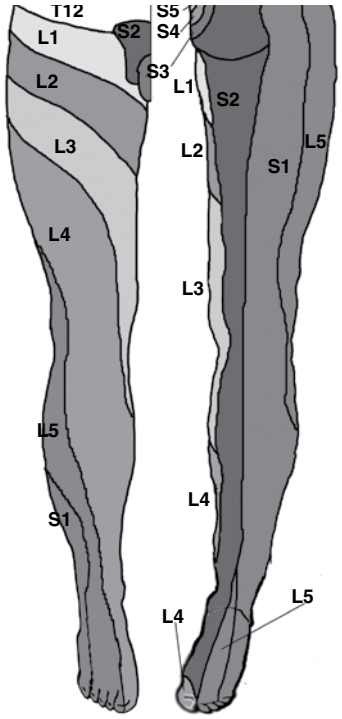

Figure 21.1 Typical dermatomal distribution of the nerve roots. On sensory neurological examination, the offending nerve root can be identified by the distribution of symptoms.

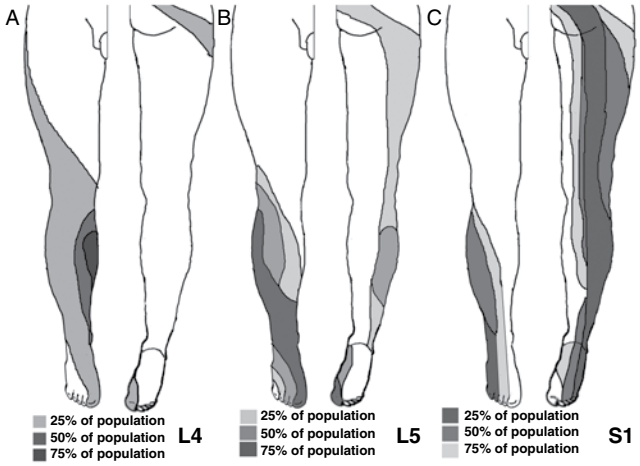

Figure 21.2 **(A–C)** Main variations of sensitive dermatomal distribution of the nerve roots L4, L5, S1.

Table 21.1 Segmental Innervation of Some Muscles of the Lower Limb

Spinal Roots	Muscle Innervated
L2-L3	Hip flexors
	Iliopsoas
L3	Hip adductors
	Adductor longus
L3-L4	Knee extensors
	Vastus lateralis
	Vastus medialis
L5	Ankle dorsiflexion eversion and inversion + hip abductors
SI	Ankle plantar flexion + hip extensors

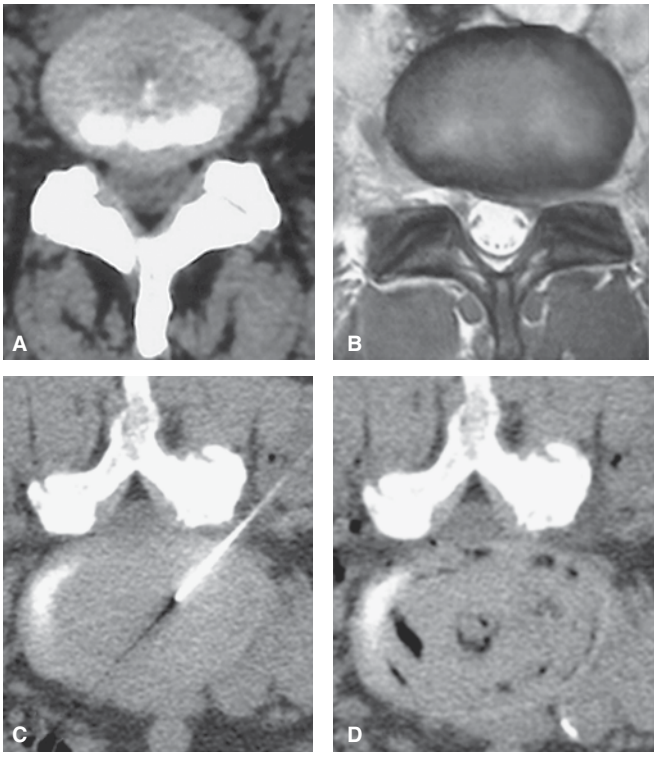

Figure 21.3 Disc bulging is defined as the condition in which the outer annulus extends over greater than 50% (180°) of the circumference of the disc and usually less than 3 mm beyond the vertebral body. It can be symmetrical **(A)** or asymmetrical **(B)**. It is considered related to the rupture of collagen bridges among annular fibers, the latter being preserved. In **(C)** a spinal needle is in a bulging disc. After oxygen-ozone injection **(D)** gas bubbles are distributed among the fibers of the annulus, confirming the pathophysiological mechanism.

in association with anterior thigh pain. The evaluating spine specialist should take these variations into account when interpreting imaging abnormalities. Radiographic studies of the lumbosacral spine of patients experiencing radicular pain should be critically examined for a specific lesion that could explain that patient's signs and symptoms.

MAIN CAUSES OF RADICULOPATHY

Degenerative Disc Disease

The main cause of lumbosacral radiculopathy is disc herniation, although other kinds of degenerative disc changes can be responsible for radiculopathy. Symptoms are usually due to nerve root compression and/or tension and focal demyelination induced by disc material. Radicular pain usually has an inflammatory component as well (radiculitis). The side and the level of the symptoms depend on the location of the disc herniation and the anatomic relationship with the nerve root. In the literature and in clinical practice, the definition of the forms of disc degeneration is often not univocal, and different terms are often used for the same entity and vice versa,

generating confusion among physicians and patients. The most accepted nomenclature for disc herniation was published in 2001 [4]. According to this classification, disc abnormalities include bulging disc (Figure 21.3) and disc herniation, which is subdivided in protrusion and extrusion (Figure 21.4). Protrusion is defined when the greatest distance between the edges of herniation is less than the distance between the edges of the base in the same plane, extrusion when anyone of the distance between the edges of disk material is greater than the distance between the edges of the base. A commonly adopted classification of disc herniation divides contained and uncontained disc herniations. The first is presumed to correspond, in term of imaging, to protrusion, whereas the second to extrusion. In the former, the displaced disc tissue is contained within an outer perimeter of uninterrupted outer annulus, whereas in the latter the outer annulus is disrupted allowing egress of nuclear material (Figure 21.5). This concept was very important in establishing the indications for percutaneous injection of chymopapaine. Newer interventional techniques do not need this specific information. Plain films of the lumbar spine are of limited value in patients with radiculopathy due to intervertebral disc injury. However, they can show indirect signs of disc collapse and degenerative spondylosis, also allowing to rule out of other causes of pain (tumor, fracture, infection); therefore, in many cases of elderly patients with LBP and radiculopathy, plain films allow acceptable imaging if no invasive treatments are planned and if radiologic and clinical patterns are congruous [5]. In acute disc herniation, plain films can be negative, especially in young patients. When disc herniation is associated with degenerative disc changes, the most evident sign is reduction of the intervertebral space due to loss of disc height, often with evidence of intradiscal air depicted by pockets of darkness within the disc space (Figure 21.6) [6]. Sclerosis of the endplates is also common, often in association with osteophytes (Figure 21.7). In patients with radiculopathy, oblique projections are useful to diagnose osteophytes protruding in the neural foramina. Discography with postdiscography computed tomography (CT) may be useful to detect latent disc hernia which may be difficult to detect on static imaging. CT itself is accurate in discriminating between intervertebral disc bulging, due to lamellar separation, and herniation due to annular fissuring. CT allows adequate evaluation of the dimensions and location of the herniated disc, anatomic relationship, and detection of free fragments. Because the disc is hypodense, the nucleus pulposus cannot be exactly identified; therefore, the

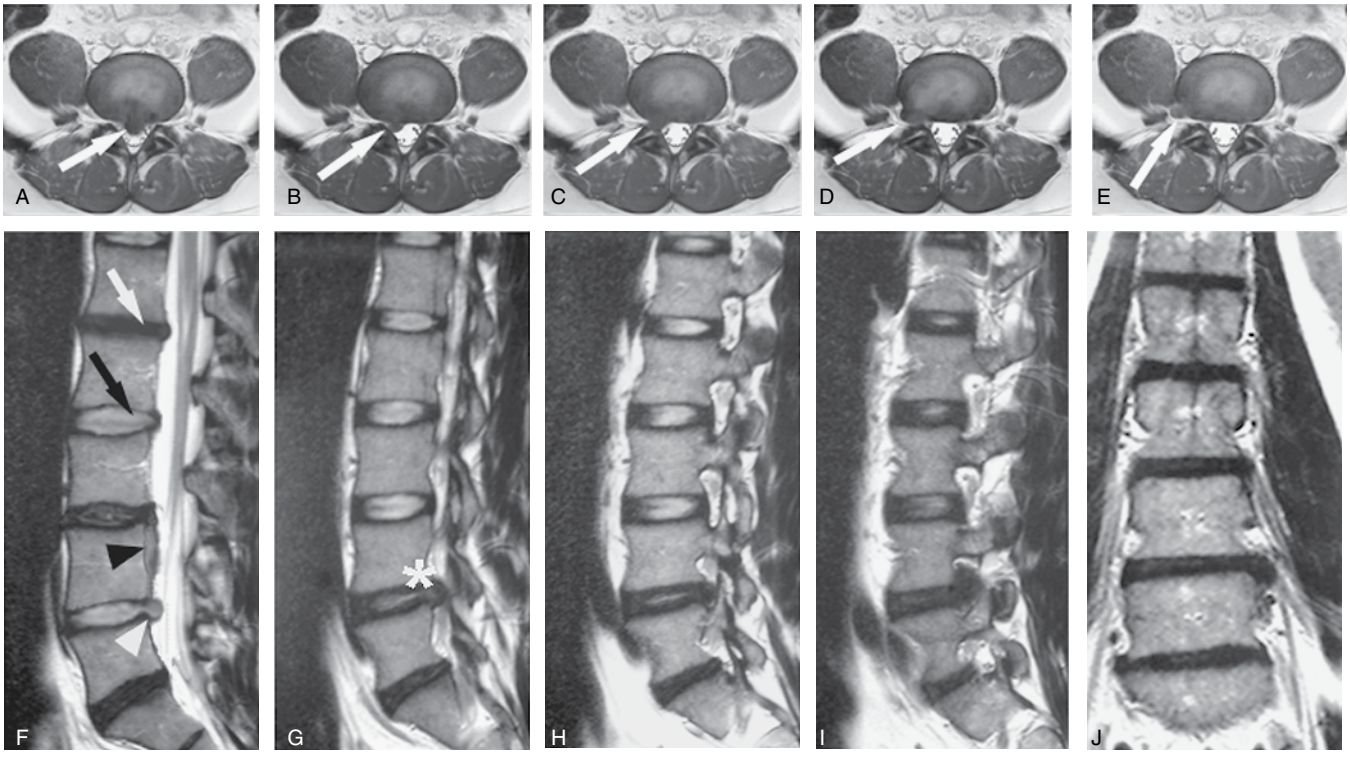

Figure 21.4 Different types of disk diseases as from a computer simulation. In "**F**" white arrow indicates thinning and dehydration; black arrow indicates intranuclear cleft, white arrowhead indicates extrusion (noncontained herniation), black arrowhead indicates free (migrated) fragment. In "**G**" asterisk indicates a contained herniation (usually represented at imaging as protrusion). The upper row shows a possible collocation on transverse plane, at L4-L5 level (sagittal and coronal cuts on the lower row corresponds to the transverse ones). In "**A,F**" midline herniation; in "**B**,**G**" lateral; in "**C**,**H**" subarticular; in "**D**,**I**" intraforaminal; in "**E**,**J**" extraforaminal.

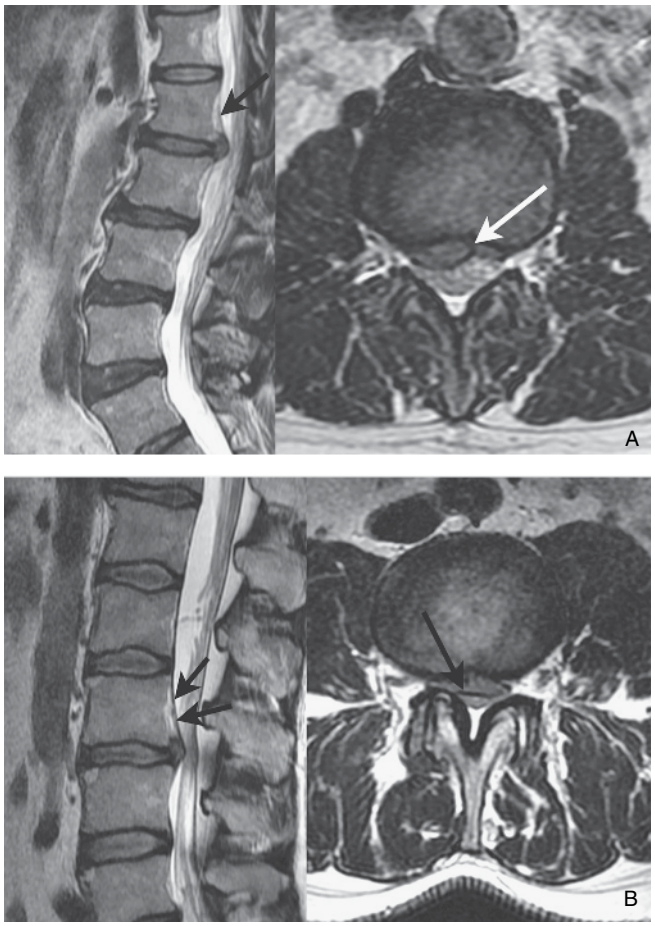

Figure 21.5 Different types of extrusions. MRI is rarely able to provide the correct distinction between contained and noncontained disc herniation, this differentiation being essentially anatomical. Usually extrusion corresponds to noncontained herniation. Moreover, distinction between retro and trans ligamentous herniation is also difficult. Sometimes it can be presumed taking into account that the posterior longitudinal ligament (PLL) strictly adheres through the ligament of Trolard, to the posterior aspect of the vertebral body. The ligament of Trolard thus inhibits a contralateral extension of herniated material. In "**A**" white arrow indicates the ligament of Trolard along the midline, forming a barrier versus disk herniation. Black arrow indicates presumably PLL together with dura mater. In "**B**" disc material is extending across the midline, likely having crossed the PLL. Black arrows indicate dura mater.

differentiation between contained and uncontained herniations is weakly accurate. CT allows easy identification of calcified herniations and associated spondylotic changes better than magnetic resonance imaging (MRI). The accurate visualization of bony structures provides accurate diagnosis and quantification of stenoses of the neural foramina, the lateral recesses and the vertebral canal.

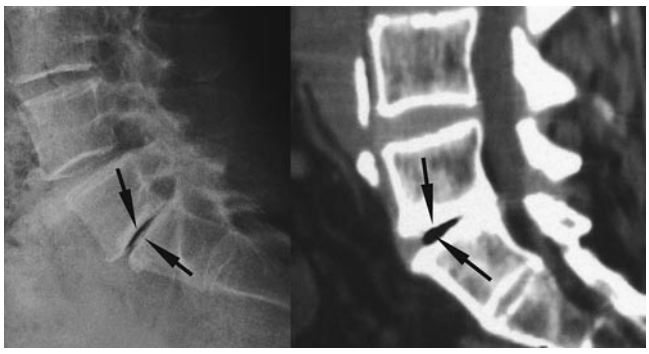

Figure 21.6 X-ray LL projection and midsagittal reformatted CT cut of a "vacuum phenomenon" at L5-S1 level. Arrows indicate air in substitution of degenerated disc material. The endplates are in contact due to disc collapse, and sclerotic changes are evident.

MRI is the most accurate technique for evaluating herniation of the intervertebral disc [7]. Disc degeneration appears as loss of disc height and hypointensity on all sequences due to dehydration of the nucleus pulposus. In elderly patients, herniations are usually associated with signs of chronic disc degeneration, whereas in young patients, herniations can occur in normal signal discs (Figure 21.8).

Disc bulges are usually associated with degenerative changes and are easily recognized as wide base hypointense prominence of the outer contour of the disc without interruption of the annulus. Herniations are focal and their signal can vary depending on their age and composition. Acute herniations can be hyperintense on T2-weighted fast spin-echo images; the hyperintensity of the fragment is due to the higher water content of the nucleus compared with the annulus [5]. Intravenous gadolinium administration is not necessary in most lumbosacral spine MRI studies. Herniated fragments in acute phase usually show peripheral enhancement [8] (Figure 21.9). Gadolinium administration is useful in selected cases of recurrent radicular pain after surgery to determine whether the herniation is a remnant or recurrent herniation, and if hypertrophic scar tissue is present, which show strong enhancement (Figure 21.10) [9]. After spine surgery, enhancement is usually visible in different structures in normal conditions: granulation tissue, nerve roots, meninges, paraspinal muscles. It is however considered that enhancement of meninges or nerve roots persisting more than 6 months after surgery is strictly correlated with recurrent pathology.

Spondylosis and Stenosis

In elderly individuals, some degree of spondylosis is found invariably during diagnostic imaging examinations and can be considered physiologic. After the age of 50, spondylosis is found in 60% of women and 80% of men regardless

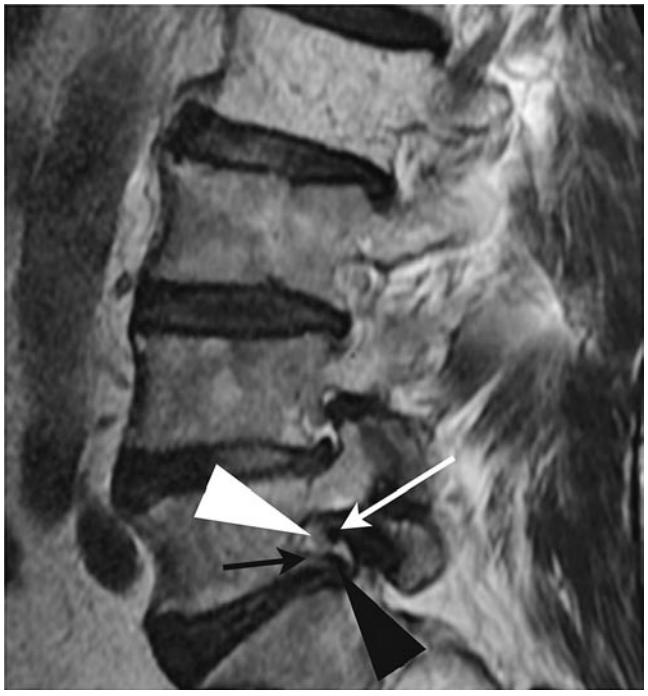

Figure 21.7 Typical appearance of degenerative foraminal stenosis. Disc degeneration produces a cascade of events mainly represented by disc height reduction, consequent intracanalar compression on the superior articular facet that becomes sclerotic (dark signal). Its tip is stimulated to osteoblastic activity, thus producing osteophytes (white arrow). The vertebral endplates receive osteoblastic stimulation due to Sharpey's fibers traction consequent to disc bulging (black arrowhead). Osteophytes, therefore, form in the intervertebral canal (small black arrow). Narrowing of the canal produces impingement of the nerve root (white arrowhead).

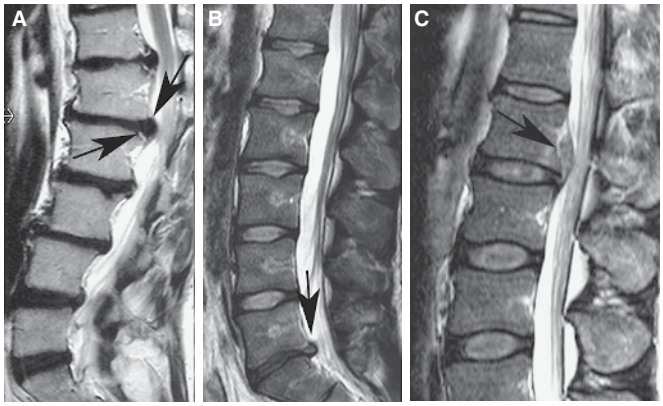

Figure 21.8 Three cases of different signal patterns in disc herniations. In **(A)** "black disc" with "black herniation" associated to multiple degenerative discovertebral degenerations in a subject aged 78. In **(B)** L4-L5 hyperintense disc extrusion from a midintensity disc, reduced in height, in a 34-year-old man. In **(C)** L1-L2 hyperintense disc fragment migrated upward, coming from an hyperintense disc of a 26-year-old subject.

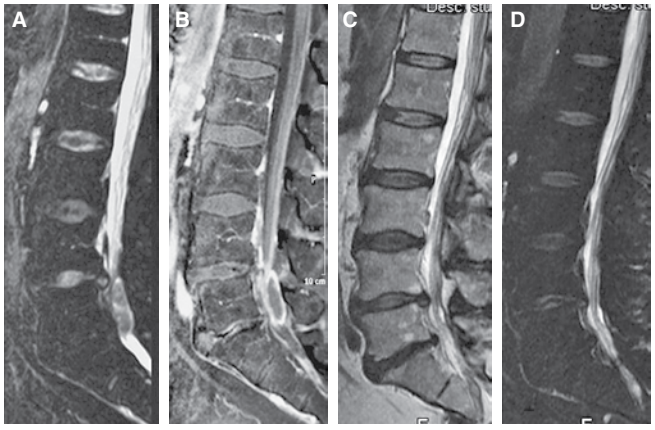

Figure 21.9 Peridural disc fragment in a patient with lumbar stenosis. Acute phase, in **(A)** T2-weighted fast spin-echo (FSE) in acute clinical phase; in **(B)** fat-sat T1-weighted SE after contrast administration. Note the peripheral enhancement of the disc material. In **C and D**, respectively, T2-weighted FSE and fat-sat FSE after 6 months shows the complete disappearance of the fragment.

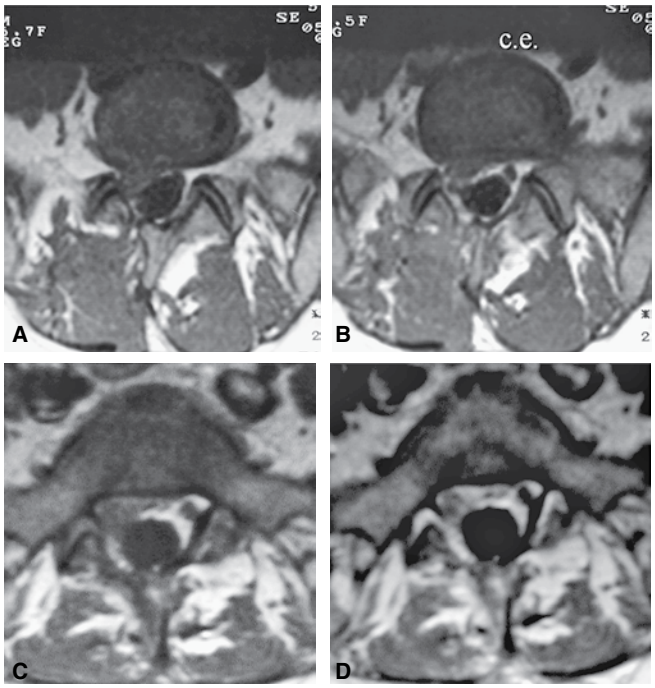

Figure 21.10 Postoperative spine. **(A,B)** Residual herniation—the disc fragment shows peripheral enhancement during the first 10 minutes after injection **(B)**. **(C,D)** Granulation tissue—diffuse and intense enhancement **(D)** of the hypointense tissue surrounding S1 nerve sleeve.

of symptoms. Lumbar spine degenerative changes should be considered pathologic when they are severe or symptomatic. Lumbar spondylosis is often associated with LBP, but severe degenerative changes can produce central canal and foraminal stenosis, leading, respectively, to cauda equina syndrome or radicular symptoms. Spinal stenosis is defined as a clinical condition in which the dimensions of the spinal canal have reached a critical value resulting in neurologic signs and symptoms, related to direct compression of its contents.

Spinal stenosis may be classified according to etiology in congenital, acquired, or combined. According to location, it can be divided in central, lateral, foraminal, and concentric, sometimes with overlap between the various types (Figure 21.11). Congenital lumbar spine stenosis is rare, but combined forms are quite common. The most usual idiopathic factor predisposing to stenosis is congenital shortness of the pedicles, whereas anomalies in vertebral development, metabolic disorders, and skeletal dysplasia are much more rare. The most common causes of acquired stenosis are degenerative spine changes, including spondylosis with or without concomitant posterior arch and disc abnormalities.

The classic radiographic signs of spondylosis are osteophytosis and endplate sclerosis, which are often coupled with thinning of the disc space because of concomitant disc degeneration. Plain films are adequate for the diagnosis of spondylosis and are helpful for the differential diagnosis between osteophytosis and other bony excrescences with different origin. However, plain films cannot adequately assess the presence and severity of disc pathology and degree of canal or foraminal stenosis. Therefore, CT or MRI is useful as second-level examinations. Upright dynamic plain films are useful to evaluate spinal instability which can lead to radiculopathy [10]; in selected cases, upright MRI examination can reveal a dynamic stenosis (Figure 21.12), thus showing increased narrowing of the canal, as already observed by x-ray myelography (Figure 21.13). This application can be useful in patients with radiculopathy without significant changes at baseline MRI.

Posterior Arch Pathology

Facet Joints Arthropathy

Facet joints are frequently involved in osteoarthritis, often associated with disc degeneration and canal stenosis. Facet joint syndrome typically presents as median LBP, which is often distributed to the buttock and posterior aspect of the thighs and can become indistinguishable from radicular pain; therefore, facet pain enters in the differential diagnosis with radiculopathies. Sometimes, facet arthropathy contributes to radiculopathy by encroaching upon the lateral canal and or foramen.

Plain films can show joint space narrowing, subchondral sclerosis and cysts, osteophytosis, and associated spondylosis. CT and MRI more accurately characterize complex anatomic changes, including facet bony hypertrophy, thickening of the ligamentum flavum, and intra-articular fluid. Severe facet osteoarthritis can determine lateral recess and neural foramen stenosis; less frequently, canal stenosis can be observed. CT is more accurate for determining bony abnormalities, but MRI more clearly shows neural structures and soft tissues [5].

Facet joint osteoarthritis may be associated with vertebral instability. Weight-bearing MRI can be useful in selected cases to diagnose facet joint instability, determining appearance of foraminal stenosis only during axial loading (Figure 21.14) [11]. A possible complication of facet joint degeneration is the formation of synovial cysts, which originate from the joint and can keep or occasionally lose the connection with the joint. Usually, cysts contain synovial serous fluid so they are hypodense on CT and have fluid-like signal on MRI. Sometimes, they contain gelatinous material, air, or blood. Long-lasting cysts can fully

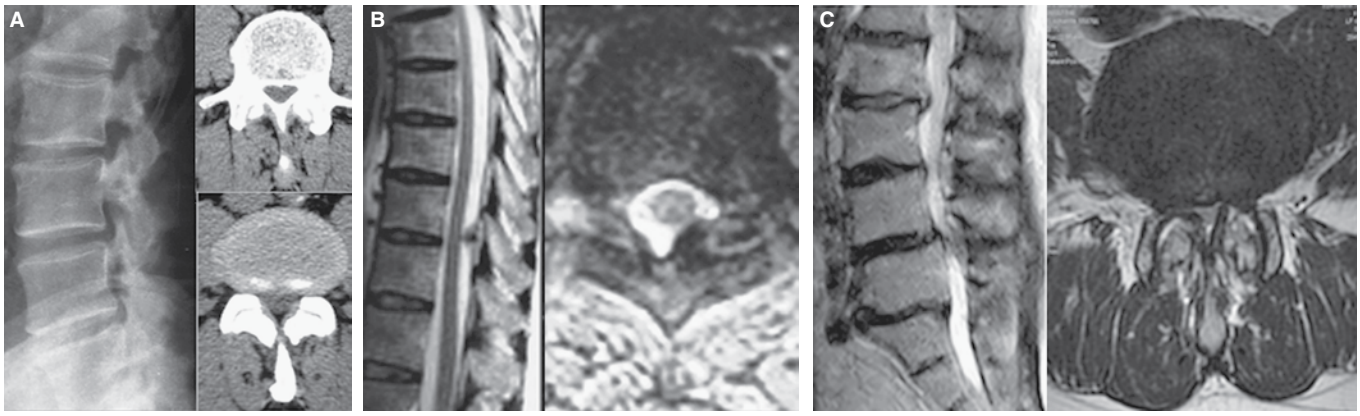

Figure 21.11 Different examples of stenosis. **(A)** Congenital stenosis due to short pedicles. It mostly affects the midsagittal diameter (central stenosis). **(B)** Lateral stenosis due to flavum ligament thickening. **(C)** Concentric stenosis due to the coexistence of both congenital and degenerative forms (mixed stenosis).

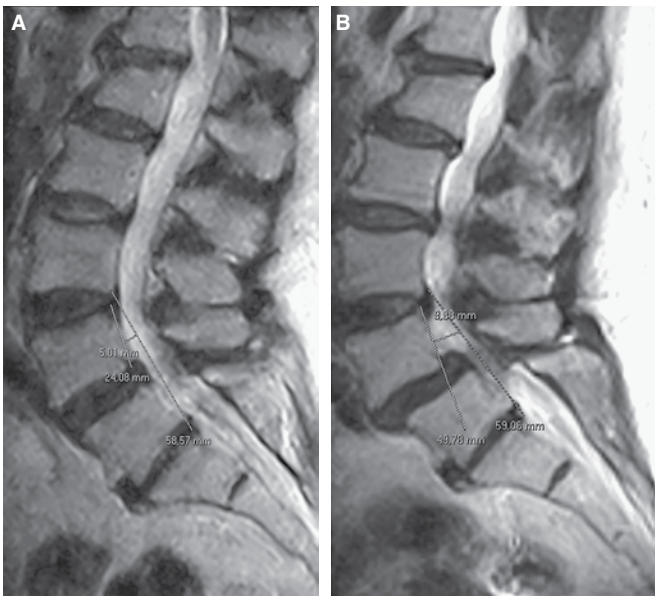

Figure 21.12 Supine and upright T2-weighted midsagittal cuts in a patient with degenerative segmental instability. The distance between the line connecting the superior and the inferior angles of the instable segment, and the line connecting the inferior angles of the upper and lower vertebral bodies increases about 100% in upright position.

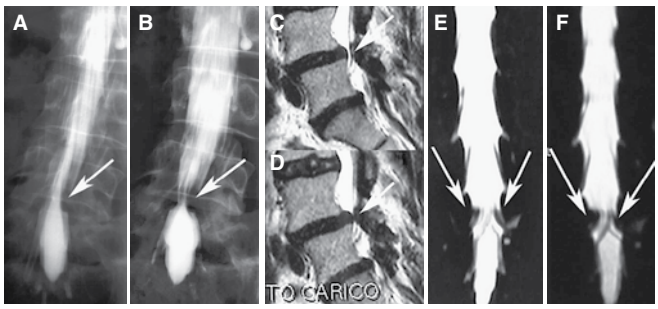

Figure 21.13 L4-L5 segmental stenosis. **(A,B)** X-ray myelograms. **(A)** Patient in squatting position. **(B)** Prone position. In squatting, a decreased compression on nerve sleeve is evident. **(C,D)** T2-weighted FSE cuts, respectively, in rest condition **(C)** and under axial loading **(D)**. **(E,F)** Myelo-MR, respectively, in rest and under loading. During loading **(D,F)** increase of recessual stenosis and compression on nerve root sleeve becomes evident.

calcify. The diagnosis of synovial cysts can be confirmed by percutaneous aspiration, also useful for curative purposes (Figure 21.15).

Spondylolysis and Spondylolisthesis

Six types of spondylolysis have been defined: dysplastic, isthmic, traumatic, pathologic, iatrogenic, and degenerative (pseudospondylolysis). The most common kind of lumbar spondylolysis is the isthmic type, which is a typical pathologic condition of children, adolescents, and

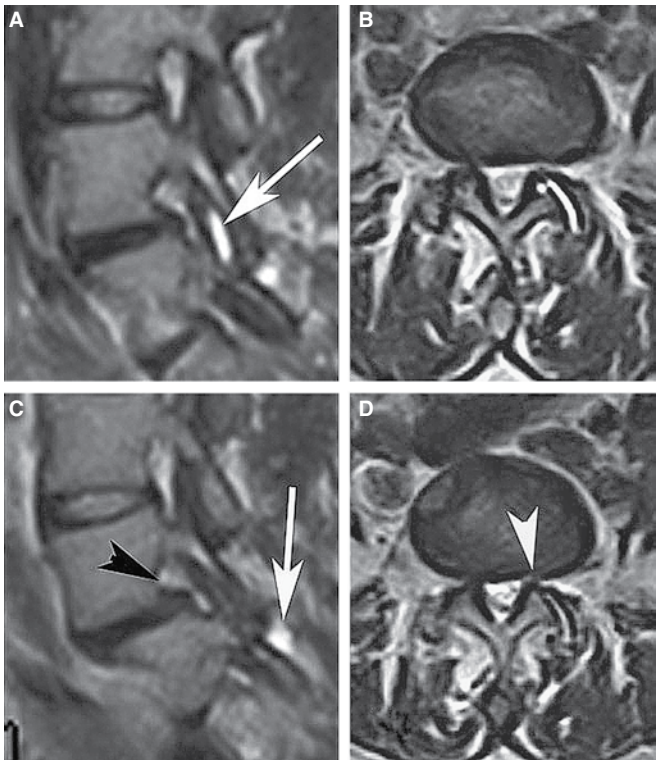

Figure 21.14 Dynamic recessual stenosis. **(A,C)** T2-weighted sagittal and axial cuts show interapofiseal synovial leakage. **(B,D)** Same cuts and sequences in upright position: the leakage is squeezed into the inferior articular recess, whereas the upper vertebra splits anteriorly. **(D)** Splitting of the facets gives rise to subarticular stenosis and nerve root compression.

young adults. Isthmic spondylolysis can be defined as a defect of the pars interarticularis of the vertebra, and it is considered a fatigue fracture produced by abnormal mechanical stresses on an otherwise normal bone. The most common site of spondylolysis is L5 (81%), followed by L4 (14%). The prevalence of spondylolysis in the general asymptomatic population is approximately 3% to 7%, but it is higher in participants in sports activity [12,13]. Plain films are useful for diagnosing spondylolysis: a lateral view often allows identifying the isthmic lysis as a defect of the pars interarticularis with sclerotic borders, but the examination should be completed by oblique view, where spondylolysis can be recognized for the classic sign of the "Scottish terrier's collar." CT is accurate for the detection of lysis, which appears as transverse isthmic fracture with irregular rim and sclerosis. Images should with acquired with a reverse gantry angle (15%–25%) or reviewed with reconstructions parallel to the axis of the isthmus; otherwise, differentiation between the lysis and normal facet joints can be difficult. MRI is less sensitive but less accurate than CT; lysis can be distinguished on MRI as an interruption of the normal bony signal. With time and persistence of microtrauma and weight bearing, spondylolysis often leads to spondylolisthesis, which is defined as anterior or posterior slippage of a vertebral body. In the elderly population, spondylolisthesis is

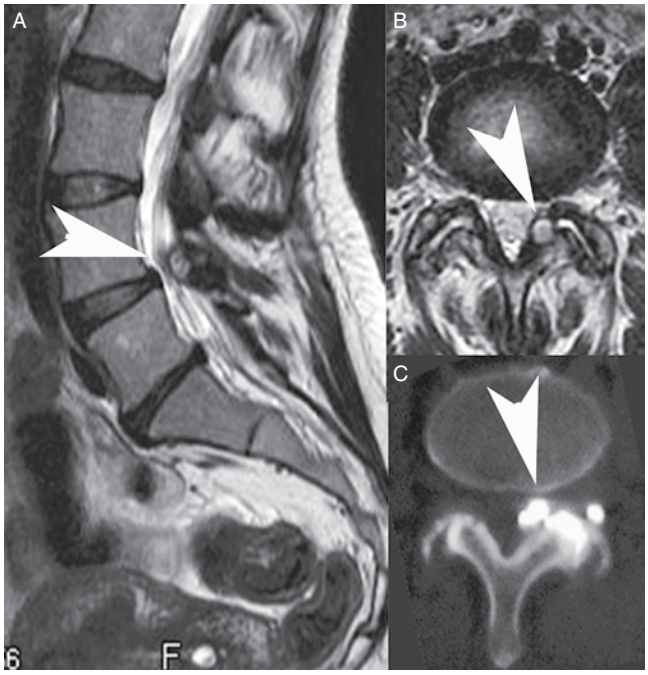

Figure 21.15 Synovial cyst at L4-L5 level. Cysts are invariably associated with signs of segmental instability (i.e., intra-articular leakage). **(A,B)** T2-weighted sagittal and axial cuts, respectively. **(C)** After putting a needle inside the articular space, a small amount of iodinated contrast has been injected in order to obtain an arthro-CT.

frequent (approximately 4%) and is usually not related to spondylolysis but to severe degeneration of the facet joints. The typical sites of degenerative spondylolisthesis are L3-4 and L4-5 because of the more sagittal orientation of the joints. Anterior spondylolisthesis can be classified in four grades according to Meyerding's classification. Degenerative spondylolisthesis is usually grade I (slippage below 25%) [12].

If spondylolisthesis is caused by isthmic lysis, the anterior slippage causes widening of the vertebral canal. Conversely, when spondylolisthesis has a degenerative origin the canal undergoes anteroposterior narrowing because of slippage of the posterior vertebral arch and facet hypertrophy. Radiculopathy is possible in both cases. In isthmic spondylolysis with spondylolisthesis, radiculopathy is due to contact of the root with the lower portion of the interrupted isthmus and to development of fibrotic adherences between the root and the tissue surrounding the lysis; therefore, mechanical stress of the unstable segment are transmitted to the root. In degenerative spondylolisthesis, anterior slippage of the lower vertebra causes direct bony stenosis of the neural foramen with subsequent root compression.

Upright plain films are necessary for a correct diagnosis and grading of spondylolisthesis. Dynamic radiographs in hyperflexion, hyperextension, and lateral bending are useful for evaluating associated vertebral instability, which is characterized by loss of alignment of one or more vertebral lines. Radiographic signs of instability obtained with dynamic films are evidence of anterior or posterior vertebral slippage during motion or under load, pedicle length variations, neural foramina narrowing, and loss of intervertebral disc height. Other associated signs are intradiscal vacuum and traction osteophytes. Conventional MRI can show spondylolisthesis, but its value is limited for functional information. MRI often shows "pseudobulging," which usually occurs at the level of the lysis, and narrowing of the neural foramina. Axial loaded CT and MRI or upright MRI can provide functional information about vertebral stability and spinal response to physiologic load conditions (Figure 21.16) [14].

UNCOMMON CAUSES OF RADICULOPATHY

Spinal Tumors

Spinal tumors are relatively common. Extradural vertebral metastases are the most frequent kind of spinal tumor, and radiculopathy can be the first clinical sign or develop at some point during the course of the disease. Radiculopathy is due to neural foramen or central canal invasion and stenosis. Plain films can show focal bony lesions, and pedicle disruption can often be suggestive for foraminal involvement. Bone scan is useful to rule out the presence of metastatic tumors in patients with known primary tumors, and CT or MRI should be used to confirm and better evaluate bone scan findings. In patients without known malignancies, the presence of radiculopathy resistant to conventional treatments with negative plain films can be an indication to perform bone scan or MRI.

Less commonly, primary vertebral neoplasms can became manifest with radiculopathy.

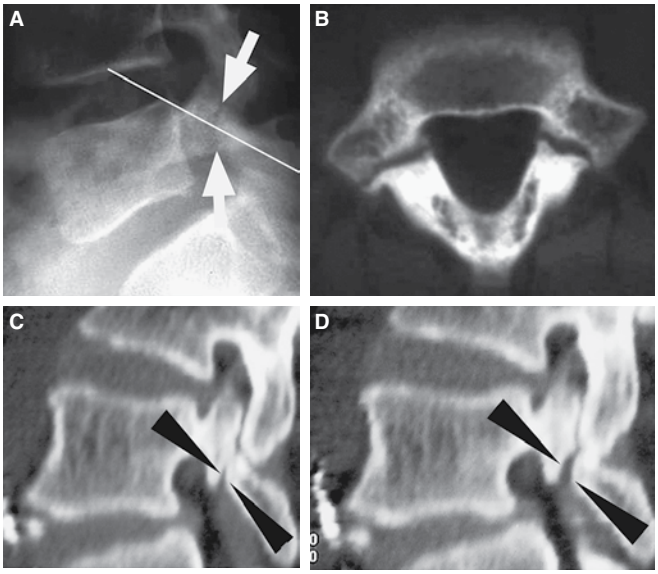

Figure 21.16 Isthmic spondylolysis as demonstrated by LL conventional x-ray film **(A)**, CT reformatted cut perpendicular to the isthmus and CT sagittal reformatted images **(C,D)**. **(C)** examination performed in rest position; **(D)** during application of axial loader. Note the increased distance between the two segments under stress (arrows).

Also intradural extramedullary tumors, such as schwannomas and meningiomas, can be a cause of radiculopathy. In those cases, the neoplasm can be intraforaminal and differential diagnosis can sometimes be difficult. MRI with gadolinium administration can be useful for this purpose.

Arachnoiditis

Adhesive arachnoiditis is usually secondary to spinal surgery. Rarely, it is secondary to infections, myelography, or intrathecal drug administration. MRI provides best information about this condition. Nerve roots appear clumped and the thecal sac looks like "empty" because of adhesion of the nerve roots to its walls. It is possible to observe an intrathecal "mass-like" tissue with a broad dural base, representing a large group of clumped roots. A helpful diagnostic tool can be achieved by reexamining doubtful cases in prone position: in normal cauda equine nerve roots move following gravity, toward the anterior aspect of the sac. In adhesive arachnoiditis, some or even all cauda equine roots do not, being glued to the arachnoid membrane (Figure 21.17). Contrast enhancement of the thickened meningeal scarring and intrathecal roots may be observed, but it is a rare finding.

Multiradiculo Neuritis

Guillain-Barré syndrome is an acute autoimmune disorder against peripheral nervous system. Symptoms include weakness in the legs spreading to the arms and upper body. These symptoms can increase until a generalized peripheral palsy. Usually Guillain-Barré occurs a few days or weeks after the patient has had symptoms of a viral infection. MRI shows multiradicular enhancement in the cauda equine, eventually spreading also to upper nerve roots (Figure 21.18). Lumbar puncture is useful for diagnosis: cerebrospinal fluid contains more protein. Chronic inflammatory demyelination of the peripheral nerves is a rare chronic form of autoimmune involvement of the peripheral nervous system. Painful or sensitive onset is typical, followed by progressive deficit of multiple roots within a couple of months. Thickening and enhancement of the nerve roots is typically registered at MRI.

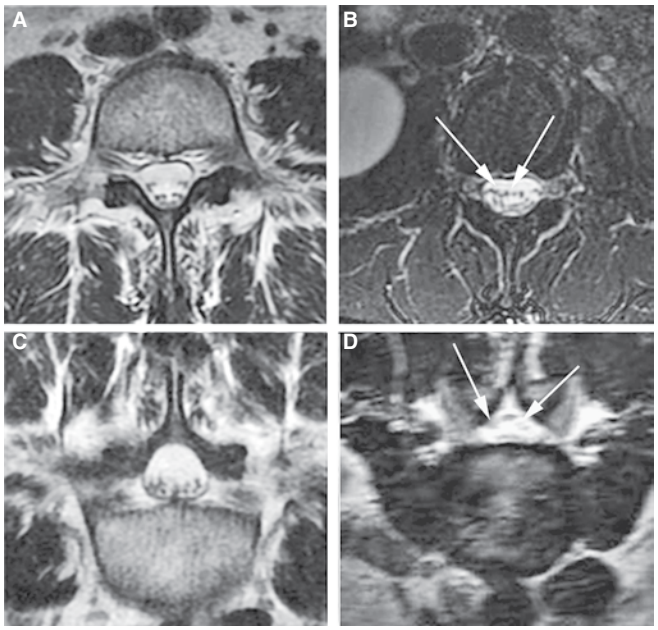

Figure 21.17 Adhesive arachnoiditis. **(A,B)** Normal patterns: nerve roots are free to move and lie in the lower position of the thecal sac, moving anteriorly in prone position. **(C,D)** Supine and prone positions, respectively, in a case of adhesive arachnoiditis: nerve roots are not ordinate along the posterior aspect of the sac and tend not to move anteriorly in prone position.

Tarlov Cysts

Tarlov or perineural cysts are a very common incidental finding seen in MRI studies of the lumbosacral spine most frequently encountered in the sacral region, especially at the S2 and S3 levels. In most of cases, these cysts have no clinical relevance, but in a small number of patients, the cysts are responsible for LBP, radicular pain, or urologic disorders. It is often very difficult to decide what role the cyst plays in the patient's symptomatology.

The cysts usually develop at the junction of the dorsal root with the dorsal root ganglion. Cysts are often perineural, but at times the nerve rootlets are embedded in the cyst's walls. The cysts have a fluid-like appearance, their wall is thin, and they do not enhance with contrast; sometimes cysts can cause scalloping of the sacral canal or foramina because of increased intracystic pressure, maybe due to a valve mechanism [15]. Cysts can be closed or communicate with cerebrospinal fluid, which is important if percutaneous aspiration or sclerosing has to be attempted.

EVIDENCE-BASED REVIEW OF THE PREDICTIVE VALUE OF RADIOGRAPHIC FINDINGS

Although diagnostic imaging studies often reveal pathologic findings in people affected by LBP and radiculopathy, the presence of radiologic signs of degenerative spine

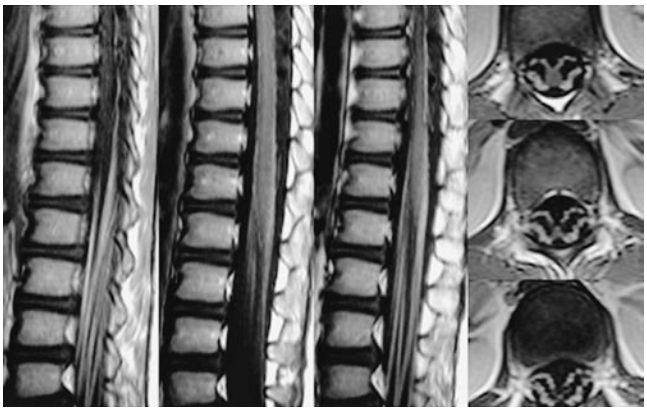

Figure 21.18 Typical patterns of Guillain-Barré syndrome. All cauda equine nerve roots diffusely enhance after contrast administration.

abnormalities is very common also among the asymptomatic population. Many studies have demonstrated that a significant proportion of asymptomatic individuals have moderate to severe degenerative spine lesions [16,17]. Most asymptomatic adults have imaging findings of mild degenerative disc changes with disc dehydration (black disc), and lumbar disc herniations or bulging discs have been reported in 27% to 67% of the population. Nevertheless, according to the results of a large meta-analysis, plain film degenerative changes (disc space narrowing, osteophytes, and endplates sclerosis) are consistently and positively associated with nonspecific LBP [17].

The association between radiculopathy and disc herniation is probably more specific, although meticulous clinical correlation is necessary to establish a relationship between clinical and radiologic findings [18].

Lumbar spine MRI performed in asymptomatic adults confirmed a very high prevalence of bulging disc and disc herniation in a normal population (respectively, 52% and 24%); the prevalence of significant stenosis was much lower (4%) [16]. Given the high prevalence of degenerative disc changes in asymptomatic individuals and high prevalence of back pain in the adult population, the MRI evidence of bulging or small protrusions in symptomatic people may frequently be coincidental and not related to symptoms.

There are no widely accepted guidelines concerning the role of diagnostic imaging in patients with LBP or radiculopathy. Some authors suggest that in people younger than 50 years with nonspecific LBP without signs or symptoms of systemic disease, diagnostic imaging does not improve treatment of LBP. For patients 50 years of age and older or those whose findings suggest systemic disease, plain films and laboratory examinations can usually rule out underlying systemic diseases. According to this review, advanced imaging should be reserved for patients with severe pain, amenable to surgery, or those in whom systemic disease is strongly suspected [19,20]. In a recent study, all randomized controlled trials that compared immediate lumbar imaging (radiography, MRI, or CT) versus usual clinical care without immediate imaging for LBP were analyzed. Overall, the investigators did not observe any significant differences between immediate lumbar imaging and usual care without immediate imaging for primary outcomes at either short-term or long-term follow-up. Therefore, lumbar imaging for LBP without indications of serious underlying conditions does not improve clinical outcomes [21].

In clinical practice, MRI is often performed in patients with radiculopathy. However, MRI can underestimate the clinical significance of lateral recess stenosis causing root compression. Compared with surgical decompression, MRI underestimated lateral recess root compression in 29% of cases, whereas conventional myelography failed to document nerve impingement in only 5% to 7% of affected cases [22]. This MRI pitfall must be carefully evaluated especially in the elderly, because multilevel radicular compression is often present [19].

More recently, longitudinal CT and MRI studies have allowed us to better understand the natural history of disc herniations, which is favorable in most cases. Long-term follow-up studies in patients with symptomatic lumbar disc herniation showed that complete regression or reduction of symptoms occurred in 71% to 95% of cases after 1 year, and stability or worsening occurred in 5% to 29% of patients [23–25]. Long-term follow-up of surgically and conservatively treated patients showed similar outcome [26,27]. MRI follow-up studies 6 to 12 months after the diagnosis of disc herniation showed that spontaneous volume reduction occurs in up to 63% cases [23,28,29]. The possible reasons of spontaneous reduction of herniated material are as follows: fragmentation related to matrix degeneration and loss of proteoglycans integrity; shrinkage caused by dehydration; and immunomediated phagocytosis of the disc material [5].

MRI provides some information useful to presume spontaneous regression of disc herniations with a good predictive value. The main MRI findings that positively predict spontaneous regression after 6 months are as follows: free fragments (100%; Figures 21.9 and 21.19), hyperintense

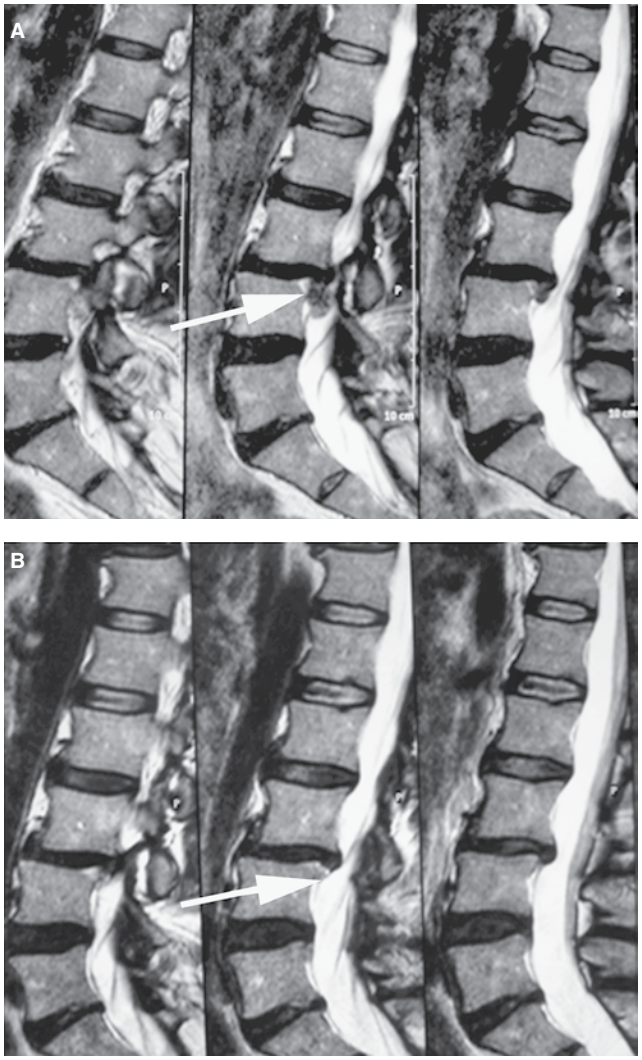

Figure 21.19 Big disc fragment at L3-L4 level, associated with diffuse degenerative changes and signs of segmental instability. (**A**) Acute clinical phase. (**B**) Spontaneous anatomical and clinical regression after 6 months.

herniated disc material on T2-weighted images (83%), and peripheral enhancement after gadolinium administration (80%; Figure 21.9). More acute onset of clinical symptoms correlated positively with reduction in herniation volume, registered in up to 75% of cases [30,31]. Among the types of disc herniations, protrusions are usually more stable than extrusions, perhaps because the nucleus pulposus is still covered by annulus layers. Bulging disc history is different, because it usually does not undergo spontaneous anatomic regression and symptoms are more stable.

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22 Electrodiagnostic Evaluation of Lumbosacral Radiculopathy

Jeff Strakowski and Ernest W. Johnson

INTRODUCTION

Assessment of radiculopathy is an integral element of comprehensive spine care. Radiculopathy accounts for a large amount of morbidity related to spinal disease and is the most common reason for spinal surgery and less invasive spinal procedures. Electrodiagnosis (EDX) is an important tool in evaluating radiculopathy and remains the only objective physiologic test in assessing peripheral nerve function [1,2,3]. It is frequently cited as a measure of confirming the presence or absence of radiculopathy and determining the specific root involved; however, establishing the relative severity of the neurologic deficit is likely a more important role. Determining the degree of neurapraxic block versus axonal injury, with potential prognostic implications, in the face of clinical weakness, is not readily accomplished by clinical means prior to the development of muscle loss and atrophy in more chronic situations [4,5,6]. Electrodiagnostic techniques are also extremely valuable in distinguishing radiculopathy from, and in the presence of, many comorbid conditions such as a generalized peripheral neuropathy or concomitant peripheral nerve entrapments [7]. The objective nature of EDX is a desirable feature in the realm of sometime inconsistent subjective patient complaints. It also can be helpful in distinguishing factitious weakness from true peripheral neuropathic abnormalities.

A comprehensive understanding of the appropriate peripheral nerve anatomy and pathophysiology as well as the relative utility of electrodiagnostic techniques is necessary for adequate performance, reporting, and interpreting electrodiagnostic findings [8]. Knowledge of potential sources of error and ability to accurately report the results are also critical for maximizing the benefit of this powerful diagnostic tool.

THE BASICS OF ELECTRODIAGNOSTIC TECHNIQUES

Needle Electromyography

Motor units are recorded by obtaining the electrical potential given off by the muscle membrane and running the signal through a differential amplifier to enhance the biological signal and reduce unwanted atmospheric interference [9,10]. A monopolar or concentric needle is inserted into the muscle to obtain this recording (Figure 22.1).

The muscle is first evaluated at rest to assess insertional and spontaneous activity [11]. This includes investigation for positive sharp waves (PSW), fibrillations (fibs), complex repetitive discharges (CRDs), and fasciculations. PSWs and fibs (Figure 22.2) reflect instability of the muscle cell membrane and are manifestations of spontaneous depolarizations of single muscle fibers. They are an important clue for identifying a peripheral nerve injury pattern in radiculopathy. The muscle cell membrane components require continuity with the anterior horn cell to maintain its integrity [12]. This integrity is ultimately lost by muscles, innervated by the particular root, in radiculopathy. This process may take up to 8 to 21 days, depending on the degree of axonal injury and distance of the muscle from the site of the injury [13,14].

PSW and fibs (Figure 22.2) are generally graded on a scale of 1 to 4 reflecting the relative number. This is not an ordinal scale; therefore, 4+ PSWs and fibs do not reliably reflect a more severe lesion than a smaller number. A higher degree of PSWs and fibs simply provide higher confidence that an abnormality is present. Because 4+ PSWs and fibs can occur with only 20% of the surrounding muscle fibers fibrillating, and the degree typically diminishing over time, this is not an accurate parameter for determining the severity of a peripheral nerve lesion [15]. CRDs are

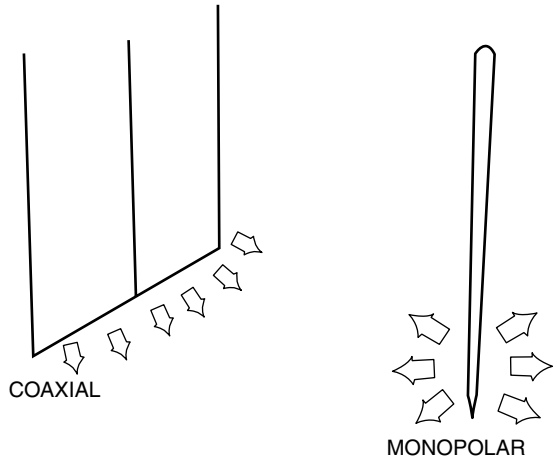

Figure 22.1 Illustrations reflecting the relative shapes of a coaxial (concentric) and monopolar needle. Of note, a concentric needle is directional and records a semicircular field, whereas a monopolar needle records a circular (360°) field.

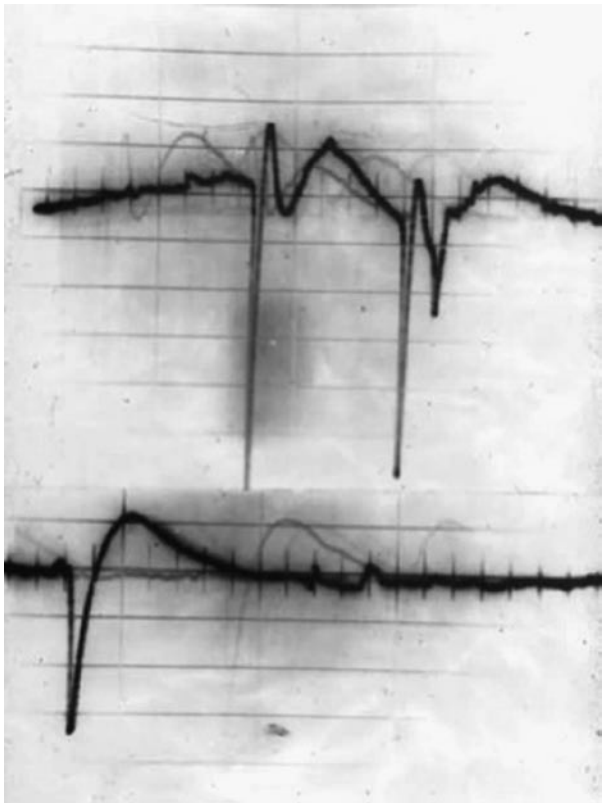

Figure 22.2 Waveforms of fibrillations (top trace) and positive sharp waves (bottom trace). Both are waveforms that result from spontaneous depolarizations of single muscle fibers.

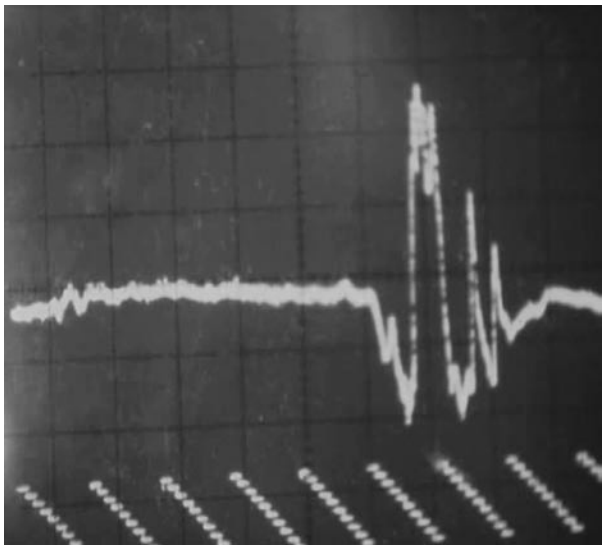

Figure 22.3 Complex repetitive discharges resulting from high frequency ephaptic activation of hyperirritable denervated muscle fibers.

often seen in more chronic conditions, and do not necessarily reflect on-going denervation [16] (Figure 22.3). Fasiculations can be seen in neuropathic processes such as radiculopathy and reflect spontaneous instability of an entire motor unit [17]. These should be identified as simple

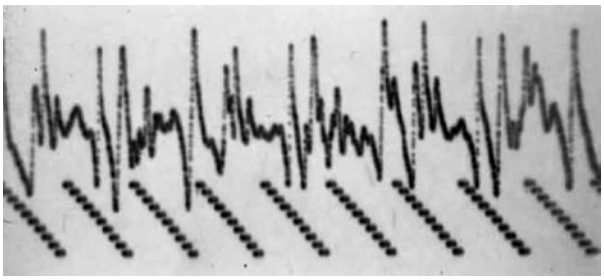

Figure 22.4 Waveform of a complex fasciculation, a spontaneous discharge of a single motor unit.

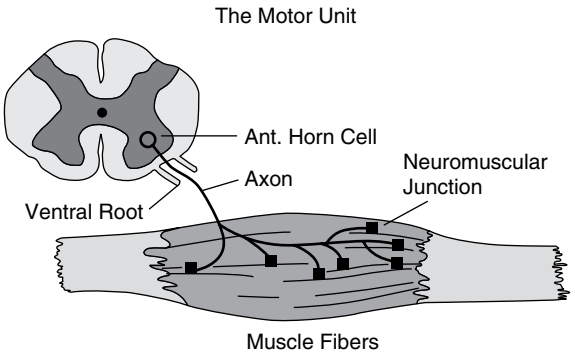

Figure 22.5 Anatomy of the motor unit.

or complex (Figure 22.4), and it should be understood that they can often be benign and not represent disease. Fasiculations should be judged by the other electrodiagnostic and clinical clues that accompany them [18].

Insertional activity reflects bursts of electrical activity with mechanical movement of the needle electrode. It is decreased in conditions of fibrosis, edema, and scar [19]. It is not appropriate to report "increased insertional activity," as this is subjective, not reliably measurable and has no clear meaning. Instead, abnormal spontaneous activity, if present, should be described.

Motor Unit Analysis

The motor unit is the anterior horn cell, peripheral nerve, and all of the muscle fibers that it innervates (Figure 22.5). The motor unit action potential (MUAP) consists of the recorded electrical activity of the muscle fibers innervated by the single anterior horn cell [20]. Motor unit recruitment assessment is often done by first evaluating the pattern at minimal contraction and then a more vigorous contraction [21]. The motor unit recruitment is reported as a frequency of the first MUAP when the second MUAP comes in at a stable rate (Figure 22.6). A normal value is generally 8 to 12 Hz. In peripheral neuropathic conditions, the first MUAP is moving abnormally fast when the second appears because of a loss of firing axons. Recruitment frequency of greater than 15 Hz is considered abnormal (Figure 22.6). The recruitment frequency abnormalities

correlate roughly with the degree of weakness present, but not necessarily axonal loss [22,23]. Recruitment abnormalities can result from axonal loss or simply demyelinating lesions with conduction block. More maximal recruitment is not necessary for defining recruitment patterns but is needed to evaluate the size and shape of the later recruited (i.e., type-II) MUAPs [24,25].

Evaluation of the size and shape of the MUAP can be a helpful clue with respect to chronicity of radiculopathy. In chronic neuropathic conditions with axonal loss, resprouting of terminal axons to denervated muscle fiber can result in large duration and amplitude MUAPs (Figure 22.7). Polyphasicity reflecting a variation in the normal MUAP territory can also be present [26]. Precise technique is needed for reliably quantifying the degree of polyphasicity in any given muscle.

Assessment of relative stability of the MUAP with trigger-delay techniques can suggest the process of reinnervation such as sprouting from neighboring axons (Figure 22.8) [27].

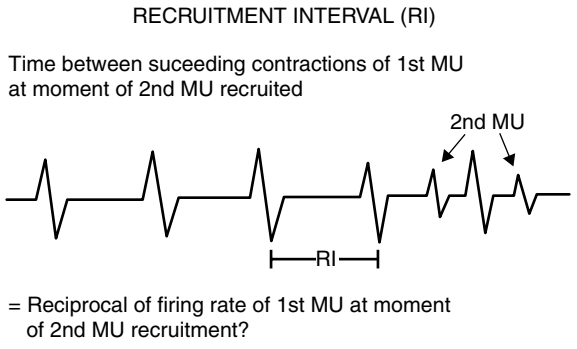

Figure 22.6 Illustration of calculation of the recruitment frequency, which is the rate of firing of the first recruited motor unit when the second motor unit comes in a stable rate. Note that the recruitment interval is the reciprocal of the recruitment frequency.

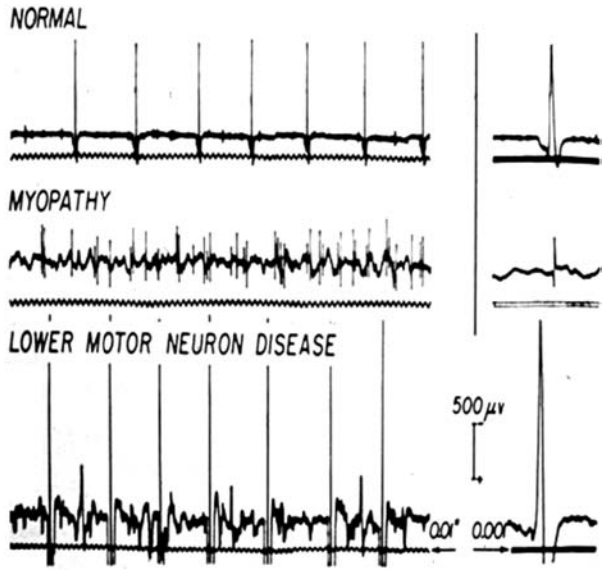

Figure 22.7 Illustration of the recruitment and waveform pattern in a normal muscle (top trace); myopathy with short duration motor units and early recruitment (middle trace) and neuropathic pattern typical in chronic radiculopathy (bottom trace) with decreased recruitment at rapid firing rates and motor unit action potential changes of increased amplitude and duration.

Nerve Conduction Studies

Nerve conduction studies (NCS) are important adjunctive techniques to needle electromyography for assessment of radiculopathy. Assessment of sensory and motor NCS provides recognition of relative severity and concomitant conditions, such as distal entrapment neuropathies, and more generalized peripheral neuropathies [28].

Motor NCS

Motor NCS are performed by depolarizing a motor nerve with electrical stimulation, and recording the response over the muscle fibers intended (Figure 22.9). This creates a compound muscle action potential (CMAP) [29] (Figure 22.10). Besides the importance of motor NCS to assess the general health of motor nerves in the patient being evaluated, the distal CMAP amplitude often reflects the integrity of the remaining axons in radiculopathy. The distal CMAP amplitude with recording over a pertinent muscle can be the single most important determinant of axonal loss and therefore relative severity. Because there is individual variation in

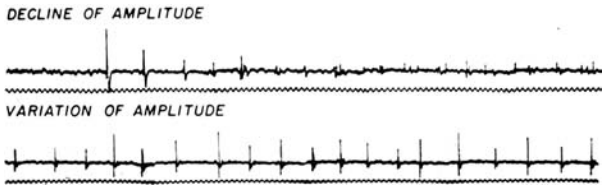

Figure 22.8 Illustration of unstable repetitively firing motor units.

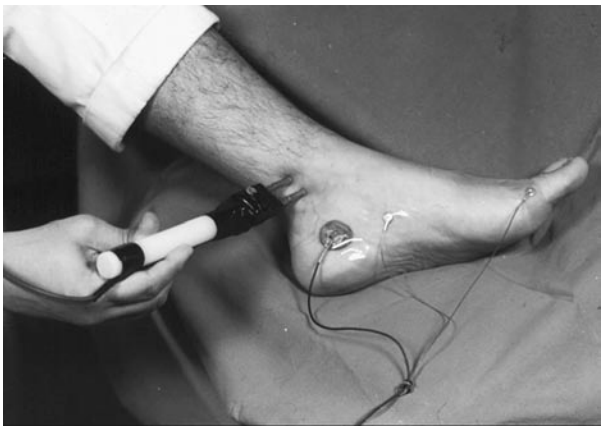

Figure 22.9 Picture demonstrating a typical arrangement for performing a motor nerve conduction study.

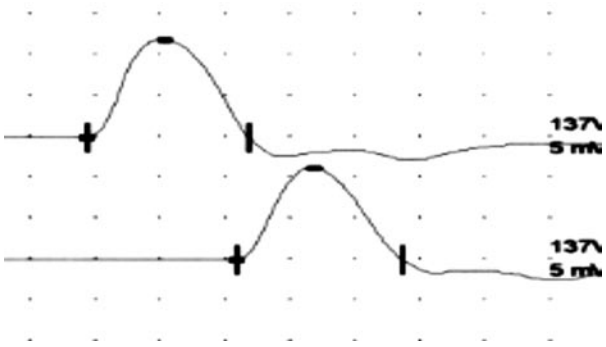

Figure 22.10 Waveforms of typical compound muscle action potentials. In each waveform, the first cursor measures the onset latency, the second cursor measures the peak amplitude, and the third cursor measures the action potential duration.

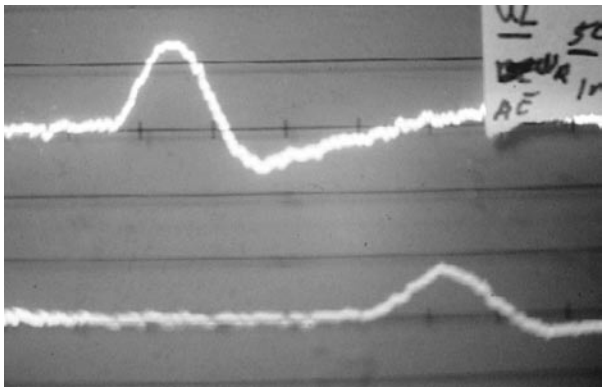

Figure 22.11 Waveforms of sensory nerve action potentials.

CMAP amplitude size for most muscles, a side-to-side comparison in unilateral radiculopathy can be helpful. When the CMAP amplitude is less than 50% on the affected side relative to the contralateral, the radiculopathy is considered relatively severe with axonal injury [30].

Sensory NCS

Sensory NCS are performed by depolarizing the sensory axonal with electrical stimulation and recording from the sensory axons at another site with recording electrodes. This creates a sensory nerve action potential (SNAP; Figure 22.11). The sensory studies are generally normal in most lumbar radiculopathy and abnormalities in these nerves could suggest a more generalized peripheral neuropathy or a focal neuropathy that is distal to the dorsal root ganglion [31].

Special Techniques

H-Reflexes

The tibial H-reflex is performed by creating a submaximal electrical impulse of the tibial nerve at the popliteal crease while recording over the soleus (Figure 22.12). This is an orthodromic measurement of the sensory (afferent) and

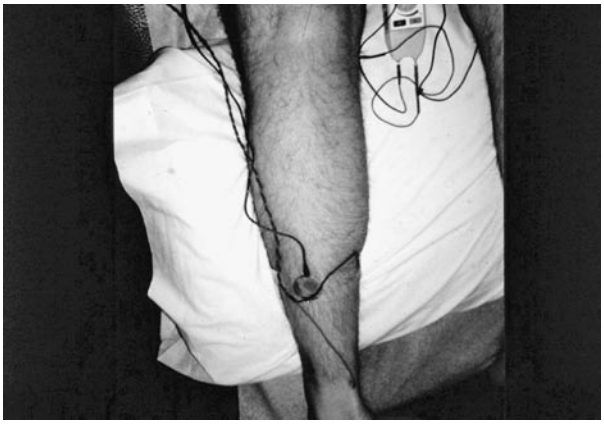

Figure 22.12 Picture depicting a typical arrangement for recording the tibial H-reflex from the soleus. The submaximal stimulation (needle electrode) is provided at the popliteal crease. The E1 recording electrode (black wire) is placed over the soleus and the E2 electrode (red wire) is placed over the tendon. The ground electrode (green wire) is placed between the stimulus and recording electrodes.

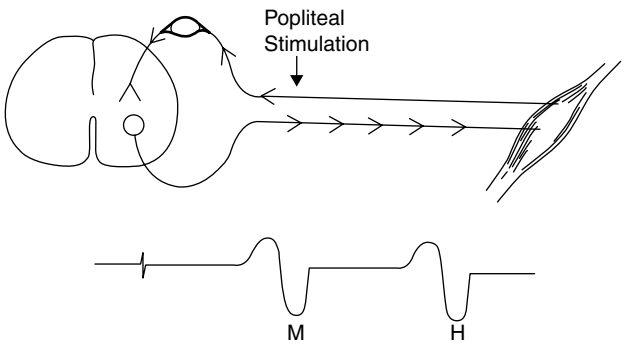

Figure 22.13 Illustration of the impulse pathway of the tibial H-reflex. The initial M response results from the direct orthodromic stimulation of the tibial muscle fibers. The H-reflex results from the depolarization of the sensory fibers with a reflex response to the tibial muscle fibers.

motor (efferent) pathway of the tibial nerve (Figure 22.13). This is felt to be a relatively sensitive technique to assess the integrity of the S1 root [32,33]. Slowing of greater the 1.0 ms on the affected side relative to contralateral suggests abnormality. This abnormality can occur immediately after onset of radiculopathy but can also persist long after relative resolution of other findings. An H-reflex, when performed properly, should have a constant latency [34] (Figure 22.14). Amplitude of the H-reflex response is not an important measured value in a conscious patient because simple relaxation and facilitation can result in dramatic variations of this parameter [35].

F-Waves

F-waves are performed with a supramaximal stimulation of a motor nerve and recording at a more distal site [36] (Figure 22.15). Unlike the H-reflex, the latency of responses

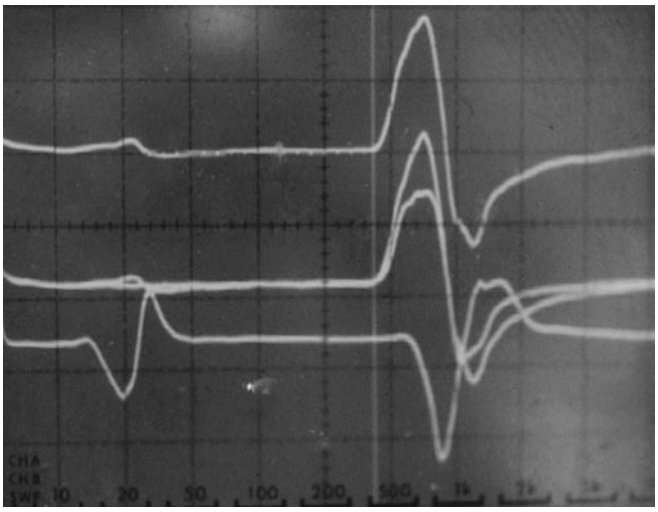

Figure 22.14 Waveforms depicting typical tibial H-reflexes. The H-reflex is seen as the larger second waveform in each tracing.

is variable; therefore, multiple responses should be evaluated with attention to the fastest, mean and persistence. The F-waves reflect recurrent discharge from the anterior horn cell. The use of F-waves in assessment of radiculopathy is controversial [37,38,39,40,41]. Because F-waves are typically more useful for assessing the long pathway of a particular nerve [42], focal radiculopathies should not be diagnosed with F-waves as the single abnormal parameter.

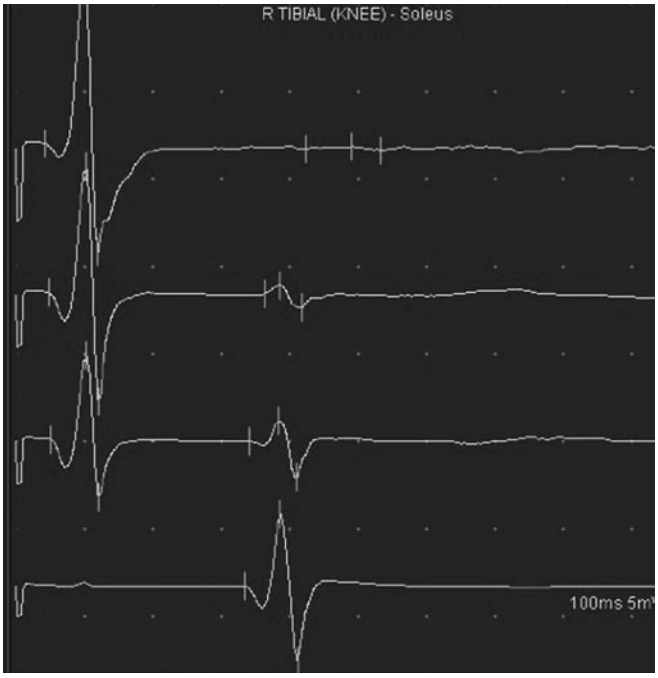

Figure 22.15 Waveforms depicting F-waves (middle traces). Note that the bottom trace is obtained with a submaximal stimulation, producing an H-reflex.

CLINICAL APPROACH TO THE PATIENT

When performing EDX studies, the practitioner needs to ensure that the techniques employed are both adequate to encompass the differential diagnosis, and appropriate to prevent unnecessary testing. As with all medical testing, EDX is only worthwhile when accompanied by an adequate history and physical examination. A reasonably comprehensive differential diagnosis should be developed prior to any testing. As EDX is a dynamic evaluation, the testing is often changed as results are obtained and the differential narrows. Sufficient testing should be performed to obtain the most precise conclusion feasible [27]. The testing should also address the differential and concerns of the referring physician.

When performing the examination, the relative comfort of the patient should be a priority. Efforts should be made to minimize anxiety. Testing should be performed in a relaxed atmosphere. The testing process should be explained in reasonable detail. Unpleasant terms such as "poke, stick, or shock" should be avoided in favor of "insert, stimulate, examine." A "pin" or "fine wire" might be preferable to "needle." Avoid showing the patient the needle as many patients associate needle length to the extent of discomfort. The patient should be positioned in an accessible but comfortable position. For minimizing pain from the needle electrode, the examiner should remain relatively superficial in the muscle and withdraw the needle somewhat prior to recruitment. There is less pain if the muscle contractions are isometric. It is also helpful to apply light pressure after needle insertion to minimize the likelihood of ecchymosis.

GENERAL APPROACH TO RADICULOPATHIES

For reliable interpretation of findings, the electrodiagnostic examiner must have an expert knowledge of pertinent anatomy and peripheral nerve pathophysiology. The time course and chronicity of findings should be well understood and not add confusion to the interpretation.

There may be circumstances in which testing for lumbosacral radiculopathy is done very early after the onset of symptoms. Within the first 7 to 10 days, there is no expectation of manifestations of Wallerian degeneration [43]. This time period is too early to expect the presence of membrane instability such as PSWs and fibs, and too early to expect a decrease in any loss of the distal CMAPs of the affected muscles [44,45,46]. For this reason, there is less sensitivity for milder radiculopathies and prognostic information is not yet present in more severe ones. EDX can provide utility in acute cases, for localization with changes in motor unit recruitment patterns and even H-reflex abnormalities. There is increased motor unit recruitment frequency in weak muscles acutely after the onset of a nerve root injury [47]. Slowing or blocking of the H-reflex also occurs acutely in an S1 radiculopathy of sufficient severity [48].

It is generally preferable to wait 2 to 3 weeks after the onset of clinical signs and symptoms of radiculopathy to maximize sensitivity of electrodiagnostic testing. At this time, the full constellation of abnormal signs, if present,

including PSWs, fibs, recruitment abnormalities, and low CMAP amplitudes, can be seen [49]. Fibrillation size has been described by some authors as a clue in determining the temporal onset of the condition, with the amplitude generally becoming smaller as the condition becomes more chronic. With this line of thinking, radiculopathy is generally felt to be acute when majority of fibrillation amplitudes are larger than 200 μV and more chronic when the majority are less the 50 μV [50]. This is believed by others to be a relatively nonspecific parameter [51]. Fibs and PSWs can be seen in both acute and chronic radiculopathies, but more frequently in subacute injuries [52].

As radiculopathies become more chronic, the fibs and PSWs may decrease significantly. If the radiculopathy is relatively mild, the electrodiagnostic abnormalities can disappear with no residual findings ultimately evident. In chronic radiculopathies that have sufficient axonal loss, the remaining viable axons often resprout to innervate denervated muscle fibers, producing large duration and amplitude, and often polyphasic, MUAPs. These larger MUAPs have been reported as early as a few weeks after the onset [53], but more typically present after a few months and can persist indefinitely, even after a complete resolution of signs and symptoms. This re-innervation can also result in ultimately larger CMAP amplitudes of the affected muscles, potentially diminishing the utility of these amplitudes for prognostic purposes in more chronic conditions [54].

Comorbid conditions must also be reliably identified in order to adequately interpret abnormal findings when investigating potential radiculopathies. Generalized peripheral neuropathies can potentially be confused with lumbar radiculopathy when an inadequate investigation is performed. Length-dependent, symmetrical neuropathies, typical for conditions such as diabetes mellitus, will often display abnormalities in many of the common distal muscles and nerves assessed in L5 and S1 radiculopathies. Sufficient sampling of both proximal and distal muscles, including paraspinals, should be performed, as well as sensory and motor NCS to distinguish these processes [55]. Comparison study of an additional limb is sometimes helpful to confirm a more generalized neuropathy. In conditions where axonal loss is present, the use of SNAP amplitude evaluation can be helpful to distinguish a nerve root injury that is proximal to the dorsal root ganglion (normal SNAP amplitude) or a lumbosacral plexopathy or other entrapment neuropathy that is distal to the dorsal root ganglion (low SNAP amplitude).

ELECTRODIAGNOSIS OF SPECIFIC RADICULOPATHIES

The determination of the appropriate level of nerve root injury is typically done by identifying abnormalities only in the affected myotome—the muscles that are innervated by that particular nerve root. Ideally, the abnormalities should be identified in more than one peripheral nerve distribution. Because most muscles are innervated by more than one root level, sufficient sampling should be done to "circumscribe" the lesion to a single myotome. Individual muscles can also be sampled both proximally and distally as well as medially and laterally in order to increase sensitivity for finding membrane instability in muscles where abnormalities are expected. More than 90% of lumbosacral radiculopathies are either L5 or S1 and there are a multitude of muscles in the lower limb innervated by these roots. There is little in the anterior rami distribution of L1 or L2 radiculopathies to reliably confirm a more rare abnormality involving these roots [56].

POTENTIAL SOURCES OF ERROR

There are many potential sources of error that can occur when performing and interpreting electrodiagnostic studies. Inadequate understanding of peripheral nerve and muscle anatomy and pathophysiology can lead to inappropriate interpretation of findings. Inexperience and a lack of skill with electrodiagnostic techniques can lead to inadequate or incorrect information resulting in false-positive and false-negative results. Other potential sources of error include: over- or underinterpretation of findings, an inadequate differential and incomplete sampling, inappropriate timing of testing, and failure to identify comorbidities. It should be understood that electrodiagnostic evaluations are the practice of medicine and should only be performed by practitioners who are adequately trained to conduct them.

REPORTING AND COMMUNICATION OF RESULTS

Emphasis should be given to the quality of the report for the electrodiagnostic evaluation. A quality study is of little value if the report does not provide adequate information to the referring physician. A precise history and physical and purpose for doing the evaluation should be provided. Tabulated data should be sufficiently available to facilitate outside interpretation and a reasonable ability to repeat a similar study at a later date. Although actual copies of the waveforms are requested in some situations, this is generally not necessary for most reporting purposes. A summary of the data interpretation is often helpful to those reading the report. The conclusion and diagnosis should be made as precisely as possible and should be readily evident on the report.

CONCLUSION

EDX is a valuable diagnostic and prognostic tool that can provide important clinical information when assessing lumbosacral radiculopathy that includes not only confirmation but also more precise localization, relative severity of the neuropathy, and identifying comorbid neurologic conditions. It is unique in that it is a physiologic assessment of peripheral nerve function as opposed to simply an anatomic assessment provided by most other forms of clinical tests. As with all medical testing, it should be considered only an extension of a careful history and physical examination and performed only by practitioners who are highly skilled in performing and interpreting EDX. Caution should be exercised to avoid making diagnostic

conclusions on single components of the evaluation and instead the constellation of clinical presentation and electrodiagnostic clues should be integrated in their entirety.

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23 Epidural Steroid Instillation for Lumbosacral Radiculopathy

Thomas R. Saullo, Stefan C. Muzin, John C. Keel, and Gregory E. Lutz

Most individuals who experience acute low back pain, with or without radicular symptoms, have resolution of symptoms without specific therapeutic intervention. Approximately 60% to 70% recover within 6 weeks and up to 90% recover within 12 weeks [1]. Patients seek further medical care when the pain is severe, it is associated with neurologic symptoms, or it fails to resolve within a reasonable timeframe. In addition to clinical resolution, anatomic changes can also be expected to resolve spontaneously with partial or complete resolution of 76% of disc herniations and 26% of disc protrusions within 1 year of symptom onset [2]. Clinical improvement often precedes radiographic improvement in the degree of disc herniation [2–8]. It is estimated that only 3% to 14% eventually require surgical decompression [2,9,10].

Use of epidural steroid injections (ESIs) may be indicated if the acute or subacute low back and/or leg pain is severe and unresponsive to oral medication, or if the pain is moderate and persistent after at least a month or more of conservative treatment. Additionally, in those patients with more chronic conditions, such as spinal stenosis, where patients have demonstrated prolonged benefits (6–12 months), ESIs can be repeated on a regular basis to continue to manage patients' symptoms conservatively [11,12]. In general, ESIs are indicated more for patients with radicular pain [13–19]; however, there may also be a role for the use of ESIs in patients with low back pain if a discogenic source is suspected [20,21]. Indications for use of epidural steroids include persistent radicular pain and paresthesias, predominant leg pain, and history and physical consistent with nerve root pain [22]. Please refer to Table 23.1 for a list of contraindications to ESI [23–25].

It is important to accurately identify patients suffering from lumbosacral nerve root involvement who may benefit from ESIs. Vroomen et al. [26] identified the positive predictors in the history and physical examination of patients with nerve root compression and radicular leg pain who exhibited a favorable response to ESIs. Age greater than 40 years and duration of symptoms (15–30 days) were the patient characteristics most associated with benefit from ESI. Significant correlation was also seen with ESIs in patients with predominant leg pain, dermatomal distribution of pain or cold sensations, and pain exacerbated by coughing, sneezing, or straining [26]. A positive straight leg raise test has also been associated with increased inflammatory mediators at the interface of the nerve root and disc, especially with sequestered and extruded discs [27]. This would imply the presence of inflammation and a rationale for use of targeted ESI.

TABLE 23.1 Epidural Steroid Injection Contraindications

Absolute contraindications
Uncontrolled bleeding/anticoagulation (INR > 1.5 or platelets < 100,000)
Local infection near the injection site
Systemic infection
Hypovolemia
Uncontrolled diabetes mellitus
Uncontrolled glaucoma
High doses of local anesthetics in multiple sclerosis patients
Cauda equina syndrome
Relative contraindications
History of laminectomy at proposed level of injection
Allergy to contrast media or local anesthetic
Immunosuppression
Pregnancy

From Refs. 23,24,25.

Patient characteristics that tend to have a less favorable response to epidural steroids have also been studied. Those with longer duration of symptoms, nonradicular symptoms, and pain not increased by activity or improved with medications were less likely to respond [28–30]. Smoking, taking an increased number of pain medications, and unemployment due to pain were also negative predictors [28–30].

RATIONALE FOR USE OF EPIDURAL STEROIDS

Patients with acute sciatica often have improvement in their symptoms 2 to 6 weeks after onset before the actual compression is relieved (resorption of disc herniation), and severe symptoms can persist despite complete surgical decompression [22]. The presence of a disc herniation on imaging studies does not necessarily correlate with clinical symptoms of a lumbosacral radiculopathy. Asymptomatic individuals often have abnormalities of the lumbosacral spine, especially with advanced age [31], which can be seen on myelography [32], computed tomography [33], and

magnetic resonance imaging (MRI) [31]. Disc herniations have been found in 40% of persons with no history of sciatic pain on postmortem examination [34]. These findings all support a biochemical component of radicular symptoms independent of nerve root compression alone.

In 1972, Macnab [35] demonstrated that mechanical compression of normal spinal nerves produced paresthesia and motor weakness but not pain. Lower extremity weakness and numbness will typically present hours or days before radicular pain [22]. The dorsal root ganglion may generate pain because of mechanical compression; however, the nerve root only begins to transmit prolonged, pain-generating discharges in response to pressure and manipulation after being sensitized by inflammation [36]. Therefore, mechanical compression of spinal nerves alone does not account for the pain associated with radiculopathy [35,37]. Pain generation in radiculopathy is multifactorial, but inflammation has been indicated as a major contributor [38,39].

Nerve root biopsies taken at surgery from patients with sciatica due to disc herniation have demonstrated histologic signs of inflammation [38]. Epidural and perineural fibrosis has been seen in patients with radicular pain, also indicating an inflammatory process [40]. High levels of phospholipase 2, a known inflammatory mediator present in the nucleus pulposus and released with disc injury, have been reported at the site of lumbar disc herniation [41]. Cytokines, glycosphingolipids [42,43], and other chemical mediators have also been implicated in the pathogenesis of inflammation and radiculopathy, particularly tumor necrosis factor-α [44]. One recent study has demonstrated promising results with epidural injections of tumor necrosis factor-α inhibitors in the treatment of sciatica [45].

ESIs may decrease this local inflammatory response and reduce radicular symptoms by several possible mechanisms. One is the inhibition of phospholipase 2, thus interrupting the arachidonic acid cascade [46]. Corticosteroids also inhibit leukocyte functions, particularly leukocyte aggregation at sites of inflammation, stabilizing lysosomal and other membranes, and preventing degranulation of granulocytes, mast cells, and macrophages [47]. Local application of corticosteroid has been shown to have an anesthetic effect by blocking transmissions from nociceptive C-fibers [48]. Epidural steroids may also stabilize neuronal membranes, thereby decreasing ectopic discharges [49,50]. In theory, ESI is superior to oral steroid dosing because it allows a higher dosage of medication to reach the desired area. Oral steroids may also be less effective in compressive lesions due to reduced blood flow [51]. The addition of local anesthetic also contributes to decreasing the inflammation [23,52–55] and improving neural blood flow [56].

GENERAL EPIDURAL INJECTION PROCEDURE

Epidural injections are generally considered safe and are usually performed on an outpatient basis. Screening for contraindications as well as a documented time out verifying the correct patient and side/level to be injected should be performed prior to any procedure. Vital signs, including blood pressure, heart rate, and oxygen saturation, are routinely monitored throughout the injection procedure, and resuscitation equipment should be readily available. Injections should be carried out after the appropriate imaging, radiographs and usually MRI, has been reviewed and the aforementioned steps completed. The success of the injection is directly correlated with correct placement of the medication at the source of the pain [57,58]. Clinicians performing these procedures should not rely solely on radiographic findings on determining where to place the injection. Patients often have multilevel radiographic findings, and clinical correlation with their pain drawings, history, and physical examination findings will improve success rates.

Choice of needle and injectate will vary with physician preference, injection technique (Figure 23.1), patient habitus, and with the presence of any contraindications or allergies. When needle placement is confirmed under fluoroscopy and satisfactory to the practitioner, contrast is injected to document epidural flow to the target area under real-time visualization (Figure 23.2). After this has been achieved, the injectate of medication can be administered simply by changing syringes. Extension tubing connected to the needle hub allows this to be performed without needle movement. A sensation of increased pressure, discomfort, or pain during injection can be experienced and may be minimized with a decreased rate of injection. Once the injection is completed, the needle may be removed and hemostasis should be readily achieved with minimal bleeding, if any. The site is cleaned and dressed with a

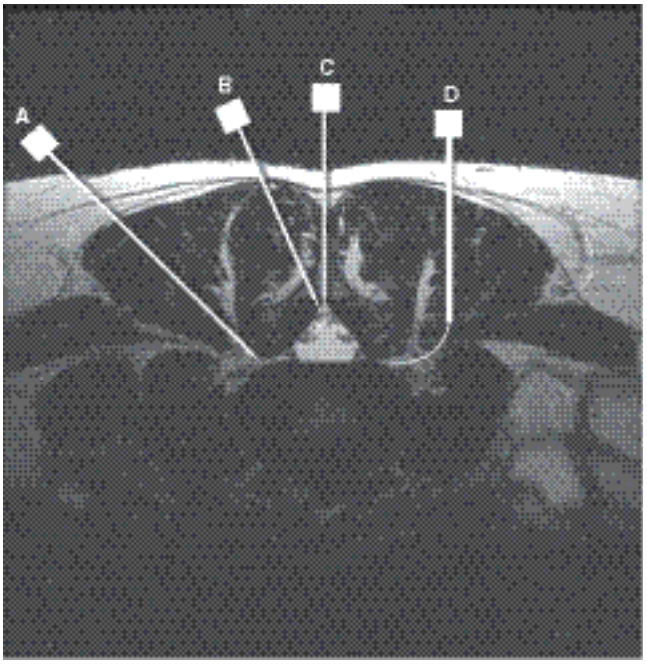

Figure 23.1 Comparison of epidural injection techniques. **(A)** Single-needle transforaminal, **(B)** parasagittal interlaminar, **(C)** midline interlaminar, and **(D)** double-needle transforaminal needle placements pictorially represented on axial MRI scan.

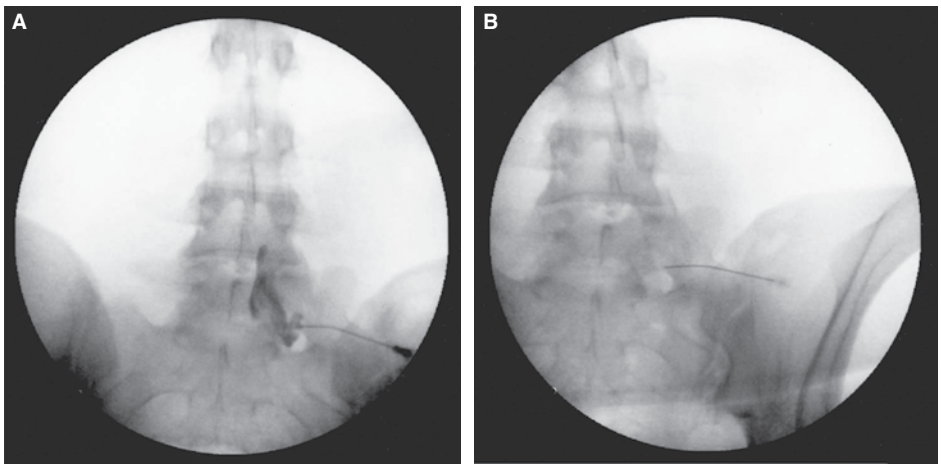

Figure 23.2 **(A)** Oblique view of L5-S1 TFESI with needle placed in the foramen, there is sacralization of the L5 vertebrae. **(B)** Epidural contrast confirms placement with no vascular uptake.

small bandage. The patient is assisted from the table and the involved lower extremity assessed for any weakness. The patient should be monitored in a recovery area for at least 30 minutes prior to leaving the site, and activities should be limited for at least 8 to 12 hours after the procedure [59]. Patients should begin to feel the full affect of the procedure in approximately 72 hours, but up to 1 week may be necessary to achieve therapeutic benefit.

CAUDAL EPIDURAL STEROID INJECTIONS

Caudal epidural steroid injections (CESI) access the epidural space through the sacral hiatus commonly located at the lower third of S4 [60]. This approach may be preferred over interlaminar injections in patients with previous spine surgery [61] and can be effective up to at least the L4-L5 level [62,63]. They are generally considered to be a relatively safe and less technically demanding epidural approach but may require a higher volume of injectate, thus diluting the steroids, to reach the lumbar region [11,64]. However, even in experienced hands, up to 40% of blind caudal epidural injections are not in the sacral canal and fail to deliver the injectate to the epidural space [10,57,65,66]. This underscores the importance of performing all these injections with fluoroscopy guidance with the use of contrast dye to confirm adequate needle placement and to ensure that the flow is not vascular.

History of Caudal Epidural Injections

Interventional spine procedures have been employed as a treatment for radicular pain since the early 1900s. Cathelin [67] is believed to be the first to use cocaine in 1901 as an anesthetic in the caudal epidural space for pain relief and obstetric deliveries. Sicard [68] and du Pasquier and Leri [69] were also reported to have performed injections into the epidural space around this time. In the 1920s, Viner and Evans [70,71] used epidural saline and procaine to treat chronic and severe sciatica.

Cyriax [72], in 1957, concluded that patients with low lumbar disc lesions, nerve root compression, and symptoms of sciatica would benefit from epidural injections. As recorded in the Italian literature, Cappio was the first to introduce epidural steroids via the caudal approach in 1957 [73]. The first reported use of caudal epidural steroids in the United States was in 1960. Brown [74] successfully treated four patients for chronic sciatica with 80 mg of methylprednisolone with complete relief of pain at 2 months. Goebert et al. [75] reported a greater than 60% relief of symptoms in 72% of patients with sciatica using procaine and 125 mg of hydrocortisone.

An early study by Coomes [76] compared two groups of 20 patients with sciatica for an average of 31 days. One group was treated conservatively with bed rest and the other received a caudal injection of 50 mL procaine. Mean recovery time was 11 days in the epidural group, with the bed rest group taking 31 days for similar relief. Neurologic deficits (strength, reflexes) also improved twice as fast in the epidural group. Similar relief was seen regardless of the level of involvement (L3 to S1), age, sex, or duration of symptoms. Although this study did not use steroids, it demonstrated therapeutic benefit from caudal epidural injections of anesthetic compared with conservative treatment.

Caudal Epidural Injection Technique

MRI should be reviewed prior to performing a caudal epidural injection to determine the sacral anatomy and the level of termination of the thecal sac [77]. The median sacral crest and the sacral cornua, which border the sacral hiatus, can usually be palpated at the cranial border of the gluteal cleft [77]. By palpating the tip of the coccyx and measuring up 2.5 in, the sacral cornua usually can be felt. Another method of finding the cornua would be to measure the

distance between the posterior superior iliac spines and making an equidistant triangle. The sacral cornua would be then palpable at the tip of the triangle. If there is still doubt, the sacral hiatus can be visualized under fluoroscopy in the lateral plane (Figure 23.3). A gauze pad is placed in the gluteal fold to prevent irritation of the perineal region from the iodine. After the skin is prepped and draped in a sterile fashion, the target area is anesthetized down to the periosteum with 1% lidocaine. Delivery of some local anesthetic through the sacral hiatus will allow for more pain-free needle placement.

This injection is commonly performed with a 22-gauge spinal needle inserted at a 45° angle of entry, through the sacrococcygeal ligament [78]. The needle should enter the sacral canal located between the sacral pedicles on anteroposterior (AP) fluoroscopy [77]. Once the needle is on bone and midline placement is confirmed on AP view, the procedure continues with lateral fluoroscopy (Figure 23.4). The needle is then withdrawn slightly, the bevel is rotated, and the needle hub is dropped into an almost horizontal orientation. The needle is advanced with a gentle twisting motion to decrease the chance of entering the bone or periosteum [78]. Aspiration should be negative for any blood or cerebrospinal fluid (CSF) and the needle tip should be below the level of S2-S3 to avoid dural puncture. The use of a shorter 2.5-in needle reduces the risk of reaching the dura.

Placement is confirmed with injection of 2 to 4 mL of nonionic contrast into the epidural space. Contrast should flow freely from the needle tip along the dural sheath and nerve roots creating a classic "Christmas tree" appearance (Figure 23.5) [77,78]. If contrast flows quickly away in a torturous course indicating vascular uptake, a small adjustment of the needle may be adequate, but in some cases redirection or repuncture may be necessary. The injectate should be administered with little resistance when placed correctly and post-injection films can document the spread of the injectate [23].

The typical injectate volume and composition for a CESI is 8 to 10 mL total volume containing 80 to 120 mg of kenalog with 6 to 8 cc of 0.5% lidocaine depending upon whether the injectate needs to reach the L5-S1 or L4-L5 levels, respectively [79].

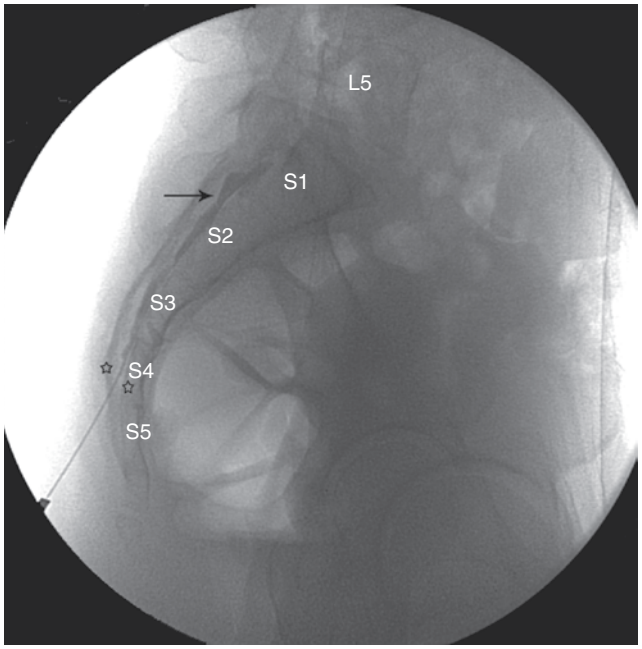

Figure 23.3 Lateral fluoroscopic view of the sacrum. The needle is inserted through the sacral hiatus (stars) and advanced into the sacral canal. Cephalad and ventral contrast flow can be seen (arrow).

Efficacy of CESIs

Early studies of CESIs have been criticized for the lack of control groups, small sample size, limited follow-up, and a lack of fluoroscopic guidance. However, there appeared to be enough success with this type of injection to maintain interest. Even patients expected to be past the usual window of natural spontaneous recovery, those with chronic low back pain and sciatica, have shown improvement with repeated caudal injections [24,80,81].

More recent studies have focused on improved study design and methodology. Two prospective, randomized studies of patients with unilateral sciatica demonstrated a positive short-term benefit from CESIs [82,83]. Patients receiving steroids reported significant pain relief and increased mobility resulting in improved quality of life at 4 weeks [82]. Mathews et al. reported improvement over the control group; however, the results were not significant. The most significant relief with treatment was noted at 3 months [83]. No statistically significant long-term benefit was shown.

Another study by Dincer et al. [84] compared CESI of 1 mL (40 mg) of methylprednisolone, 2 mL (8 mg) of dexamethasone, 7 mL of 2% prilocaine, and 10 mL of saline to nonsteroidal anti-inflammatory drugs (diclofenac 75 mg q12 hours) in 64 patients with low back pain and sciatica of a 1 to 12 month duration. The steroid group improved more rapidly and demonstrated a statistically significant improvement over the nonsteroidal anti-inflammatory drugs group in visual analogue scale and straight leg raise at 15 days, 1 month, and 3 months after injection. The Oswestry scores were also significantly improved in the steroid group at 15 days and 1 month but not at 3 months [84]. Longer durations of back pain prior to injection have been correlated with worse outcomes on the Oswestry disability index [64]. Although all of these previous studies revealed short-term benefit with epidural steroids, they were still limited by the lack of fluoroscopic guidance.

Few studies have utilized fluoroscopic guidance with CESIs. A prospective, randomized, double-blind trial in 60 patients with chronic sciatica was reported by Dashfield et al. [85] in 2005. Patients were injected with 10 mL of 1% lidocaine and 40 mg of triamcinolone via either a CESI with fluoroscopy or endoscopic injection at the nerve root. Targeted endoscopic placement of steroids failed to achieve better results than caudal injection with no significant differences identified between treatment groups. Although

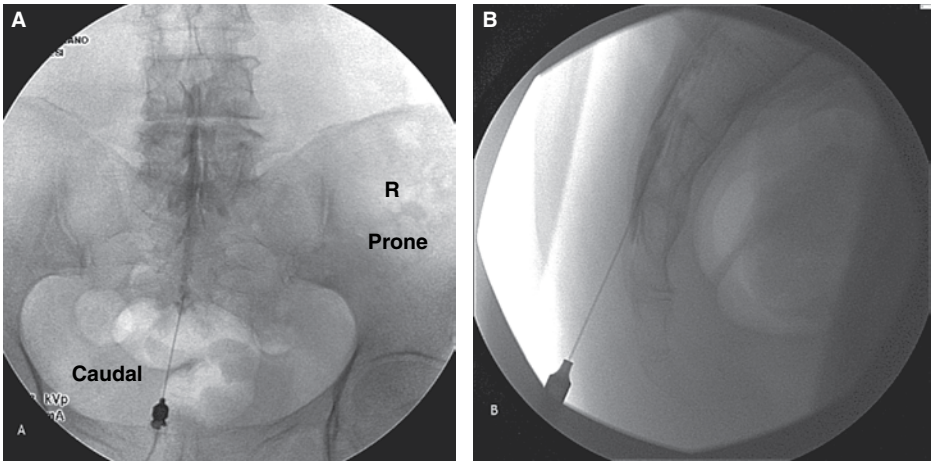

Figure 23.4 **(A)** AP and **(B)** lateral fluoroscopic images depicting correct needle placement with CESI.

both groups improved from baseline, patients receiving caudal injection had better outcomes with statistically significant improvement in visual analogue scale and anxiety at 6 weeks, 3 months, and 6 months and decreased depression at 6 months. This is one of the few studies demonstrating statistically significant improvement with fluoroscopic CESIs beyond short-term relief.

The effect of CESIs on 177 patients with radicular pain longer than 4 weeks with an MRI confirmed prolapsed disc at either L4-L5 or L5-S1 were studied by Mohammed et al. [62]. Fluoroscopically guided CESIs consisting of 40 mg of kenalog, 10 mL of 0.25% marcaine, and 10 mL of saline were performed. When treatment was effective, both radicular pain and back pain were relieved. CESI was found equally effective for both L4-L5 and L5-S1 prolapsed discs [62]. No control group was included in this study.

Manchikanti et al. [86] reported preliminary results from a randomized, double-blind trial of 84 patients with chronic (at least 6 months) disc herniation and radiculitis treated with fluoroscopically guided caudal injections with and without steroids in 2008. Injections of either 10 mL 1% lidocaine or 9 mL of 1% lidocaine plus 6 mg of betamethasone or with 40 mg of methylprednisolone followed by 2 mL of saline were performed. Repeat injections were performed when pain relief dropped below 50%. After 1 year, patients reported 50% pain relief in 79% and 81% of cases without and with steroids, respectively. Oswestry scores improved by 40% or more in 83% and 91% of cases without and with steroids, respectively. Other significant outcomes included decreased opioid use and increased employment. Improvement was seen in both groups, but the differences were not statistically significant. Both groups required an average of three to four procedures per year with 12 to 14 weeks of relief per injection after the initial two injections. This study is significant because all of the injections were performed with fluoroscopic guidance and significant improvements were demonstrated with repeated injection. It is not clear if the addition of steroids is of any increased benefit over that of anesthetic injection alone.

Safety and Anatomic Pitfalls of Caudal Injections

Fluoroscopic guidance with contrast is recommended for caudal epidural injections to ensure entry into the epidural space and lack of vascular uptake [10,57,65,66]. Problems can occur with inadvertent anterior needle placement that can puncture the intestines or pelvic cavity increasing the

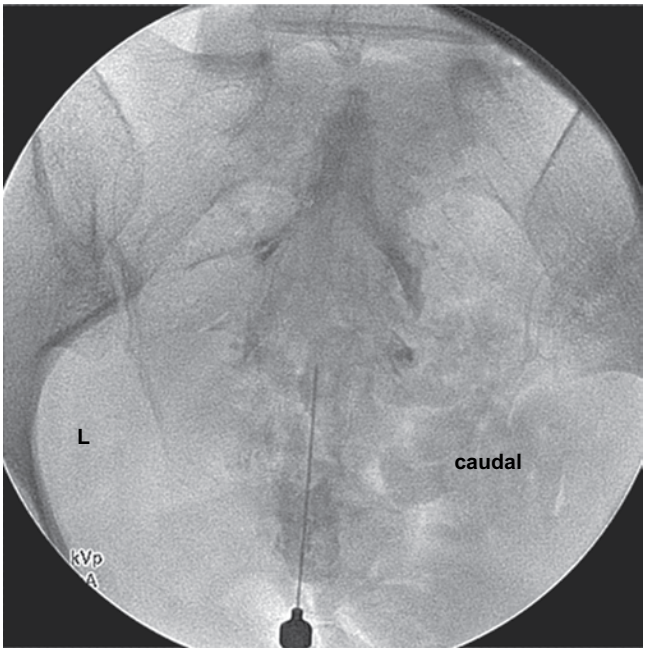

Figure 23.5 Example of AP fluoroscopic image with epidural contrast flow forming a "Christmas tree" pattern during a CESI.

risk of introducing gram-negative anaerobes [87,88]. In a small study of 10 patients undergoing caudal epidural injection, Ergin et al. [89] reported that routine intermittent fluoroscopy only revealed one intravenous contrast injection, whereas real-time imaging demonstrated four intravenous injections (40%) even after contrast confirmed needle placement. White et al. [10] found at least 6.4% of caudal needle placements to be intravascular.

Sacral anatomy varies widely and anatomic variations can cause failed or misplaced injections and complications. The sacral canal contains the sacral nerves, fatty tissue, Tarlov cysts, and the sacral venous plexus, which lies on the anterior wall and usually ends at S4 [90]. The dural sac normally ends at the middle of S2 within the sacral canal. However, caution is warranted, as Sekiguchi et al. [91] found that 20% of the time the apex of the hiatus was cranial to the S3 vertebrae, thus increasing the risk of dural puncture. Closed sacral canals can make injection difficult or impossible in 3% of patients. Other variations such as absence of a hiatus, a bony septum at the hiatus, a narrow sacral canal, and complete agenesis can be present in up to 8% of the population. These abnormalities result in an overall 3% to 11% failure rate for caudal epidural injections [91,92].

Retinal hemorrhages have also been described with large volumes of epidural injectate (20–120 mL) due to rapidly increased intracranial pressure [93–98]. We typically use no more than 10 cc total when doing caudal injections. Decreasing the rate of injection or volume of injectate may reduce the risk of this rare complication [97]. Significant recovery of vision occurs in most cases [98].

INTERLAMINAR EPIDURAL INJECTIONS

The interlaminar injection delivers the medication to the posterior epidural space, located between the dura anterior and the lamina and ligamentum flavum posteriorly. Several techniques use an interlaminar approach to access the epidural space including traditional midline, parasagittal, lateral parasagittal, and perineural approaches.

It is current practice to inject no more than two levels below the level of pathology if possible [99]. Even in the hands of experienced practitioners, up to 30% of blind interlaminar injections fail to deliver the injectate to the epidural space [10,65]. The "loss of resistance" technique can erroneously identify the epidural space when the needle tip is in the fat overlying the ligamentum flavum. Even lateral fluoroscopy can be inadequate to confirm placement without injection of contrast [100].

History of Interlaminar Epidural Steroid Injections

The midline lumbar interlaminar epidural technique was first used by Pages [101] in 1921. The lumbar interlaminar approach is more technically demanding than the caudal approach but has the advantage of placing the injectate closer to the desired level. Interlaminar epidural steroid injections (ILESIs) are preferred over CESIs if the level of pathology is above L5-S1 as caudal injections may not flow above the L4-L5 level [61]. However, one study documented an average flow to the level of L3-L4 with a 10 mL caudal injection [102]. The caudal injection, however, may be favored because it can deliver the medication to the ventral epidural space and has a reduced risk of dural puncture. In our clinical practice, the interlaminar approach is now less popular because of the increased use of transforaminal techniques for targeted placement of injectate.

Interlaminar Epidural Injection Technique

The traditional blind technique for epidural injections as described by Barry and Kendall [103] has the patient lying in the lateral decubitus position. With the use of fluoroscopy, the patient is positioned prone on the procedure table with a pillow or bolster under the abdomen in an attempt to reverse the lumbar lordosis and open the interlaminar space [99]. The level of interest is visualized, centered on fluoroscopy, and magnified. The spinous processes at the level above and below the target level should be centered between the pedicles and the vertebral endplates should be parallel in relation to the fluoroscopic image. The entry site is identified and marked on the patient followed by a sterile prep and drape of the area.

Needle placement for interlaminar injections is usually midline (within the lateral borders of the spinous processes) or paramedian (lateral to the spinous process, also referred to as a paraspinal approach). Local anesthetic, usually 1% lidocaine, is injected subcutaneously in the intended path of the spinal needle after negative aspiration. Injectate, needle gauge, and needle length vary according to clinician preference and patient habitus. Triamcinolone and betamethasone are the most commonly used steroids for ESIs and may be diluted with preservative-free anesthetic or saline.

A 20-gauge Tuohy needle is directed toward the interlaminar space under fluoroscopic guidance. The patient is instructed to alert the practitioner performing the injection if they experience any sudden increase in their pain, especially reproduction of sharp back pain or radicular pain, during the needle placement. Resistance increases as the needle tip enters the ligamentum flavum. A small amount of contrast is injected through extension tubing as the needle is advanced through the ligamentum flavum under lateral fluoroscopic guidance. A "loss of resistance" occurs as the needle enters the epidural space and placement is confirmed by epidurography (Figure 23.6) [59]. If contrast is seen only on one side of the epidural space, the needle is repositioned [104]. Post-injection films should be taken to document injectate spread. The typical injectate volume and composition for a lumbar ILESI is 6 to 8 mL total volume containing 2 mL of steroid, plus 4 to 6 mL of anesthetic agent [105].

Efficacy of ILESI

Systematic reviews of the literature have been inconclusive. In 1986, a review of ESIs [106] concluded that steroids plus anesthetic or saline was better than steroids alone, and nerve root irritation may be effectively treated regardless of the cause. A 1995 report by Koes et al. [107] reviewed 12 randomized clinical trials of which 6 reported positive

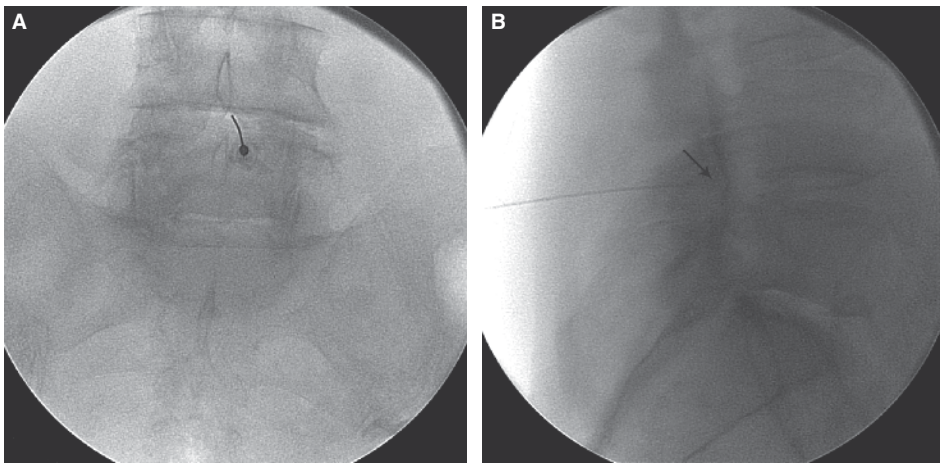

Figure 23.6 Interlaminar epidural injection. **(A)** AP fluoroscopic image of L4-L5 interlaminar needle placement. **(B)** Lateral image of L4-L5 ILESI with epidural contrast.

outcomes and 6 reported negative outcomes leaving the efficacy of ESIs uncertain. That same year, a meta-analysis by Watts and Silagy [108] of 11 placebo-controlled trials concluded that ESIs are an effective treatment option for short-term management of sciatica. They further stated that the clinical pain relief from ESIs could minimize opioid use, hospitalization, and unnecessary surgery.

In 1999, Nelemans et al. [109] reviewed 21 randomized trials of patients with low back pain for longer than 1 month and included other injections along with epidurals such as facet and local injections. The review concluded that injection therapies for low back pain needed further well-designed trials to show effectiveness. A review of invasive treatment modalities in 2006 concluded that not only epidural injection but that facet joint, trigger point, and sclerosant injections have not been proven effective and were not recommended [110]. Subsequent review articles have concluded that there is strong evidence indicating that ESIs are effective for short-term relief of pain due to disc herniation and radiculitis and weak evidence for long-term relief [111–113]. ESIs did not improve the time to return to work or the eventual need for surgery.

The most recent review of blind ILESIs for chronic radicular pain is by Parr et al. [113]. They focused on five studies (detailed later) [114–118] that met the criteria of a randomized trial providing appropriate management with outcome evaluations over at least 6 months. The authors concluded that blind ILESIs were only indicated for short-term relief of pain due to disc herniation and radiculitis.

Snoek et al. [114] compared interlaminar epidural injections with 2 mL (80 mg) of methylprednisolone or 2 mL of saline in 51 patients with symptoms and radiographic evidence of a herniated lumbar disc. Patients were symptomatic for an average of 3 months and were assessed prior to and 2 days ± 24 hours after the injections. Objective and subjective results were better in the steroid group, although the difference was not statistically significant between the groups. Follow-up was at 14 ± 6 months and again showed no significant differences between groups. Results were not recorded in the 2-week to 12-week interval after injection and any beneficial effect of steroids may have been missed. They found that a single ESI is no more effective than placebo for chronic symptoms related to herniated lumbar discs [68].

Cuckler et al. [115] studied 73 patients with radicular lumbar pain syndromes due to acute herniated nucleus pulposus or spinal stenosis in a prospective, randomized, double-blind fashion. Patients received interlaminar epidural injections between the L3 and L4 vertebrae of 2 mL (80 mg) of methylprednisolone and 5 mL of 1% procaine or 2 mL of saline and 5 mL of 1% procaine. Patients were assessed 24 hours after the first injection and given a second, nonblinded, ESI if there was less than 50% improvement in symptoms and considered failures. Anything less than a 75% reduction of symptoms was considered a failure at 3-month intervals post-injection. No statistically significant differences were found between the groups with regards to pain, need for surgery, or between diagnoses. It was concluded that the effectiveness of epidural steroids for treatment of lumbar radiculopathy remained unproven [115]. Epidural flow with interlaminar injection has been shown to reach an average of 1.28 vertebral levels cephalad and 0.88 vertebral levels caudal from the level of injection [119]. By injecting at the L3–4 level, the injectate may not have reached the most commonly involved lumbosacral nerve roots of S1 and L5. Considering this, it is possible that the patients in this study did not significantly improve because of a protocol that failed to effectively deliver the medication to the most likely level of involvement.

More recent randomized, controlled trials have demonstrated ESIs' effectiveness for short-term pain relief. In 1997, Carette et al. [116] compared ESIs with saline injections for confirmed disc herniations causing unilateral or bilateral radicular pain of 4 to 52 weeks duration in a randomized, double-blind trial of 158 patients. Epidural injections of either 2 mL (80 mg) methylprednisolone with 8 mL of saline or 1 mL of saline were used. The results revealed a mild-to-moderate decrease in leg pain at 3 and 6 weeks

post-injection and a decreased need for analgesics in the steroid group.

Wilson-MacDonald et al. [117] injected 93 patients with severe lumbosacral nerve root pain for greater than 6 weeks with MRI findings of lumbar disc herniation, spinal stenosis or both in a prospective, randomized trial. Patients received an injection of 8 mL of 0.5% bupivacaine and 2 mL (80 mg) of methylprednisolone either into the lumbar epidural space or as an intramuscular injection. The epidural group experienced a significant reduction of pain at 10 days post-injection that persisted at 35 days post-injection. Epidural steroids were found to be effective for short-term relief in patients with lumbosacral nerve root pain [117].

Arden et al. [118] demonstrated short-term benefit of ESIs in patients with unilateral sciatica of 4 weeks to 18 months duration in the WEST study. This multicentre, double-blind, randomized, controlled trial of 228 patients compared interlaminar injections with interligamentous saline injections. Epidural injections of 80 mg of triancinolone acetonide and 10 mL of 0.25% bupivacaine were performed at weeks 0, 3, and 6. The control group received 2 mL of saline injected into the interspinous ligament. A significant improvement in self-reported function and a decrease in leg pain were seen at 3 weeks after ESI compared with control.

Safety and Anatomic Pitfalls of Interlaminar Injections

Anatomic abnormalities, unknown pathology, or displacement of the spinal cord can lead to severe complications in an otherwise routine procedure. For this reason, it is imperative to obtain and view all films prior to ESI. The lateral position of the internal posterior vertebral venous plexus makes intravascular placement more common with lateral needle placement during ILESI [120]. The use of the "loss of resistance" technique has not only decreased accuracy but also increased risk. Blood flashback is an unreliable indicator of intravascular placement [10,65] and an injection of air may produce an embolus in the vasculature [93]. Fluoroscopic guidance with contrast is recommended for interlaminar epidural injections to ensure entry into the epidural space and lack of vascular uptake [121].

Contrast flows mostly in a cephalad direction but has also been shown to flow at least one segment or more in the caudal direction, independent of needle placement [122]. A 6-mL volume of contrast injected at the L4-L5 level will consistently spread above L1 and down over the sacrum [123]. Bilateral flow of injectate occurs more often with midline needle placement than lateral placement (55%–92% vs. 24%–83%) [122,124]. One reason for this may be the presence of a complete midline tissue septum in the posterior epidural space, the plica mediana dorsalis [104], limiting bilateral flow from a nonmidline injection.

Despite confirmed needle placement, injectate spread is variable between patients and with repeat injections on the same patient [61,124]. Previous back surgery has been associated with limited cephalad and caudad flow [61]. Spread to the anterior epidural space occurs with 36% to 79% of fluoroscopic guided interlaminar injections [109,122,124]. The unreliable flow of injectate to the pain generators in the anterior epidural space may account for the inconsistent efficacy seen with posterior injections.

An arterial gas embolus as a result of ILESI caused syncope, arrhythmia, cardiac ischemia, and neurologic deficit in a case described by MacLean and Bachman [125]. Hawley et al. [126] described a sudden onset of a severe posterior headache and diplopia after ILESI with the "loss of resistance" technique. The patient was found to have a subarachnoid pneumocephalus from subarachnoid injection of air that resolved on head computed tomography over a 6-day period.

Injection of large amounts of epidural air can also mimic a mass lesion as seen in a case by Ammirati and Perino [127]. The patient experienced a sudden onset of new

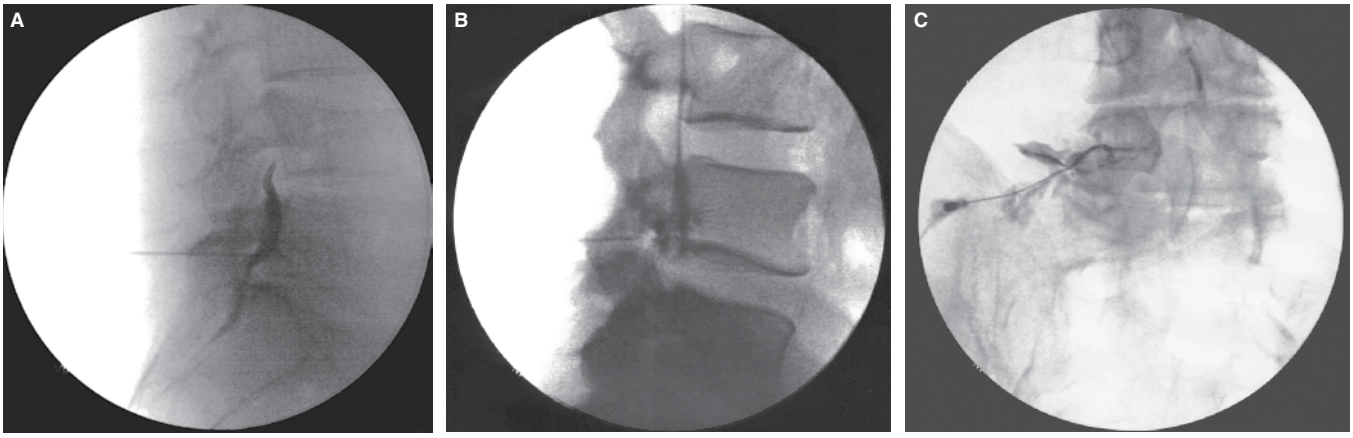

Figure 23.7 Fluoroscopic images of single-needle transforaminal injections. **(A)** Lateral image of L4 TFESI with anterior epidurogram. **(B)** Lateral image of L5 TFESI with anterior epidurogram outlining discs. **(C)** AP image of L5 TFESI with outline of DRG.

neurologic symptoms after ILESI. A pocket of trapped air was found on MRI causing displacement of the dural sac.

In our practice, ILESIs have been largely replaced by caudal and transforaminal epidural steroid injections (TFESIs) because of their theoretical and practical advantages. Future studies are needed to validate the theoretical advantages of these techniques over ILESIs.

TRANSFORAMINAL EPIDURAL STEROID INJECTIONS

The transforaminal route of injection is advantageous in that it delivers the steroid directly to the site of inflammation (i.e., the anterior epidural space in close proximity to the nerve root). This allows for the steroid solution to bathe the epidural space of a specific spinal nerve root in proximity to its dorsal root ganglion (Figure 23.7). A smaller volume and more potent injectate can be used compared with the caudal or interlaminar approaches. Another potential advantage of the transforaminal approach is that it can be very helpful diagnostically to localize the side and level of pathology by monitoring the pain responses during the injection phase and the degree of pain relief after, during the anesthetic phase [128]. In the literature, they are often referred to by many different titles such as "selective nerve root blocks," "selective lumbosacral radiculography," "periradicular infiltration," or "nerve root sleeve injections" [129].

History of TESIs

Lumbar TFESIs were first described in 1952 in the Italian literature by Robecchi and Capra [130] who reported successful pain relief in a woman with sciatic pain after an injection around the first sacral nerve root. Over the years, they were further described primarily in the Italian and French literature. There was an increasing need to accurately diagnose the site and level of the pathology since it was discovered that not all radiographic findings necessarily correlated with symptomatology [31–33]. Conversely, there were cases where no specific lesion was found on myelography, yet the patient still had severe symptoms. In addition, many patients failed to improve with surgery [131].

In the American literature, Macnab in 1971 was the first to report that "selective nerve root infiltration" can be helpful preoperatively to demonstrate the affected level in cases where myelography does not reveal any abnormality [132]. Subsequently, Tajima et al. performed selective lumbosacral "radiculography" on 106 patients with radicular symptoms. They reported that the method was safe, not technically demanding, and "very useful in determining the limit of the lumbosacral nerve root." They also commented that this technique would be useful for symptomatic relief [131].

In the early 1990s, Derby described the diagnostic and therapeutic effects of TFESIs in patients with lumbosacral radiculopathy. He postulated that this type of injection in theory delivers a high concentration of injectate directly to the posterior annulus and the ventral epidural space [133].

Derby also found that the patients' response to ESIs was highly predictive of their response to surgical decompression. They retrospectively studied patients with predominant leg pain and found that for patients with pain lasting more than 1 year, who responded to steroid injections, there was greater than 95% success rate in obtaining approximately 90% pain relief following surgery. Conversely, for patients who did not respond to steroids, their percentage of pain relief after surgery was only approximately 25%. Derby's work contributed to a more widespread acceptance and use of TFESIs in the treatment of lumbosacral radiculopathy followed by multiple efficacy studies over the years.

Transforaminal Injection Technique

During a TFESI, the patient is placed prone on the fluoroscopic table. The skin over the patient's back is then sterilely prepped and draped. Afterwards, the skin is typically anesthetized with 1% lidocaine.

Various techniques have been described, including double and single-needle techniques, and the fluoroscopic views obtained will depend on the technique used (Figure 23.8). The technique used largely depends on operator preference and comfort as well as the particular patients' anatomy. In general, if more precise localization is required, then the double-needle technique has been found to be helpful when trying to maneuver the needle into a smaller area because the smaller 25-gauge needle can be threaded through the 20-gauge introducer.

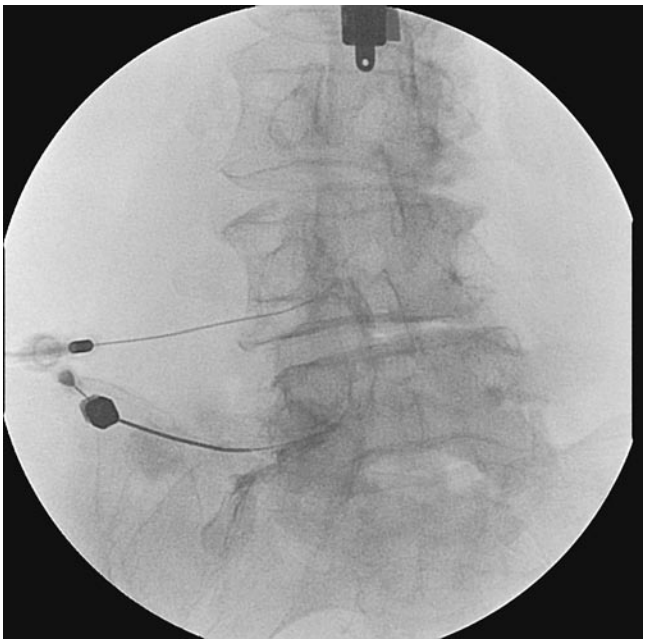

Figure 23.8 Comparison of single and double-needle transforaminal techniques. AP fluoroscopic image of L4 single-needle transforaminal needle placement and double-needle L5 transforaminal needle placement.

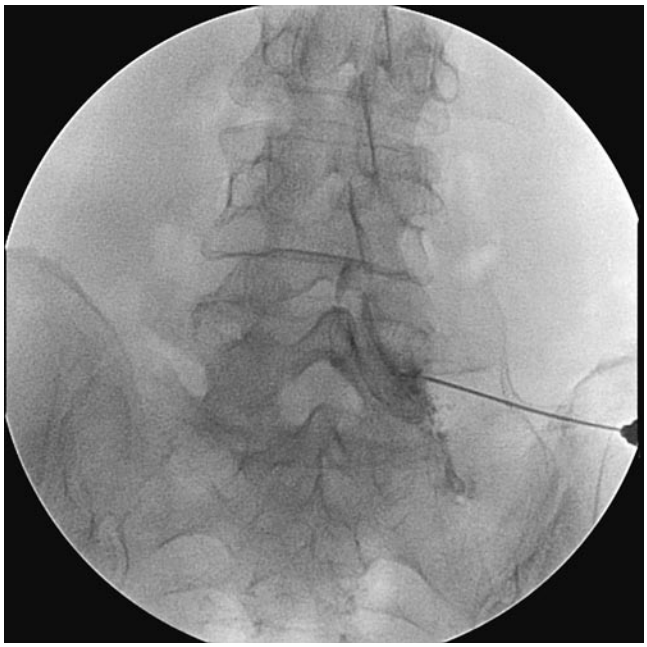

Figure 23.9 Single-needle lumbar transforaminal injection. AP fluoroscopic image of right L5 single-needle TFESI with epidural contrast flow.

If using a single-needle technique, initially oblique fluoroscopic views will be obtained to ensure that the needle is on the correct trajectory (i.e., 6 o'clock position inferior to the pedicle). As the needle is advanced, the physician usually checks an AP view to ensure that the needle has not drifted too medially (so as not to puncture the dural sac) (Figure 23.9). The position of the needle should also be confirmed with a lateral view to ensure that the needle is sufficiently, yet not too far ventral.

In our experience, with the exception of S1 transforaminal injection (Figures 23.10 and 23.11), we typically use the double-needle technique in which a 20-gauge needle introducer is advanced underneath the transverse process and lateral to the superior articulating process. Once the introducer needle is in place, we thread a curved 25-gauge needle superiorly and medially into the superolateral aspect of the neuroforamen, the so-called safe triangle [58]. The safe triangle (Figure 23.12) is composed of the base of the corresponding pedicle, the lateral border of the vertebral body, and the lateral border of the exiting nerve root. Needle placement is typically confirmed with AP and lateral views (Figure 23.13).

After aspirating to make sure there is no flashback of CSF or blood, the contrast medium is injected using

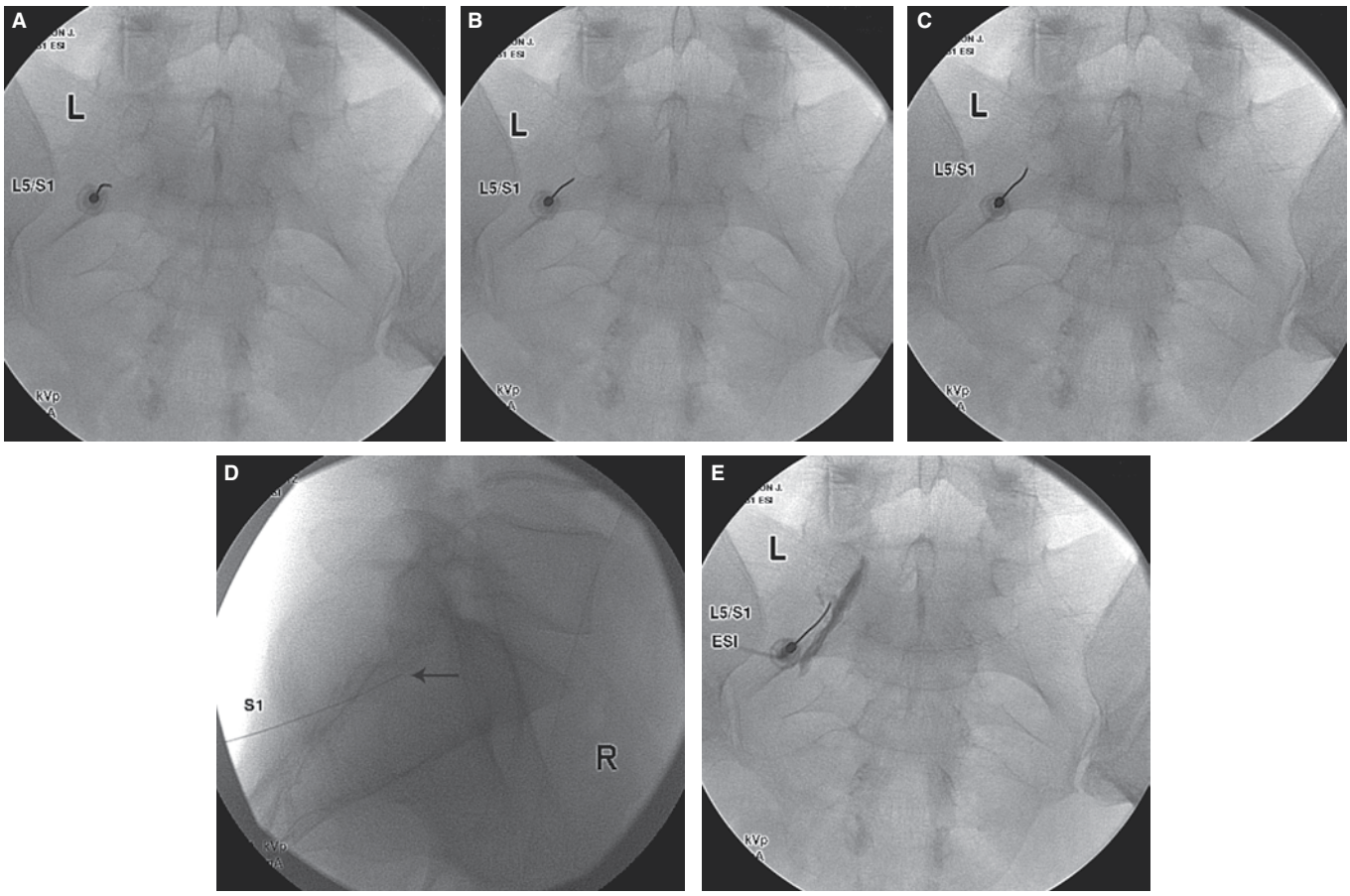

Figure 23.10 Single-needle S1 transforaminal series. **(A–C)** AP fluoroscopic image of progressive single-needle placement for S1 TFESI. **(D)** Lateral fluoroscopic image of single-needle placement in S1 foramen. **(E)** AP fluoroscopic image of contrast flow with S1 TFESI.

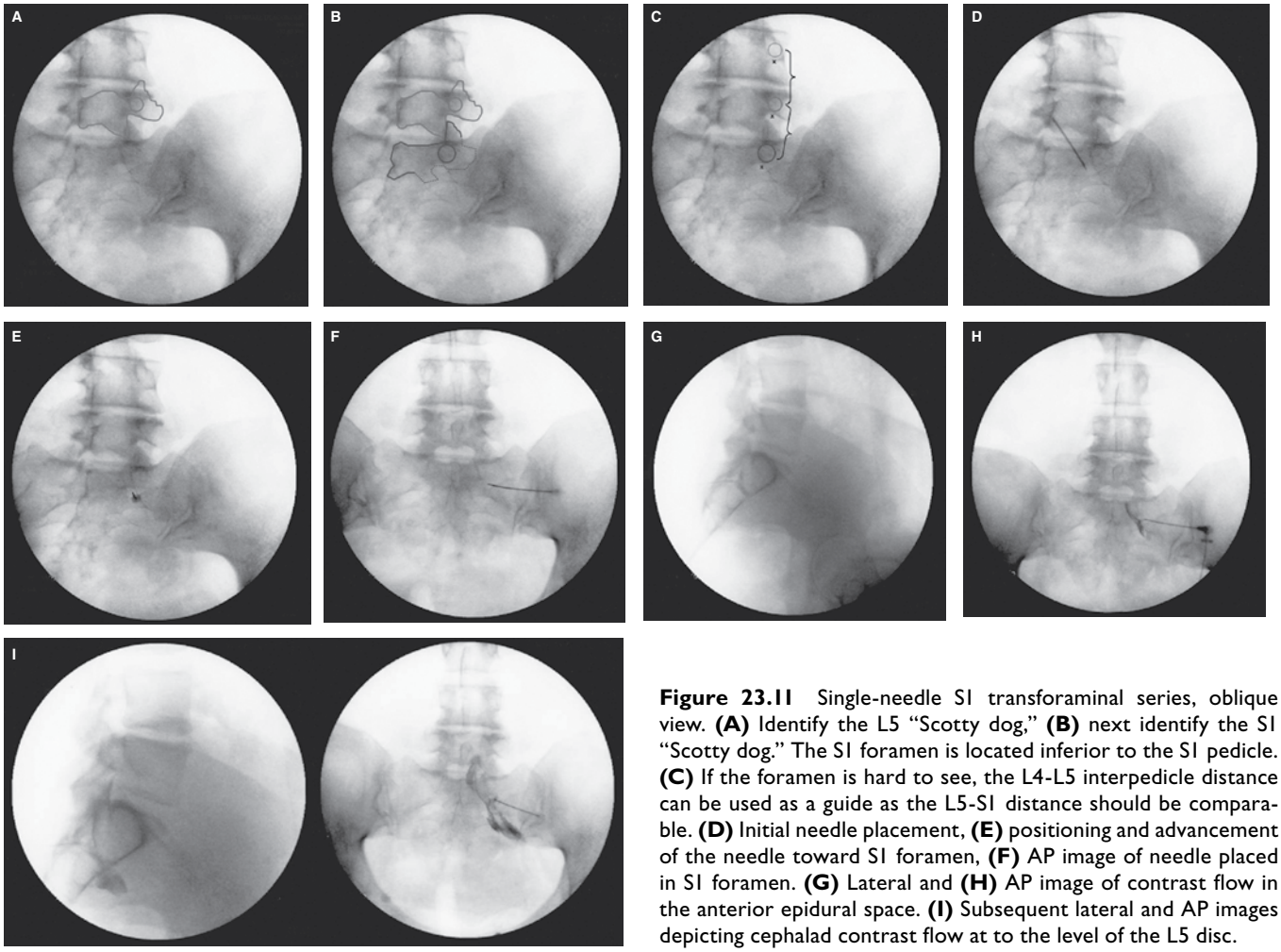

Figure 23.11 Single-needle S1 transforaminal series, oblique view. **(A)** Identify the L5 "Scotty dog," **(B)** next identify the S1 "Scotty dog." The S1 foramen is located inferior to the S1 pedicle. **(C)** If the foramen is hard to see, the L4-L5 interpedicle distance can be used as a guide as the L5-S1 distance should be comparable. **(D)** Initial needle placement, **(E)** positioning and advancement of the needle toward S1 foramen, **(F)** AP image of needle placed in S1 foramen. **(G)** Lateral and **(H)** AP image of contrast flow in the anterior epidural space. **(I)** Subsequent lateral and AP images depicting cephalad contrast flow at to the level of the L5 disc.

real-time imaging, resulting in a foraminal epidurogram. Just because there is evidence of epidural spread does not mean there was no vascular uptake. As described in recent studies [134,135], we have frequently witnessed combined vascular and epidural spread with real-time imaging that would have been missed with just a static post-injection image. Extension tubing allows for safe transfer of syringes without creating any needle movement.

A typical TFESI consists of 2 mL of steroid and 1 to 2 mL of anesthetic agent. At the conclusion of the procedure, the needle is removed and a sterile bandage is placed at the site of injection. Normally, the procedure lasts approximately 15 to 20 minutes.

Efficacy of TESIs

In one of the original outcome studies of TFESIs, Lutz et al. prospectively studied 69 patients with lumbar disc herniations and radiculopathy who presented with predominant leg pain and failed to improve with at least 4 weeks of conservative treatment [136]. The average preinjection symptom duration, however, was 22 weeks. The injections were followed by a course of exercise-based physical therapy program. They found that with an average 80-week follow-up, more than 75% reported a "successful outcome," which was defined as greater than a 50% reduction between pre- and post-injection pain scores. In addition, 78% of subjects were satisfied with their final outcomes.

Interestingly, Lutz et al. noted no significant difference between responders and nonresponders in terms of level of herniaton, preinjection pain level, age, and sex. The duration of symptoms prior to injection was found to be a statistically significant predictor of outcome. Of the patients who had pain for less than 36 weeks prior to the injection, approximately 80% had a successful outcome as compared with only 65% in patients who had suffered for more than 36 weeks.This initial study was very encouraging as the follow-up was long term (average of 20 months) and only and average of 1.8 injections were given per patient.

Around the same time period, Weiner and Fraser prospectively studied 30 patients with lumbosacral radiculopathy secondary to disc herniations (foraminal or extraforaminal) who were significantly restricted in

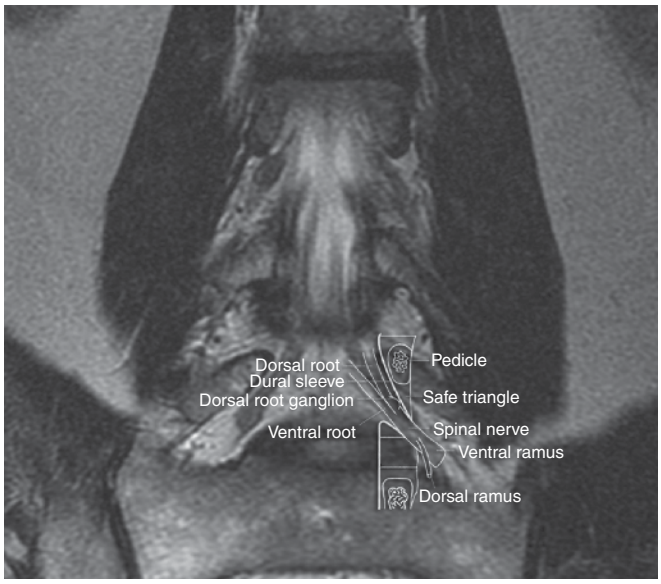

Figure 23.12 Safe triangle. Diagrammatic representation of the "safe triangle" over coronal MRI section of the lumbar spine, the target area is below the pedicle and superolateral to the exiting nerve root. With permission from [37].

their activities secondary to the pain [137]. The average follow-up was about 3.4 years. Of the 28 patients available for follow-up, 22 received long-term relief of their symptoms after undergoing one injection and only six went on to surgery. They concluded that for patients with radiculopathy secondary to foraminal or extraforaminal disc herniations, a TFESI is recommended because it provided relief in 79% of the patients.

Vad et al., in the first prospective randomized (by patient choice), controlled trial compared the efficacy of TFESIs to trigger point injections in 48 patients with lumbosacral radiculopathy [138]. The patients were followed for an average follow-up of 16 months. Their results are very impressive in that 85% (21 out of 25 patients) in the grouped treated with TFESI (average 1.7 injections) showed improvement as compared with 48% (11 out of 23 patients) in the trigger point group (average 1.6 injections). Vad et al. also found that factors associated with poor outcomes were preinjection symptoms duration of greater than 1 year as well as the presence of spondylolisthesis.

In a randomized controlled, double-blind study, Riew et al. found that transforaminal injections of betamethasone with bupivacaine prevented the need for surgery in

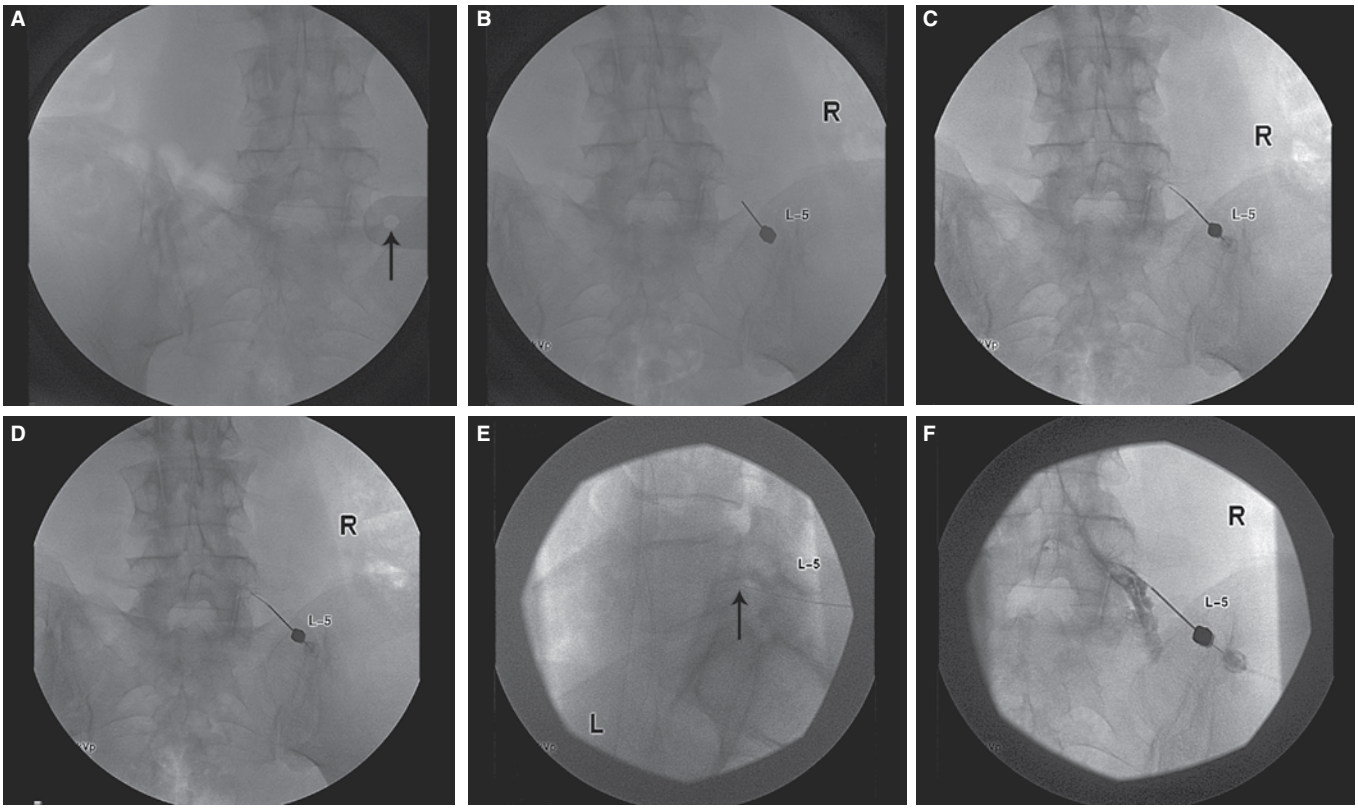

Figure 23.13 Double-needle lumbar transforaminal series. **(A)** AP fluoroscopic image of marker (arrow) used for needle placement for L5 double-needle transforaminal injection. **(B)** AP fluoroscopic image of placement of 22-gauge introducer needle for transforaminal injection. **(C)** AP fluoroscopic image of 25-gauge needle through introducer. **(D)** 25-gauge needle is advanced further toward the 6 o'clock position of the pedicle for access to the anterior epidural space. **(E)** Lateral fluoroscopic image with needle tip (arrow) in the ventral epidural space. **(F)** AP fluoroscopic image of epidural flow with L5 TFESI.

71% of their patients (20 out of 28) compared with 33% in the group treated with bupivacaine alone [139]. The aforementioned studies are very encouraging and strongly support the use of TFESIs in treating lumbosacral radiculography.

In contrast to the aforementioned studies, Karppinen et al. studied 160 patients with radiculopathy who were randomized to an injection of either methylprednisolone with bupivacaine or saline to examine its efficacy and costs [140]. They found that the combination of methylprednisolone and bupivacaine offered only short-term clinical and economic benefit as compared with saline. At 1-year follow-up, there were no differences between the groups in terms of treatment effects. However, Karppinen et al. completed one TFESI per patient which may have been subtherapeutic [136,139] and utilized epidural saline which may provide therapeutic benefit [141–143] as a control.

In a subsequent subgroup analysis, the rate of surgeries and costs were compared between the patients with contained herniation versus the patients with disc extrusions who had received TFESIs [144]. Interestingly, in the case of contained herniations, the steroid injection was associated with short-term efficacy and decreased the amount of surgeries at 1-year follow-up. In the patients with disc extrusions, the steroid seemed to increase the operation rate and overall cost treatment cost.

A recent randomized placebo controlled trial by Ghahreman et al. [145] studied both the route of administration and the injectate used for treatment of radicular leg pain due to a proven disc herniation in patients recommended for surgical intervention. They compared the effect of TFESIs to transforaminal injection of anesthetic alone and to transforaminal injection of saline. Intramuscular steroid injection assessed systemic effect and intramuscular saline injection represented placebo. The effect of TFESIs was clearly superior to placebo. The TFESI group reported at least 50% pain reduction in 54% of patients with 27% experiencing complete relief and 47% > 50% relief for at least 12 months. In addition, 50% of patients who received TFESI without initial relief reported relief after a second TFESI. These results demonstrate that the target-specific placement of steroids has an increased efficacy compared to anesthetic or saline and above that of the systemic effect of steroids. Importantly, this study suggests that a second TFESI may result in sustained pain relief even in patients unresponsive to an initial TFESI. A subsequent prospective trial is underway to evaluate the role of multiple TFESIs in curing radicular pain.

Safety and Anatomic Pitfalls of TESIs

There are a few risks specific to TFESIs in addition to the inherent risks common to all ESIs. The risk of infection from gram-negative anaerobes due to penetration of the pelvic cavity is more specific to transforaminal injections (especially S1 injections). Also, in lumbar injections, there

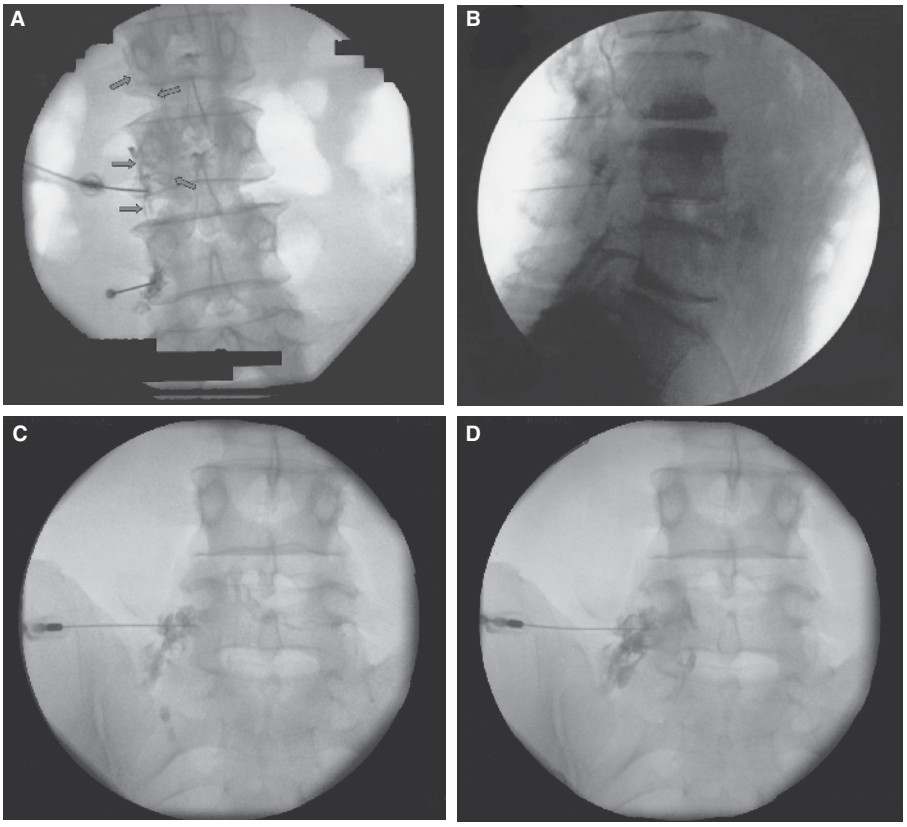

Figure 23.14 Fluoroscopic images of vascular contrast flow. **(A,B)** AP and lateral images of L3 TFESI with extensive vascular flow (arrows). AP images of TFESI with **(C)** vascular flow (arrows), then **(D)** epidural flow after repositioning of needle.

is also potential to go too far ventral or lateral and pierce the intestinal cavity [88]. This underscores the importance of the interventionalist's anatomical knowledge as well as the need to perform these injections with caution, being mindful of the depth and checking needle position on AP and lateral views. In our experience, we have found that for S1 injections, it is helpful to first advance the needle down to bone, near the inferolateral aspect of the S1 neuroforamen. This helps gauge the depth of the needle. Once this is performed, the needle can then be advanced slowly into the superolateral aspect of the neural foramen.

Other complications more specific to TFESIs include intravascular injections found in 11.2% of all transforaminal injections and up to 21.3% of all S1 injections (Figure 23.14) [134]. Smuck et al. [135] found that intermittent fluoroscopy missed 57% of vascular injections with the transforaminal approach. The artery of Adamkiewicz is often implicated in these cases as it supplies the anterior spinal artery and travels with the nerve root through the T10-T12 neural foramen in 74% of cases [146]. It is located between T9 and L2 85% of the time and 63% are on the left side [146–148]. Intra-arterial uptake of particulate steroids can have severe consequences. Cases of spinal infarction and paraplegia have been reported after TFESI with particulate steroids [147–149]. The risk of intra-arterial injection has prompted some practitioners to use dexamethasone, a nonparticulate steroid, with all TFESIs [88]. However, a recent study demonstrated that dexamethasone is less effective in treating lumbosacral radiculopathy then triamcinolone [150]. This risk can be minimized with the use of injections of live contrast prior to injecting the steroid to ensure that the flow is not vascular. At the L2 level and above, an anesthetic challenge can be administered in which the patient is queried as to the presence of lightheadedness, dizziness, metallic taste, tinnitus, and motor or sensory changes in all four limbs at 90 to 120 seconds after instillation of 0.8 cc of 2% preservative-free xylocaine. The steroid is instilled after the patient affirms the absence of any of these signs or symptoms. The use of extension tubing allows for the safe transfer of syringes so that needle movement does not occur. The use of separate syringes, one for the contrast and one for the medication mixture, is also recommended.

Direct trauma to either the spinal nerve or dorsal root ganglion can occur with transforaminal injections [88]. In our experience, avoiding patient sedation, monitoring their pain response, and not manipulating the needle once it is in the foramen have all been helpful at minimizing this complication. If the double-needle technique is used, the 25-gauge needle should first be withdrawn into the 20-gauge introducer prior to the removal of both needles. This will prevent possible scoring of the exiting nerve root during removal of the needle.

COMPARISON STUDIES OF EPIDURAL STEROID INECTION TECHNIQUES

A comparison trial of fluoroscopic guided caudal, interlaminar, and transforaminal injections deemed all efficacious; however, the transforaminal group had better short- and long-term outcomes [151]. Radicular symptoms in 90 patients with confirmed herniated nucleus pulposus on MRI were studied. Anterior spread of injectate positively correlated with increased pain relief. Contrast spread to the anterior epidural space was seen with all transforaminal injections but only 50% to 53% with the other injections [151].

Manchikanti et al. retrospectively studied and compared the caudal, interlaminar, and TFESIs for patients with chronic low back pain. The interlaminar group received the injection via "loss of resistance technique" and the caudal and transforaminal injections were done with fluoroscopic guidance. They found that all three routes of administration were effective, although the caudal and transforaminal routes were better for long-term relief. They concluded that transforaminal injections were superior to caudal and interlaminar injections as they provided longer, more effective relief and were the most cost effective [152].

TFESIs were found to be more effective than ILESIs in a retrospective study of 40 patients with radicular pain [153]. Patients reported significant short-term improvement in pain in 75% of the transforaminal injections compared with 45% in the interlaminar group. In addition, the transforaminal group underwent fewer surgical procedures in the long term [153]. Another study compared fluoroscopic TFESIs with blind ILESIs and found increased short-term efficacy with the transforaminal route [154]. The interlaminar injections were done blind to reflect common practice. In contrast, Kolsi et al. [155] did not find a significant difference between TFESIs and ILESIs in relieving radicular pain but both were considered effective. Transforaminal injections are recommended over interlaminar injections in cases where patients have tissue fibrosis or scarring which may prevent anterior spread of the injectate [156].

Candido et al. [157] described a parasagittal interlaminar (PIL) epidural approach where the injectate is delivered to the lateral portion of the interlaminar space. They randomized 60 patients with low back pain and unilateral radiculopathy to either PIL or TFESIs. With the PIL approach, out of 29 patients, they reported a 100% incidence of anterior epidural spread and 97% incidence of both anterior and posterior spread with overall decreased fluoroscopy time (28.96 s vs. 46.25 s). Only a 75% occurrence of anterior epidural spread was noted with their transforaminal injections, which is significantly less than the 100% occurrence previously reported [158]. Equivalent pain relief was achieved between techniques with a theoretically lower risk of neurologic injury with the lateral PIL technique [157]. The authors believe the PIL approach to be superior to the transforaminal approach based on their findings.

Caudal and interlaminar approaches were compared in 200 patients with back pain of which 115 also had symptoms of sciatica [159]. Fluoroscopic guidance was used with all injections and found to be necessary for accurate placement in all patients except for nonobese patients undergoing interlaminar injection. Obesity was identified as a significant risk factor for malpositioned needles in both techniques. Body mass index (BMI) has

also been correlated with the depth of transforaminal injections. A 3.5-in needle is of adequate length for a BMI under 25 kg/m^2, whereas a BMI of 25 to 30 kg/m^2 requires a 5-in needle and a BMI over 30 kg/m^2 requires a 7-in needle [160].

Based on the aforementioned studies, the literature appears to support the transforaminal approach to the epidural space as it is the most efficacious and delivers the medications more directly to the site of pathology (the anterior epidural space).

OPTIMAL TIMING AND NUMBER OF ESIs

Significant improvement in symptoms has been demonstrated if an injection is performed within 6 months of symptom onset [161]; however, there is no generally accepted consensus on the optimal frequency or number of ESIs [162]. Patient response should be assessed after the initial injection and repeat injection should be delayed at least a week to allow the medication to have a therapeutic effect [163]. Green et al. [164] reported that patients who responded favorably to ESI did so within 6 days of the injection.

Clinical trials have shown that an average of 1.6 to 1.8 injections per patient are necessary for effective treatment [136,139]. This would suggest administering at least two injections per patient prior to discarding this treatment for a particular patient. Repetition of an ESI at 2-week intervals is commonly performed and indicated if there is partial resolution of radicular symptoms [10]. A third injection is reasonable if there is a favorable but incomplete improvement after a second injection. One may consider further injections if symptoms reoccur several weeks or months later after an initial favorable response [165]. If an ESI is ineffective, other pain generators should be suspected or consideration given to the scenario that the targeted pathology is not amenable and an appropriate alternate treatment pursued [10].

Winnie et al. [166] performed epidural injections with 2 mL (80 mg) of methylprednisolone on 10 patients with sciatica. All 10 patients had at least 40% to 80% improvement of symptoms, with 80% of patients reporting complete relief. An average of 2.25 injections per patient was required to achieve this result. Furthermore, using a small volume of steroids only, they concluded that large volume injections were not needed and that the steroids themselves were beneficial [163,166]. Additional studies also found that many patients required at least two injections and that a second injection can be beneficial even if the first injection was ineffective [167,168]. Dilke et al. [168] found that a third injection after no or poor response was not beneficial.

A 2007 retrospective study by Gomez et al. [9] of ESIs included 60 patients with symptoms of sciatica for over a month and lumbar disc herniation on MRI or computed tomography. Patients received ILESIs that were repeated within 72 hours (up to six injections) if the patient did not have complete pain relief. At least three injections were required for 93% of patients and 82% required six injections. A statistically significant decrease in pain was reported at 1 and 6 months in 61% and 56%, respectively, in patients with very severe pain. They suggested that the number of injections and shortened interval between injections had a beneficial effect in their patients [9]. Cluff et al. [128] found the mean maximal number of ESIs per patient per year in academic institutions to be 4.74 ± 2.6, with a range of 0 to 20 and 6.9 ± 6.9 in private practice with a range from 3 to 40.

SAFETY AND COMPLICATIONS OF ESIs

Documented side effects of ESIs are generally minor and temporary [169–171], with an overall incidence of 5.5% [170] to 9.6% [172] with TFESIs to 15.6% with CESIs. Reported side effects include injection site pain (17.1%), increased radicular pain (0.6%–8.8%), light-headedness (6.5%), increased spine pain (2.4%–5.1%), nausea (3.7%), nonpositional headache (1.4%–3.1%), vomiting (0.5%), facial flushing (1.2%), vasovagal reaction (0.3%), elevated blood sugar (0.3%), and intraprocedural hypertension (0.3%) [169,171,172].

The reported overall infection rate with spinal injections is 1% to 2%, with severe infections being extremely rare with an incidence of 0.1% to 0.001% [60]. Cases of epidural abscess [173–175], discitis [176], osteomyelitis [177,178], and meningitis [175,179] have been reported. Introduction of bacteria, usually *Staphylococcus aureus* from the skin, can occur from an epidural injection with poor sterile technique [88]. Diabetic or immunocompromised patients are particularly susceptible to infection and require close monitoring as untreated infections can spread quickly and may result in sepsis [87,174]. Hooten et al. [180] reported the average time from injection to onset of symptoms to be 7 days and in 53% of cases the presenting symptom was worsening pain. Surgical intervention was necessary in 70% of the cases reviewed and more than half of the patients did not fully recover [180].

Trauma to a blood vessel causing bleeding and hematoma formation is a possible complication with any epidural injection technique and may not be recognized on fluoroscopy [88]. Patients with a coagulopathy, liver disease, or taking oral anticoagulants such as warfarin or clopidogrel are at increased risk for these complications [59,140]. The risk of complications is significantly increased in patients taking multiple anticoagulants [179,181]. Patients are commonly asked not to take other oral medications, such as aspirin and nonsteroidal anti-inflammatory medications, several days prior to a procedure. These medications may increase the risk of bleeding even though they are not specifically contraindicated with epidural injections [179,182,183]. In patients with established coronary artery or peripheral vascular disease, maintaining the patient on aspirin may be warranted. However, this decision must be made in combination with the patient's specialist treating the vascular condition.

Current guidelines for spinal procedures in the anticoagulated patient state the international normalized ratio should be within the normal range to ensure adequate levels of all vitamin K-dependent factors. Warfarin therapy is discontinued 4 to 5 days prior to the procedure and a preprocedure international normalized ratio is checked [182].

Thienpyridine derivatives should be discontinued to allow platelets to recover [179,182]. It has been recommended to stop clopidogrel 7 days prior and ticlopidine 14 days prior to injection [182]. Similarly, GP IIb/IIIa inhibitors should also be discontinued, tirofiban and eptifibatide for 8 hours and abciximab for 24 to 48 hours [182].

Patients are often given thromboprophylactic doses of low-molecular-weight heparin (LMWH) after surgical procedures. Procedures should be delayed at least 12 hours after low-dose LMWH and 24 hours after high-dose LMWH [179,182]. LMWH should be held for at least 24 hours post-procedure [182]. Unfractionated subcutaneous heparin for deep vein thrombosis prophylaxis is not contraindicated if the total dose is less than 10,000 units per day [182]. Higher subcutaneous doses and intravenous heparin can raise the activated partial thromboplastin time and increase the risk of bleeding [182,183]. Intravenous heparin should be held for 2 to 4 hours prior to procedure to allow the activated partial thromboplastin time to normalize, and should not be restarted until at least an hour post-procedure [179,182].

Epidural hematomas occur in approximately 1 in 150,000 epidurals and may cause compression of the spinal cord or spinal nerves [182,184]. Surgical evacuation of the hematoma within 24 hours of symptom onset can minimize nerve damage [185]. Epidural hematomas have occurred despite adherence to the guidelines outlined earlier. Renal insufficiency and advanced age may prolong the half-life of LMWH and special considerations should be taken in these cases [186].

Aspiration of blood, blood from the needle with the Valsalva maneuver, and the presence of blood on the needle are all poor indicators of intravascular needle placement [10,57,134]. All epidural injections carry a risk of intravascular injection which can only be reliably detected with the use of contrast and real-time fluoroscopy or digital subtraction angiography [88,89]. The risk of intravascular injection is doubled in patients older than 50 [187]. Real-time fluoroscopic imaging with contrast is recommended for routine use with epidural injections to verify lack of intravascular, subarachnoid, subdural, or intradiscal flow [89,118,134,187].

Despite the frequency at which intravascular, presumably intravenous, injections are seen, no significant complications or adverse effects are commonly seen with regards to injectate [10,134,187]. Intravascular injection of anesthetic can cause temporary adverse side effects with severity related to the dosage used [188]. Symptoms can include dizziness, tinnitus, disorientation, muscle twitching, metallic taste, seizures, unconsciousness, and coma [88]. Loss of medication through intravenous injection may also decrease the efficacy of epidural steroids and can occur in 11% of CESIs and TFESIs, and in 2% of ILESIs [134,187].

Compared with other steroids (betamethasone, methylprednisolone, triamcinolone), dexamethasone particles have a lower density, do not appear to aggregate, and are approximately 10 times smaller than red blood cells according to Derby et al. [189]. In theory, these attributes will decrease the risk of embolic infarcts in the case of inadvertent intra-arterial injection [189]. In contrast, particulate steroids have a larger diameter than red blood cells or form aggregates large enough to cause an embolic infarct. Particulate steroids are thought to have a longer duration of action compared with nonparticulate but Dreyfuss et al. [190] did not find this difference to be clinically significant.

Dural puncture can occur when the needle tip advances past the dorsal epidural space during interlaminar injections, or by piercing the dural sleeve around a nerve root with the transforaminal approach [88,190]. In this regard, CESIs are considered safer, with minimal risk of dural puncture [63]. Dural puncture may be recognized by flashback of CSF during the procedure. The practitioner also needs to be skilled in identifying epidural contrast flow versus subdural and subarachnoid flow as CSF flashback does not always occur, especially with transforaminal injections [88]. A CSF leak from dural puncture may cause a reduction in CSF pressure resulting in a spinal headache. These headaches are usually described as a severe, dull, constant pain located in the fronto-occipital regions, worse with upright posture and relieved in the supine position [88]. Bed rest may be indicated in these cases and a blood patch may be required if symptoms are severe or persist.

Unintentional intrathecal injection of anesthetic can cause central canal, cauda equina, and conus medularis syndromes as well as persistent paresthesias, arachnoiditis, and meningitis [88]. Subdural anesthesia may also result in urinary retention, temporary respiratory depression, ascending weakness and/or sensory loss, apnea, and unconsciousness [176,191–194].

Risk from radiation is minimal to the nonpregnant patient with use of fluoroscopic guided ESIs [87]. Individuals routinely in the room during fluoroscopic procedures are at risk for complications including cancer, sterility, cataracts, bone marrow suppression, and skin desquamation [195–197]. Manchikanti et al. [197] found that the calculated exposure outside the apron for 3000 procedures was still well within the annual allowable limits. No significant exposure was found inside the apron. Low-dose radiation is cumulative over a lifetime and long-term effects are not well understood. The physicians hands and eyes receive the most exposure to radiation [197]. Exposure can be minimized by increasing the distance from the source of radiation, decreased time of radiation, and with the use of protective shielding [199]. All staff in the procedure room should wear a lead apron, thyroid shield, and dosimetry badges [197,199]. Leaded gloves and eyewear should also be considered [198].

Corticosteroids, like most other medications, have inherent side effects. Dizziness, headache, facial erythema, myopathy, transient hypotension and hypertension, gastritis, mood swings, pruritus, insomnia, and menstrual irregularities have all been described [200,201]. Fluid retention may also occur, requiring caution with use in patients with congestive heart failure [200]. Diabetic patients may notice elevated blood glucose levels because of decreased sensitivity to insulin for up to a week post-injection [200,202]. Some authors recommend waiting 2 to 3 months between corticosteroid injections, especially multilevel injections,

to avoid causing iatrogenic Cushing syndrome [203]. Corticosteroid injection also affects the hypothalamic-pituitary-adrenal response and plasma cortisol may be suppressed for up to 3 weeks [204]. Epidural corticosteroids should not be used in patients with spinal infection, malignancy, or acute fracture. Chronic spinal fractures are not a contraindication for use of ESIs for radicular or discogenic pain [21].

Patients may also experience an allergic reaction, usually within 2 to 6 hours post-injection, to the anesthetic or preservatives they may contain [205,206]. Nonionic contrast media may also cause an allergic or hypersensitivity anaphylactoid reaction [207,208]. Gadolinium can be used, with caution, as an alternative to contrast and pretreatment with antihistamines should be considered if an allergy is suspected [208–210].

ESIs—CONCLUSION

The published studies and case reports regarding efficacy of epidural steroids have resulted in contradictory and inconclusive results. These studies have been criticized for various design flaws [25] including a lack of standardization regarding patient selection, lack of control groups, type of steroid, volume of injectate, frequency of injection, site of injection, injection technique, lack of fluoroscopic use to confirm effective delivery of injectate [20], and follow-up measures. Results should be interpreted with caution from studies of blind epidural injections as these do not represent current practice and may have reduced efficacy compared with guided injections [113].

Recent studies on CESIs utilizing fluoroscopy have been encouraging [84–86]. Statistically significant reduction in pain and depression lasting at least 6 months [87] and decreased use of opioids, improved function, and increased employment have been shown with caudal epidural injection [86]. Radicular pain and back pain are relieved in patients with disc prolapse when CESI is effective [62].

ILESIs may result in some improvement in lumbosacral radicular pain at 2 to 6 weeks post-injection [211]. In general, ESIs have not been found to have an impact on average impairment of function, return to work, need for surgery, or pain relief beyond 3 months [111–113,211]. Physicians should discuss the risks and benefits of ESI with patients and present it as an option for short-term treatment of radiculopathy from a herniated disc especially in patients who are not optimal surgical candidates [212]. Neither CESIs or ILESIs allow for targeted placement of injectate.

Patients who appear to benefit most from TFESIs are those with pain in a radicular distribution associated with either numbness or paresthesias in a dermatomal distribution or weakness in a myotomal distribution, patients with predominantly leg pain, those with positive dural tension signs on physical examination (positive straight leg raise, slump test or femoral stretch test), and those whose symptoms have been of relatively short duration.

Few studies have directly compared the effectiveness of the various epidural injection techniques. The overall literature supports at least short-term relief and possibly long-term relief from lumbosacral radicular pain with various types of epidural injections performed with various injectate. Epidural injections of saline, anesthetic, steroids, and recently antitumor necrosis factor [45] agents have all shown some degree of efficacy.

Transforaminal injections, and possibly other techniques that place injectate directly in the anterior epidural space, are thought to be the most effective injections for radiculopathy because of more specific placement of the medication at the site of pathology [200]. The potential advantages of an interlaminar injection include the ability to treat bilateral and multilevel pathology with one injection and they are technically easier to perform than transforaminal injections. Caudal injections are the least technically demanding and may be used over other approaches in patients with pathology at lower lumbosacral levels, in obese patients [151], and in patients with prior history of lumbar surgery and multilevel stenosis.

The use of epidural steroids continues to be widespread but remains controversial. The consensus at this time is that ESIs are a safe treatment option that can benefit patients with acute–subacute radiculopathy by providing short-term pain relief [213]. Currently available data support the notion that one to four injections should be considered with a minimum of two injections. As stated previously, earlier studies on ESIs were performed without fluoroscopic guidance limiting the application of the data collected via these techniques.

Additional questions warrant further investigation. Who benefits the most? What is the best medication to use? How many times should the injections be repeated? In which patients are ESIs not recommended? Are there other types of materials we could inject to create longer benefit or prohealing effects? Are there other types of delivery mechanisms that could be used to reduce the risk of dural puncture or vascular penetration? These unanswered questions underscore the need and importance for further well-designed comparative clinical trials to provide definitive evidence of efficacy and further support for more widespread use of epidural injection of corticosteroids or other medications.

However, the widespread growth of these procedures does seem to speak to the apparent clinical benefits. In many patients, these injections are a means to provide windows of pain relief so that the favorable natural history of many of these disease processes can evolve. One would assume that if physicians and patients did not see and experience the beneficial effects of these procedures, there use would be limited and not growing so rapidly. Despite this, as clinicians we need to continue to improve on the science so that patients with spinal disorders can have the maximum benefit from the fewest number of injections procedures.

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24 Therapeutic Intradiscal Procedures for Lumbosacral Radiculopathy

Guiseppe Bonaldi and Alessandro Cianfoni

HISTORICAL OVERVIEW

In the early 1900s, degenerative and traumatic intervertebral disc pathology were first established as a key cause of low back pain and sciatica, and in 1934, Mixter and Barr [1] described the herniated nucleus pulposus. Radicular pain, which is usually caused by herniation of an intervertebral disc, is a common problem with an annual incidence of 5 per 1000 [2,3]. In 60% to 80% of patients experiencing their first episode of radicular pain, the symptoms recede to a nondisabling level within a period of 6 weeks [3]. The remaining group of patients qualifies for spinal imaging, usually magnetic resonance imaging (MRI). Patients suffering from radicular pain, in which spinal imaging shows an herniated disc compressing the nerve root involved in the radiculopathy, have historically been considered possible candidates for open surgical discectomy, with the intent of providing decompression of the nerve root by removing the herniated disc. Numerous surgical treatments for discogenic pain have been developed, ranging from disc excision with laminectomy, microdiscectomy, spinal fusion, to artificial disc replacement. Because of the considerable morbidity and convalescence period inherent to conventional lumbar disc surgery, there has been an ongoing search for less invasive methods of treatment [4]. Multiple percutaneous minimally invasive interventional techniques to achieve disc decompression have been described. Percutaneous access to the disc was first used in the 1950s to biopsy the disc using needles [5,6]. In 1963, Lyman Smith, an orthopedic surgeon in Chicago, first described a minimally invasive attempt to treat sciatica through a percutaneous injection of chymopapain into the disc, with intent of achieving enzymatic chemolysis of the nucleus pulposus and of its protruding fragments compressing the nerve root [7–9]. Although this technique is still widely used in the world, reports of severe allergic reactions, and catastrophic complications following inadvertent injection of chymopapain into the subarachnoid space, have dampened the early enthusiasm for this procedure. Smith opened the path, and from the seventies onwards, many other percutaneous techniques have been proposed, involving mechanical removal of the nucleus pulposus, with different types of instruments, with or without fiberscopic vision, or different types of energy (radiofrequency [RF], laser, coblation, etc.) for the reduction-decompression of the nucleus pulposus, and of its protruding components [10–16].

RATIONALE

Spinal pain is the result of a complex interplay of mechanical, biochemical, and biomechanical processes. Radiculopathy arises from direct neural compression by disc herniations and associated inflammatory and ischemic phenomena. Symptoms can also arise from a disc protrusion because of the effect on heavily innervated surrounding structures such as the outer annulus and posterior longitudinal ligament. The severity of symptoms does not always correlate with the extent of the herniation [17]. Sensitization of the central nervous system has also been suggested to be a possible causative factor of chronicity in some spinal pain conditions. With surgical approaches there is direct visualization of the herniated disc, and removal of the portion of the disc compressing the adjacent nerve root. Percutaneous discectomy techniques attempt nerve root decompression indirectly by decreasing the central disc pressure [18]. The treatment principle of percutaneous disc decompression is based on the concept of the intervertebral disc being a closed hydraulic system. This system consists of the nucleus pulposus, containing a large amount of water, surrounded by the inelastic annulus fibrosus. An increase in water content of the nucleus pulposus leads to a disproportional increase of intradiscal pressure. On the other hand a decrease of intradiscal volume causes a disproportionally large decrease in intradiscal pressure [19,20]. Central decompression is achieved by the removal of material from the nucleus pulposus. The most often stated goal of central nuclear decompression is to lower the pressure in the nucleus and to allow room for the herniated fragment to recede inward. The theory postulates that intact outer annular fibers will be able to contract enough to reduce the tension on both the nerve root and annulus. Additional suggested effects of central decompression include denaturation and fibrotic changes in the nucleus pulposus, which should in turn limit the ability of the nuclear matrix to attract water, thereby causing a long-lasting pressure reduction [21], and reinforce the inner annular fibers, reducing the tendency of the central components of the disc to herniate toward the spinal canal [22]. Although

sounding logical, there is little proof that this phenomena actually occurs in humans in vivo. Immediate pressure drop in the nucleus pulposus with different percutaneous disc decompression techniques has been experimentally proven in animals and in human cadavers, with larger effects on well-hydrated disc without signs of advanced degeneration and volume loss [23–25]. Although an experimental increase in pressure in the nucleus pulposus of a disc with annular tear transmits increased tension forces to the outer annular fibers [26,27], there are no studies showing the opposite phenomenon of a decreased tension in the outer fibers of the annulus following disc decompression. Fibrous changes in the nucleus have been shown in rabbit discs weeks after laser disc decompression [24]. An MRI longitudinal study failed to demonstrate visible evidence of disc remodeling 6 weeks after percutaneous disc decompression [28]. Nevertheless, relief of radiculopathy has been documented even in absence of a radiographically evident reduction in total disc volume [29]. There are, however, no studies that correlate the amount of nucleus removed with a decrease in tensional pressure on the outer annulus, and no one has studied how outcome correlates with the amount of nucleus removed. On the other hand, an interesting study has showed the potential of disc decompression, performed with coblation in porcine models, to activate biochemical as opposed to morphologic changes, by promoting a favorable humoral pain mediator shift, and initiating a repair response in the disc [30]; analogous result were obtained on rabbits' discs by another group of researchers [31]. Disc decompression can be accomplished with chemical, mechanical, and thermal devices. The goal is to allow sufficient tissue removal while minimizing collateral tissue damage and avoiding destabilization of the discovertebral unit [18]. Although open surgery is effective, it has well-known disadvantages, including epidural scarring, damage to bone, denervation of paraspinal muscles with consequent lumbar instability, long postoperative inactivity, and the frequent "failed back-surgery syndrome." Patients with the latter are in fact often untreatable and severely disabled. The benefits of percutaneous discectomy are greater than just avoidance of open surgery. Small contained disc protrusions have been shown to be less likely than larger disc extrusions to undergo spontaneous resorption [32] and are associated with worse surgical outcomes following discectomy [33]. Fortunately, this is the subtype of herniation most responsive to percutaneous techniques. Finally, percutaneous disc procedures have the advantage of a high patient psychological acceptance, tolerance, and satisfaction.

It should be stated here that there are new published data [34] suggesting the potential for intradiscal procedures to accelerate the degenerative disc processes. Although the discs targeted by decompression procedures are symptomatic and already affected by some form of degeneration, and we do not know at present the clinical significance of these phenomena, this aspect should be disclosed to patients in the informed consent process. From a purely evidence-based standpoint, the percutaneous minimally invasive disc decompression procedures have stood the test of time, having been performed for more than 40 years, with overall satisfaction of performing physician and of their patients, but have not yet proven their definite efficacy in reliable long-term randomized controlled trials (RCTs). Gibson and Waddell [35] in the 2009 Cochrane Collaboration review presented the results from 40 RCTs and 2 quasi-randomized controlled trials of surgical interventions for lumbar disc prolapse. This review indicated that the place for other forms of discectomy other than traditional open discectomy is unresolved. Trials of percutaneous discectomy and laser discectomy suggest that clinical outcomes following treatment are at best fair, although the importance of patient selection is acknowledged. The authors concluded that discectomy provides faster relief from the acute attack of sciatica, although any positive or negative effects on the long-term natural history of the underlying disc disease are unclear. However, the potential medical and economic benefits are too high to justify discarding percutaneous techniques as experimental or ineffective on the sole basis of insufficient scientific proof [4,36].

Recent evidence-based guidelines from the American Society of Interventional Pain Physicians-Interventional Pain Management conclude that level II and III evidence exists supporting a variety of intradiscal therapeutic procedures for lumbosacral radiculopathy [37]. The lack of good scientific evidence rests in part on the difficulty in designing a powerful scientific trial, in consideration of the heterogeneous, and somehow subjective patients' selection criteria, the difference in techniques, and operators' skills, the control choice, and the overly subjective outcome measurements [38]. One controlled trial comparing laser disc decompression and open surgery is on its way [39]. It might be argued that open surgical techniques suffer from the same lack of high-quality scientific evidence with regard to the same pathology [40], especially in the long term, while being more invasive, carrying a much higher rate of potentially severe complications, and being more expensive. Finally, it should be noted that comparing results of percutaneous minimally invasive techniques with surgical results is probably methodologically incorrect, because the disc decompression has different indications, and targets a different type of pathology, or a different stage of the same pathology (i.e., contained disc herniations; please see later), when compared with open surgery.

INDICATIONS

Percutaneous decompression has been shown effective in relieving radicular pain and to a lesser extent axial pain from contained disc protrusions. Patient selection criteria includes the presence of a *contained disc herniation documented by spinal imaging, causing radicular pain greater than axial pain, for 6 months or longer, and the patient having failed conservative measures*, including anti-inflammatory and analgesic medications and physical therapy. Imaging and clinical correlation is of utmost importance. In doubtful cases, diagnostic selective nerve root blocks, facet blocks, and provocative discography might help target the correct pain generator. The success of the procedure depends greatly on selecting the lesions to treat (Figure 24.1): the protruding nucleus pulposus must be at least partially

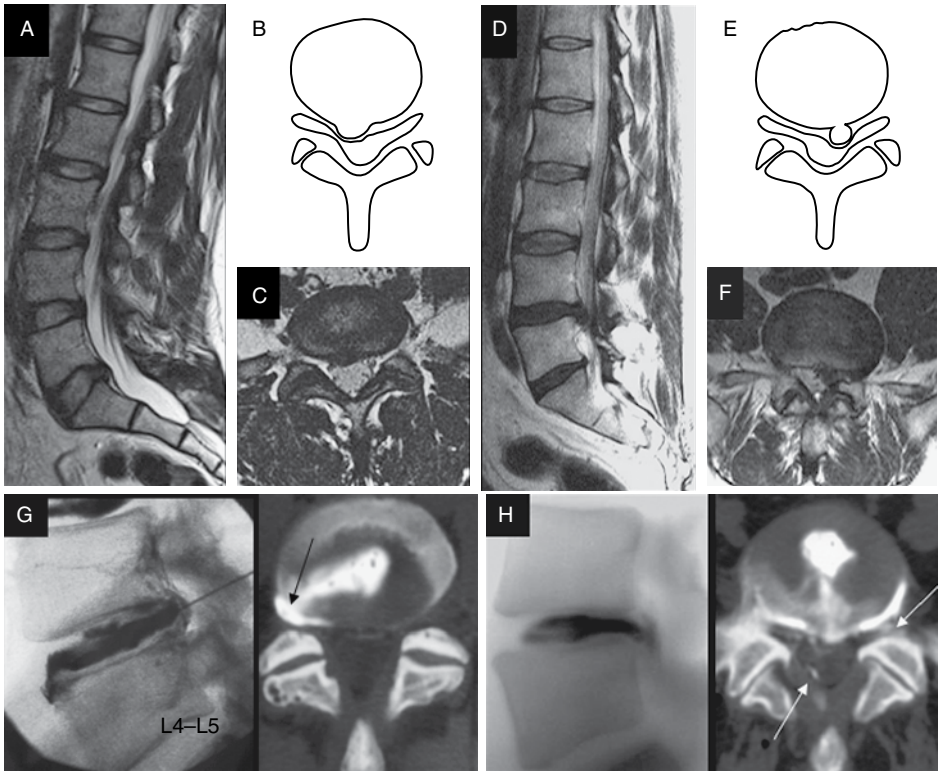

Figure 24.1 Contained and extruded disc herniations. **(A–C)** show the MR appearance and the schematic drawing of a contained disc herniation; note the broad base on axial and sagittal plane, the disc-endplates do not "pinch off" the herniation, and the disc height is preserved; in the appropriate clinical setting this might represent a good indication for disc decompression. **(D–F)** show the noncontained counterpart of disc herniation, with long sagittal dimension, and "pinched-off" aspect, that, based on morphological characteristics, is not likely to respond to a procedure of disc decompression. **(G)** shows discography and CT-discography features of a contained disc herniation (arrow), with contrast contained by the external fibers of the annulus, while **(H)** shows a noncontained disc herniation, as revealed by epidural spread of contrast injected in the nucleus pulposus (arrows). CT discography is the most accurate imaging technique to differentiate between contained and noncontained disc herniations.

contained by the external fibers of the disc, without a large extrusion or migrated fragments [41,42]. The herniation should not be pinched off by the endplates and should be without significant prolapse above or below the disc level. The disc should have maintained at least 50% of its height on imaging studies. Discs with more advanced degrees of degeneration are more difficult to access and are less likely to achieve much further pressure reduction [23]. Contained disc herniations are often circumferential bulges or protrusions, which appear broad on axial MRI or computed tomography (CT) [42–44]. Because MRI and CT do not usually enable distinction of a contained from an uncontained prolapse, in doubtful cases, discography or CT discography may help in assessing annular tears and extruded lesions, revealed by epidural spread of contrast injected in the nucleus pulposus (Figure 24.1). CT discography may also show the size of the "neck" connecting the protruded part of the disc with the central nucleus pulposus: the wider the connection, the more likely efficient transmission of pressure toward the centre of the disc, and the more likely the clinical success of the procedure [44–46].

Purely clinical criteria are also very important. Patients with a contained herniation, a good indication for percutaneous treatment, typically have a relatively long history (6 months or more) of back and/or leg pain of variable intensity, more intense under loading of the lumbar spine and particularly in a sitting position (typically, driving a car). The pain is not disabling, but becomes more and more incompatible with a good quality of life, in part because of a progressive reduction in the psychological threshold of pain tolerance. It is probable that these features correlate with a contained disc lesion, root compression becoming evident only when a static or dynamic load on the spine provokes outward transmission of pressure from the centre of the disc through rents in the inner fibers of the annulus, with secondary increase in the external diameter of the disc. The pressure within the disc and its volume decrease with rest, owing to integrity of the outer annular fibers and ligaments. Uncontained extrusions of the nucleus pulposus or sequestrated fragments, which are not a good indication for disc decompression, cause sustained, firmer compression of the nerve root (probably along with inflammatory phenomena primed by the presence of nucleus pulposus, recognized as a foreign body, in the epidural space), and therefore more constant, intense, and often disabling pain. These clinical landmarks, when

they last for at least 6 to 8 weeks, justify open surgery as the treatment of choice.

Contraindications include sequestered herniation, herniation greater than one third of the sagittal diameter of the spinal canal, progressive neurologic deficit, infection, bony deformity not allowing a safe percutaneous image-guided disc access, or other bone lesions which could compress a root and cause radicular symptoms [47]. Applying strict selection criteria, Onik estimated that only 5% to 10% of the patients with disc herniation who eventually undergo surgery would be eligible for percutaneous disc decompression [48]. Given its low morbidity, however, disclosing the lesser likelihood of clinical success of the procedure, the minimally invasive therapeutic option can be ethically offered to a wider range of patients, such as the ones with partially uncontained prolapses, as an attempt to avoid surgery, or when the risks of open surgery are higher because of age, general medical conditions, or other contraindications [48]. This typically applies to patients who have already undergone open surgery at the same level, because of the possibility of symptomatic epidural scar, and to elderly people. In fact, an observational study on a large cohort of patients reports these subgroups of patients as good responders to automated disc decompression (APLD) [22]. Although the satisfactory outcome can be attributed to several factors, the one that supersedes all is that in these patients, with a nerve root confined and compressed in a small space, either due to epidural fibrosis or arthropathic degenerative bone changes, even a small reduction in the volume of the disc by disc decompression might result in radicular decompression and clinical improvement.

TECHNIQUES OF PERCUTANEOUS LUMBAR DISC ACCESS

Fluoroscopic Guidance

To safely perform intradiscal lumbar procedures, adequate anteroposterior (AP), laterolateral (LL), and oblique fluoroscopic views must be obtained. These characteristics can be offered by C-arms, single-plane or biplane angiography units. By no means, one should undertake these procedures with the aid of only a fixed fluoroscopy unit. The fluoroscopy table should be completely radiotransparent.

AP and LL Views of the Disc Space

For the interventionalists and radiology technologists, approaching the fluoroscopic-guided spine procedures, the concept of *AP and LL views* should be reshaped. The spine has natural lordotic and kyphotic curvatures along the sagittal plane, as well as possible additional curvatures and rotations on the coronal and axial plane, which can be due to conformational deformity, such as in scoliosis and rotoscoliosis, or due to the patient positioning on the fluoroscopy table. Therefore, the classical 0° AP position of the tube defining AP view, and 90° LL position defining LL view, should be completely abandoned, and substituted by level-specific AP and LL views, that is, the AP views of L2-L3 and L4-L5 disc spaces will certainly have different tube obliquity (Figure 24.2). These views are irrespective of any predetermined tube angulation, and of the patient's body positioning, and should only be defined by the actual fluoroscopic appearance of the vertebral body at the level

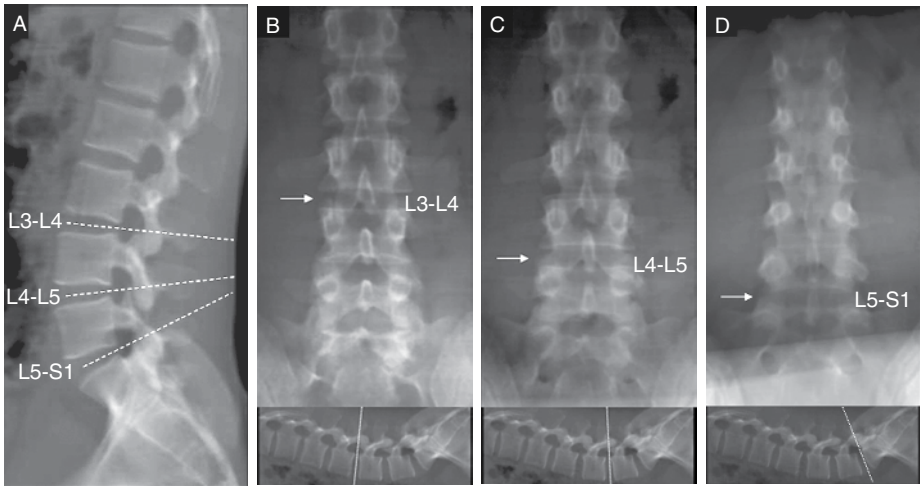

Figure 24.2 Fluoroscopic views of the disc spaces. **(A)** lateral (LL) view of the lumbar spine showing the different and individual degree of craniocaudal (CC) obliquity of the axis of the disc space at different adjacent levels. **(B–D)** show three different anteroposterior (AP) views of the lumbar spine, acquired with different CC angles of the tube (shown at the bottom), to profile the L3-L4 **(B)**, L4-L5 **(C)**, and L5-S1 **(D)** disc spaces. Note on **(B–D)** the exact midline position of the spinous process, the profiled disc-endplates, and the clear visibility of the disc space at the level of interest, defining a correct AP view, and on **(A)** the well-profiled disc-endplates, posterior vertebral walls, foramina, and superimposed posterior elements, defining a correct LL view. Obtaining correct AP and LL views at the level of interest is a crucial prerequisite to perform precise and safe image-guided procedures.

of interest. Two axis of tube angulation are required to obtain AP, and two axis to obtain LL views. The percutaneous access to the disc requires precise visualization of the disc space in orthogonal AP and LL, and oblique views.

The *AP* view of the disc space is defined by a right-to-left (RL) (axial) tube angulation in which the spinous process is projected exactly along the midline of the vertebral body, and by a craniocaudal (CC; sagittal) tube angulation in which there is profiling of the disc-endplates as two single lines, and the disc space is open and clear, at the level of interest (Figure 24.2).

The *LL* view of the disc space is defined by a RL (axial) tube angulation in which the posterior articular processes, and at the thoracolumbar junction the ribs, are superimposed, and the posterior vertebral walls of the relative vertebral bodies are profiled as two single lines, and by a CC (sagittal) tube angulation in which there is profiling of the disc-endplates as single lines, the pedicles are superimposed, the neuroforamina are seen as one, and precisely contoured, and the disc space is open and clear, at the level of interest (Figure 24.2).

It should be noted that the aforementioned general rules apply to vertebrae of normal morphology and alignment, and that there need to be major fluoroscopy tube obliquity adjustments of those views, difficult to standardize, when some form of deformity is present, such as in the case of severe scoliosis. Once the regional fluoroscopic anatomy at the level of interest has been precisely defined by obtaining strict AP and LL views, the tube angle parameters should be saved or recorded, on those units that allow it, to find these projections during the procedure, in a simple and fast manner.

The disc space of interest should be placed in the exact center of our fluoroscopic field of view, to avoid that the *parallax* effect created by the fan-shaped x-ray beam coming from a point source lead the viewer during the procedure to misjudge the position of the needle tip. The maximum possible collimation of the fluoroscopic beam should be applied, to obtain a better visibility of the region of interest, and to minimize irradiation of the patient and of the operator. Magnification of the image can be applied as desired.

Oblique View for Disc Access

The most reliable, standardized, and safe technique to perform percutaneous fluoroscopic-guided lumbar disc access implies the use of the so-called *eye-eye*, or *bull's eye*, or *tunnel vision view*. According to this technique, the fluoroscopy tube is angled along the projected path of the needle from the skin to the desired final position of the needle tip within the disc, with an oblique posterolateral paravertebral approach. To identify the correct angle and obliquity of the access, from a true AP view of the disc space of interest, the tube is angled obliquely, toward the side of the preferred approach, under continuous fluoroscopic view, until the anatomical landmark of the "Scottie dog" appears, with its ear (the superior articular process of the vertebra below) superimposed on the disc space. The target point is in the middle of the disc space, as seen on this oblique projection, just lateral and anterior to the superior articular process. The position of the superior articular process along the disc-endplate is the determinant of the obliquity of the disc access, and eventually of the position of the needle's tip in the disc space. A degree of obliquity such as that the ear of the Scottie dog appears to bisect the projection of the disc-endplate line, and ensures a final position of the needle's tip in the exact center of the disc, along midline; a more external position of the Scottie dog's ear predicts a more peripheral and ipsilateral final position of the needle's tip, along with a higher chance to hit the exiting nerve root. A more medial position of the ear of the Scottie dog along the disc-endplate allows a more posterior final position of the needle's tip within the disc, along midline, but increases the risk of straying in the epidural space, and potentially entering the dural sac (Figure 24.3). Eventually, the degree of obliquity and the trajectory of the needle as tangent as possible to the ear of the Scottie dog determine the safety of this trajectory in avoiding the exiting nerve root, usually located superior and anterior to the needle's entry point in the annulus. Once the specific oblique projection has been identified, the entry point of the needle in the skin projects over the target, and the needle is inserted accordingly to the tube angle, and along its whole path, from the skin to the target, it will appear as a single radiopaque dot superimposed to the target. Of course, as always in radiology, the position of an object must be confirmed in two orthogonal projections; in this case, the depth of the needle tip, and its final correct position on the target, must be controlled intermittently, and finally confirmed, by the two correct AP and LL views, as defined earlier. Strict adherence to this methodology guarantees the safest and most reproducible needle approach to the disc.

Specific Technique of Percutaneous Access to the Disc from L1-L2 to L4-L5

The procedure can be performed with the patient in either the prone or lateral decubitus position. When the prone position is used, bolsters are placed underneath the patient's abdomen to flatten the lordosis and open the disc spaces posteriorly (Figure 24.4). This will allow easier disc access and better transmission of the pressure drop caused in the center of the disc by the decompression procedure, to the herniated disc component. For the same reason, the patient is flexed when in the lateral decubitus position, by positioning a bolster under the recumbent side, approximately at the level of the disc to be treated (Figure 24.4). The entry route, as described earlier, is posterolateral. Correct positioning of the guiding needle in the disc is the most delicate part of the procedure and is crucial to the result. The needle must be placed with its tip in the midline, at the junction of the middle and posterior thirds of the disc, where the normal nucleus pulposus lies. In cases of large, posterior protrusions indenting the spinal canal, it is preferable to aim for a more posterior position of the needle; therefore, a more oblique view, with the ear of the Scottie dog located toward the medial end of the

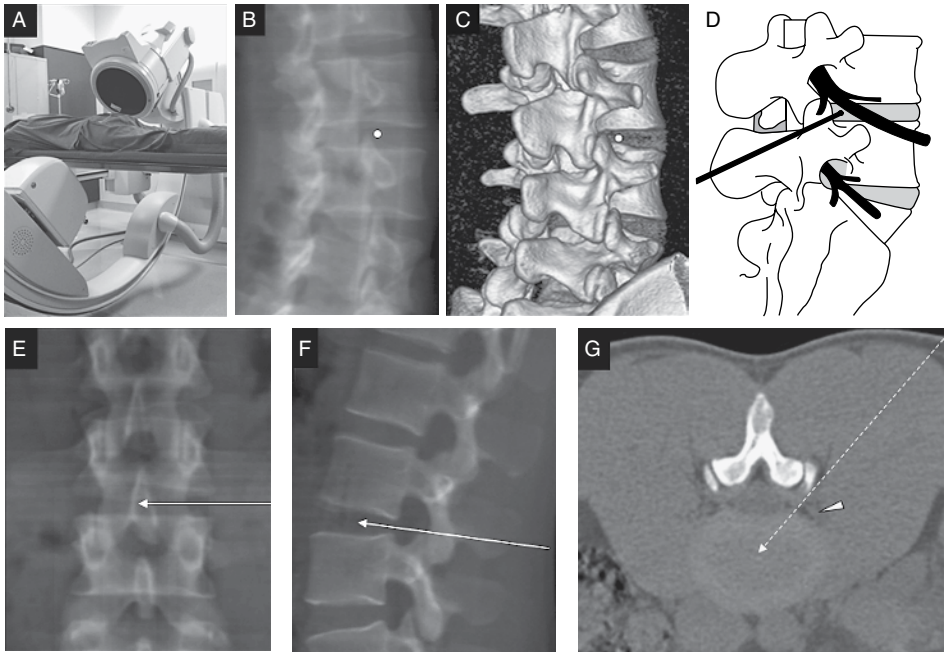

Figure 24.3 Fluoroscopy and CT correlates of disc access anatomy. **(A)** shows the fluoroscopic tube angle to obtain a correct eye-eye (EE) view for a right posterolateral percutaneous access to the disc L3-L4 **(B)**, as also shown on the correspondent 3D volume rendering CT model **(C)**. The CC angle of the tube is such that the disc space is well profiled at the level of interest, and the right-to-left (RL) obliquity is such that the superior articular process (ear of the Scottie dog) of L4 is superimposed on the midpoint of the disc-endplate line. This ensures an access-window for the needle (white dot on **B** and **C**) posterior, inferior, and medial to the exiting nerve root **(D)**. **(E,F)** show the final location of the needle (arrow) in the center of the nucleus pulposus on the AP and LL views. **(G)** shows the axial CT section through the disc space, and the ideal needle path (dashed arrow); note that steep obliquity, tangent to the superior articular process of the facet, is necessary to have a correct access to the disc, and to avoid the exiting nerve root (arrowhead).

disc-endplate line, is chosen. The most effective and safest way to accurately place the needle, as mentioned earlier, is to choose a path that passes laterally tangent and then anteriorly to the superior articular process of the zygapophyseal complex (Figure 24.3). Once the skin entry point has been marked under fluoroscopy, overlying the target point on the oblique fluoroscopic view, a skin wheal is raised. Immediately thereafter, a 22- to 25-gauge × 12- to 15-cm spinal needle is inserted and aimed just slightly more posteriorly than the anticipated route to the disc, aiming to touch the bone of the superior articular process of the facet. The bone contact is the depth control in this maneuver, assuring that the anesthetic will be delivered superficial to the foraminal space, in order not to numb the nerve root. It is critical that local anesthetic is not deposited anterior to the articular processes, because this would neutralize the warning role that radicular paresthesia has for the performing physician, alerting an undesired, too close proximity of the needle tip to the nerve root. Local anesthetic is then injected through the needle and the needle is gradually withdrawn toward the skin, allowing for anesthetization of the underlying spinal musculature.

The spinal needle of small caliber used to deliver local anesthesia can also be used to test if the trajectory from the skin to the annulus is accurate. Choosing which side to enter the disc depends upon the side of the symptomatic contained herniation. When the disc herniation has a broad base and encompasses both sides, the side of the patient's symptoms should be chosen. There are two compelling reasons to do so: first, to place the needle tip as close to the herniation as possible, particularly when the herniation is lateralized; second, to avoid the possibility of bilateral symptoms in the event of complications. In rare circumstances, a contralateral approach is preferred, such as when a correct needle positioning inside the disc is impossible from one side. That situation can arise when the needle repeatedly abuts the exiting nerve root, precluding safe advancement into the annulus, such in cases of conjoined nerve root, a flattened root compressed by the disc herniation, or in case of other anatomical variants. When a conjoined nerve root is present, both roots can exit from a single foramen, and one root can take a more caudal and inferior course, which places it in the line of a correctly placed needle. In case of a far lateral herniation, it is possible that the nerve root is in an abnormal position and pushed posteriorly resulting in obliteration of the space between the nerve and the anterior surface of the facet. An ossified bridging posterolateral osteophyte might also make the disc access impossible from one side.

The procedure is monitored fluoroscopically, using the oblique view, and advancing the needle along the eye-eye trajectory. Intermittent fluoroscopic control in the LL projection confirms that the needle is approaching the disc parallel to the disc-endplates, and serves as depth control. Once

the needle tip has passed the posterior articular elements and approaches the inferior portion of the neuroforamen, the advancement can be monitored in the LL projection, very slowly and gently, until the tactile fibroelastic resistance of the annulus (definitely different from bone and soft tissues) is encountered. If during this approach, a radicular paresthesia or a true radicular pain is elicited (to be remembered that the region of the neuroforamen has not been anesthetized), the needle needs to be retracted and redirected. If the nerve root is touched, the patient experiences radicular symptoms, usually a sensation described as a sudden "electrical shock" which may radiate as distal as the foot, depending on the root that has been abutted. In contrast, the pain originating directly from the nociceptive fibers of the external annulus is less intense and does not typically refer below the knee. The described needle trajectory should bring the needle dorsal, inferior, and medial to the nerve, which is coursing from the upper portion of the foramen anteriorly, laterally, and inferiorly (Figure 24.3). Therefore, if the patient experiences radicular pain from the needle placement, it usually occurs when the needle is placed too high in the foramen or anterior and lateral to the posterior vertebral body's margin. In such cases, as a first attempt, the needle is slightly retracted, and redirected medially and inferiorly, using the bevel steering, or if the needle is stiff enough, gently manipulating it from the external noninserted portion; if these minimal adjustments fail, a more drastic needle's retraction and redirection, and sometimes a more oblique needle's approach starting from a more lateral entry point in the skin, should be obtained. All major redirections of the needle require it to be withdrawn into the subcutaneous soft tissues before new advancement, because the fascial planes create a point of fixation that does not allow for major path corrections. If the needle is not sufficiently withdrawn, further attempts of needle placement will only result in needle bending. Once a painless needle advancement to the outer margins of the annulus has been achieved, as determined by its tactile quality, before inserting the needle in the annulus, its position must be rigorously confirmed in AP and LL views; the tip of the needle has to abut the posterior margin of the disc space, midway between and parallel to the disc-endplates, in the LL view, and be placed under the pedicle in the AP view, thereby confirming its correct position outside the spinal canal. A position of the needle's tip too anterior in the LL, and lateral on the AP views, suggests insufficient obliquity of the approach, possible entry in the lateral annulus, unlikely to reach the center of the nucleus pulposus, and also possible risk of injury of the retroperitoneal structures; a position of the needle's tip medial to a vertical line connecting the medial borders of the pedicles on AP, before having entered the annulus, warns on the violation of the epidural space, with possible damage to the thecal sac (Figure 24.5).

Once the needle tip position is confirmed on AP and LL view, the needle can be safely advanced into the annulus fibrosus to reach the center of the disc using the LL view. The needle insertion through the annulus fibrosus can cause nonradicular pain, as mentioned earlier. The fluoroscope is then repositioned to obtain an AP view, allowing the operator to confirm that the needle's tip is correctly placed on both AP and LL views (Figure 24.3). If the trajectory is too anterior, the trocar tip is visible in the center of the disc on the AP view, but extends ventral to the center of the disc on the LL view. When the trajectory is posterior, the needle's tip will appear to be in the center of the disc on the AP view, but posterior to the center of the disc on the LL view (Figure 24.3). Because the nucleus pulposus is situated slightly posterior in the center of the disc and the needle tip should be as close to the herniation as possible, a posterior trajectory placement is not only acceptable, but preferred (as previously stated, ideally at the junction between middle and posterior third of the disc).

L5-S1 Disc Access—Special Considerations

The anatomy, and consequently the fluoroscopic views, is different at the L5-S1 level, because of the prominent lordosis, and the presence of the iliac crest. The presence of the iliac crests often obstructs the desired posterior oblique

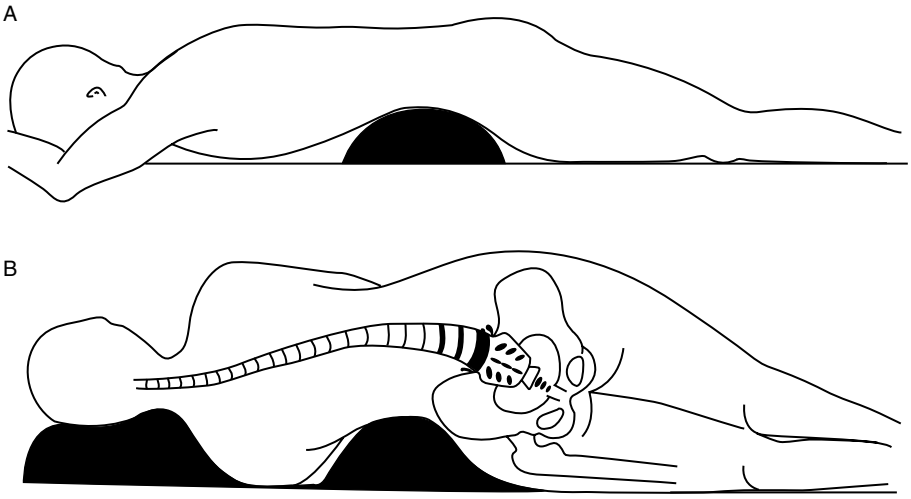

Figure 24.4 Patient positioning for lumbar discectomy. **(A)** shows the use of a bolster to be placed under the lower abdomen when the patient is in prone decubitus, to flatten the lumbar lordosis, and open the disc space posteriorly for an easier access and better transmission of the pressure drop to the herniated disc component. Similarly, the patient is flexed when in the lateral decubitus position, by positioning a bolster under the recumbent side **(B)**, approximately at the level of the disc to be treated, with the intent of opening the disc space on the entry side, and of tilting away the iliac crest for access to the L5-S1 disc.

trajectory of the needle into the disc space, whereas the lordotic angle of the L5-S1 spinal unit imposes steep fluoroscopic obliquities, and cause the divergence of the disc-endplates, resulting in a more problematic visualization of the disc space in the AP projection. Other situations that might contribute to the challenge of a percutaneous disc approach at L5-S1 are narrow disc space, spondylolisthesis, marginal osteophytes, and transitional anatomy with sacralization of a lumbar vertebral body. The procedure can be performed with the patient lying in the prone or lateral decubitus, as for the access at higher levels, but performing the procedure with the patient lying in the lateral decubitus position increases the probability of correctly entering the L5-S1 disc. A soft silicon gel cushion or other similar prop wedged just superior to the iliac crest will laterally flex and lower the iliac crest on the entry side, thus opening a trajectory to access the L5-S1 disc (Figure 24.4). Prior to beginning a decompression at the lumbosacral junction, the AP and LL views should be obtained to ascertain whether there is a transitional vertebra, an enlarged transverse process, the degree of disc height decrement, presence of osteophytes, and the height of the iliac crests. The fluoroscopic view for the L5-S1 disc access is obtained centering the x-ray beam over the L5-S1 disc space in the AP view. The fluoroscopic unit is then angled in the CC direction until the endplates of the disc are visible as single superior and inferior single lines, indicating that the beam angle is parallel to the endplates. As mentioned earlier, in patients with prominent lordosis, the disc-endplates at L5-S1 are not parallel but rather divergent, as easily noted on the LL view of the lumbosacral junction; in those cases, the tube obliquity to achieve profiling of the inferior disc-endplate of L5 is lesser than the obliquity needed to profile the superior disc-endplate of S1. Choosing either one of the tube obliquities will not reflect the disc axis, and will lead to incorrect needle trajectory. We suggest that the obliquity chosen in the AP projection to profile the disc space to be intermediate between the obliquities needed for the two adjacent disc-endplates. Practically, although craniocaudally tilting the tube in the AP projection, the profile of the L5 inferior disc-endplate will appear first; at this point, slowly increasing the obliquity will lead to a fluoroscopic view where the profile of the inferior L5 disc-endplate is suboptimal, but there is no clear profiling of the S1 disc-endplate yet, and this is our desired CC obliquity, intermediate between the disc-endplates, reflecting the disc axis. The x-ray beam is then angled toward the chosen side, laterally, without

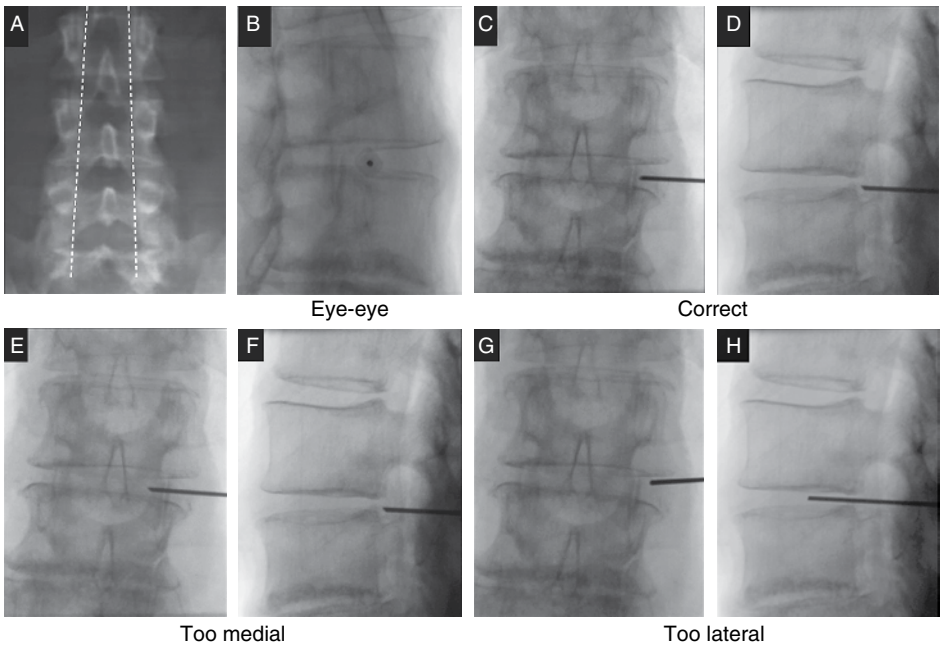

Figure 24.5 Safety fluoroscopic landmarks for disc access. **(A)** shows an AP fluoroscopic view of the lumbar spine with dashed vertical lines connecting the medial margins of the pedicles, and representing the bony landmarks of the central canal. These imaginary lines serve as safety landmarks during percutaneous disc access; the needle tip should never be medial to the medial border of the pedicle line (MBPL), until the disc annulus is penetrated, as demonstrated by the LL view. **(B)** shows the correct obliquity of the EE view to advance the needle; note that the ear of the Scottie dog bisects the disc-endplate. **(C,D)**, **(E,F)**, and **(G,H)** represent three different disc accesses obtained as a demonstration on a cadaver. On **(C,D)** the needle tip is correctly positioned just lateral to the MBPL on AP, and at the outer margin of the annulus on the LL; from this position the needle can be securely advanced to the center of the disc; on **(E,F)** the needle tip is incorrectly placed medial to the MBPL on AP, and still at the outer margin of the disc on LL, indicating potentially dangerous central canal violation; on **(G,H)** the needle tip is lateral to the MBPL, but has already advanced into the disc on LL, indicating insufficient access obliquity; the needle tip advances laterally through the annulus fibrosus, without reaching the nucleus pulposus, and could potentially stray in the retroperitoneal space.

changing the caudocranial angle, and as it is moved in an oblique orientation, the L5-S1 facet joint moves across the disc space and the iliac crest starts to overlap the anterior portion of the disc. When the beam is at approximately a 45° angle, the superior articular process of S1 is seen bisecting the S1 endplate, and a triangular window at the center of the disc space is seen (Figure 24.6). This triangle is bounded laterally by the iliac crest, medially by the anterior surface of the superior articular process of S1, and superiorly by the inferior endplate of the L5 vertebra. The center of the triangle, superimposed on the disc space, is our target. The needle can be advanced with the eye-eye technique to the target until it touches the outer annular fibers. When the needle is correctly angled along the desired trajectory, it will appear as a single dot. As explained for the disc access at other lumbar levels, once the LL view shows that the needle tip is correctly oriented along the disc space, and is approaching the region of the inferior portion of the neuroforamen, caution is recommended to avoid contact with the exiting nerve root. In this position, the obliquity of the tube can be quite extreme, sometimes the image intensifier of the fluoroscopy unit gets in close contact with the patient's shoulder, and the skin entry point can be high in the lower back, usually higher than the entry point for a L4-L5 disc access. Not uncommonly, high iliac crests cover the lateral oblique approach to the disc space, and the triangular window is visible only when the superior articular process of S1 is projected on the lateral third of the S1 endplate; any further obliquity of the x-ray beam brings the iliac crest to obstruct the path from the desired skin entry point and the disc space. Consequently, the entry route has to be less oblique (which means that the entry point in the skin is closer to the midline of the spine, for the needle to pass medially to the iliac crest), or must originate from a more cephalad starting point. With both approaches (more medial and more cephalad), there are instances in which straight instrumentation will not enter the disc correctly. If the trajectory of the needle is not obliquely angled enough, as discussed in the previous paragraph, it might be impossible to position the needle's tip in the desired position in the center of the nucleus pulposus, as it will tend to be too lateral and anterior in the disc. If the trajectory comes from a more cephalad entry point, the needle might still enter the disc, but will not be parallel to the disc-endplates, and therefore it will not advance in the disc space to the center of the nucleus pulposus. If the correct intradiscal position cannot be achieved with a straight cannula, a curved needle can be used. Although some operators might use a curved cannula as the introducing needle alone, this technique is very dependent on personal skills and expertise, and might require several attempts to achieve the proper trajectory. The most reliable and safe technique for a curved needle approach is most likely the coaxial technique. With the coaxial technique, the access is performed in a standard eye-eye

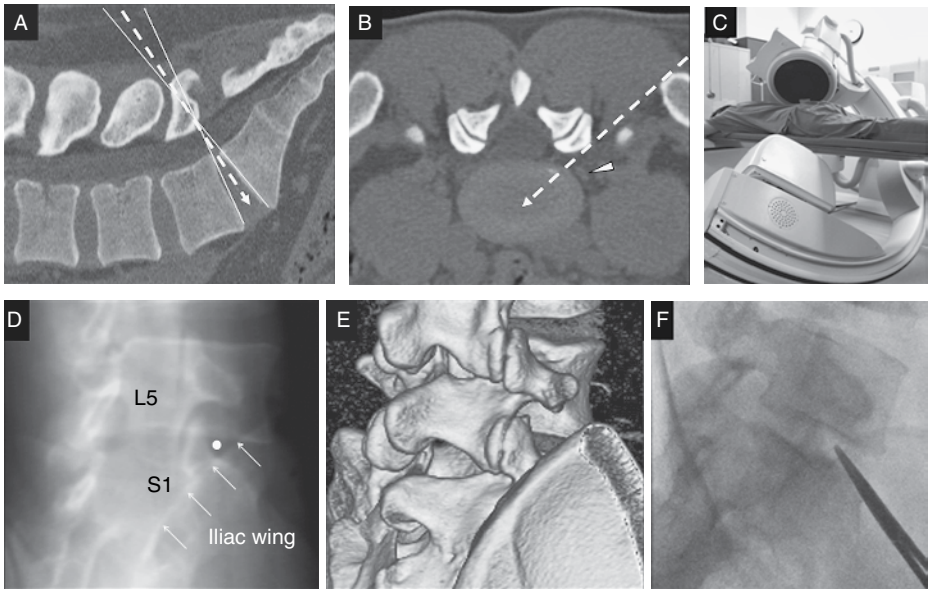

Figure 24.6 L5-S1 disc access. **(A)** shows how the lordotic curvature causes the disc-endplates not to be parallel mainly at L5-S1, and in some patients also at L4-L5. The most appropriate CC obliquity of the fluoroscopy tube to access these discs (dashed arrow) is, therefore, in between the angles necessary to profile the superior and the inferior disc-endplates (dotted lines). **(B)** shows the CT axial section through the L5-S1 disc; note that the RL obliquity of the disc access (dashed arrow) is limited by the iliac crest and by the superior facet articular process, whereas the needle trajectory needs to avoid the nerve root (arrowhead). **(C–F)** show the EE fluoroscopic access to the L5-S1 disc, with the 3D CT correlate. The angle of the tube can be extreme, in the CC and RL direction. The target (dot on **D**) is the center of the clear triangle formed by the superior margin of iliac wing, the lateral margin of the S1 articular process, and the L5 inferior disc-endplate.

fashion with a straight needle, usually a 17 or 18G, as described earlier, with the maximal obliquity allowed by the patient's anatomy. Once the straight needle has reached the external annulus fibrosus without causing any radicular pain, it can be inserted for a few millimeters in the disc. The stylet is removed, and coaxially, an appropriate size K-wire is inserted in the disc through the guiding needle; the guiding needle is withdrawn, while the K-wire is left in place in the disc, and exchanged with our working cannula, which has been previously manually bent at the distal end, to achieve the necessary angle correction from the straight approach that we obtained. The curved working cannula is now fed along the K-wire, and when it has passed the annulus fibrosus, the curve is rotated toward the portion of the disc that the operator wants to reach, a conjoined advancement of the cannula and retraction of the K-wire set the new position of the working cannula within the disc (Figure 24.7). It is strongly advised to perform this operation under real-time fluoroscopy control, to make sure that the needle tip is safely placed within the disc at each moment. A more extreme angled approach can be achieved by not inserting the guiding needle into the annulus, but by resting it against the external fibers of the annulus, where also the K-wire has to be positioned coaxially; the curved cannula is then released by the K-wire at the outer margins of the annulus and redirected for the desired entry in the disc. This is a technique that should be performed only by the most experienced operators, under absolutely compulsory fluoroscopic control, because of the risk of sliding against the annulus fibrosus with the curved cannula, and straying in the epidural space of the central canal, with possible dural sac and nerve root injury.

CT Guidance

CT guidance in spine procedures has the advantage over fluoroscopy guidance of cross-sectional anatomy visualization, much greater anatomical precision, and visualization of soft tissue structures in addition to the bony landmarks, but suffers from lack of panoramic view, from more cumbersome procedural aspects due to the necessity of localizing scans, and intermittent control scans to verify the correct needle advancement, and from lack of true real-time views. Unless CT fluoroscopy is used, at the cost of a significant radiation exposure of the patient and of the operator, the operator must advance the needle intermittently in a blind fashion, between control scans.

Target Visualization and Skin Entry

An initial localizing scan encompassing the region of interest is performed before the start of the procedure, with a localizing radiopaque grid applied on the patient's skin. The target is visualized directly on CT, and the CC level of the slice at which the optimal target is visible, expressed by a number preceded by S or I (meaning, respectively, superior or inferior) or by the signs ±, is recorded. The preferred trajectory of the needle from the skin to the target is then drawn as a straight line with the desired obliquity on the slice of interest, and the skin entry point is chosen and recorded in relationship with one of the markers of the skin grid. The distance from the skin to the target can be measured, which might help in choosing the most appropriate needle, and serve as a depth control measure. Absolutely without moving the patient, the CT bed is then slid in the gantry at the level of the selected slice, and in this way the gantry laser light defines, on the patient's skin, a horizontal (axial) continuous line that intersects the vertically oriented grid's markers. The intersection of this line with the chosen grid marker identifies the skin entry point and needs to be marked with an indelible skin marker. If the patient moves after the localizing scan and before the skin is marked, the localizing scan and the whole process needs to be repeated.

Gantry-Needle-Disc Alignment

As previously discussed, the human spine has multiple degrees of curvature in the sagittal plane, and a complex 3D anatomy. A pure axial cross-sectional view of the level of interest might not always fully represent this complex

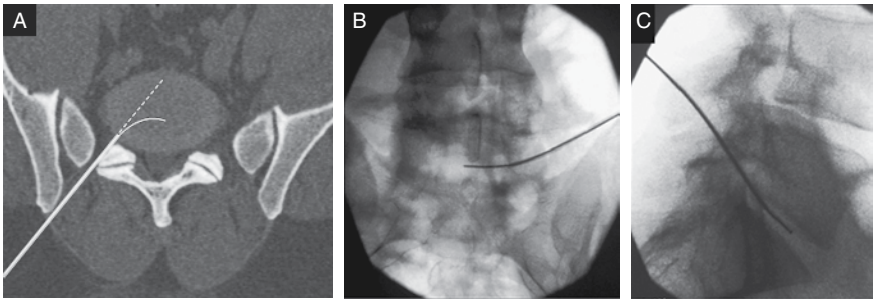

Figure 24.7 Coaxial curved needle access to the L5-S1 disc. In certain patients the degree of lateral obliquity of the access to the L5-S1 disc is limited by the iliac crest, as shown by an oblique axial CT view through the disc space **(A)**. In such cases, it is possible to reach the center of the nucleus pulposus using a coaxial system; a straight cannula is brought to the annulus, and exchanged with a K-wire; then a curved tip cannula is brought to the annulus along the K-wire; the K-wire is retracted, and the curved tip of the cannula is deployed, advanced, and directed to the center of the nucleus pulposus, as shown on **B–C**.

anatomy. Specifically, the disc space lies along different oblique axial planes at different intervertebral levels; therefore, using a straight axial CT plane results in incomplete segmental visualization of the disc space at multiple adjacent slices. To overcome some of these limitations of CT as a guide to the needle, and similarly to what usually desired on fluoroscopy-guided procedures, the concept of eye-eye view can be partially reproduced on CT by means of a gantry-needle-disc alignment. Most CT scanners allow gantry angulation in the CC and caudocranial directions of about 30º, at least for sequential nonhelical acquisition. After acquisition of the volumetric scout view of the region of interest in the two orthogonal planes, the lateral view is chosen to identify the obliquity of the desired needle trajectory along an anatomical oblique axial plane to optimally access the target disc. The gantry is tilted to reflect this angle, and the localizing scan is obtained along this oblique axial plane, which should show on a single slice the whole disc, and in its entirety, the needle trajectory, from the skin, to the target. The slice and the grid marker are selected, and the skin is then marked as usual, along the laser light of the gantry, tilted at the desired degrees of obliquity. Whatever the degree of tilting of the gantry, from −30º, to 0º, to +30º, it is crucial then, at the beginning of the procedure, to exactly align the marked entry point on the skin with the laser light of the gantry, insert the needle in the skin at this level, and align the needle shaft along the gantry angle, during its insertion toward the disc space. The correct CC angle of the needle is confirmed by the projection of the laser light line exactly at the entry point of the needle on the skin, along the needle's shaft, and precisely bisecting the needle hub (some scanners have a second external light parallel to the central one, to facilitate the operator, not obliged to lean toward the center of the CT gantry, and those two lights can be used interchangeably, as preferred). If this technical tip is correctly applied, the gantry-needle-disc alignment is obtained, the whole needle shaft, from the skin entry point to the tip, and the disc space, are in-plane with the gantry, the projected path of the needle is well discernible and predictable, on the same oblique axial slice (Figure 24.8). The adherence to this recommendation ensure greater needle control, for a faster and safer procedure, abolishing the need of complex, time-consuming, stressful, and often unreliable triangulations

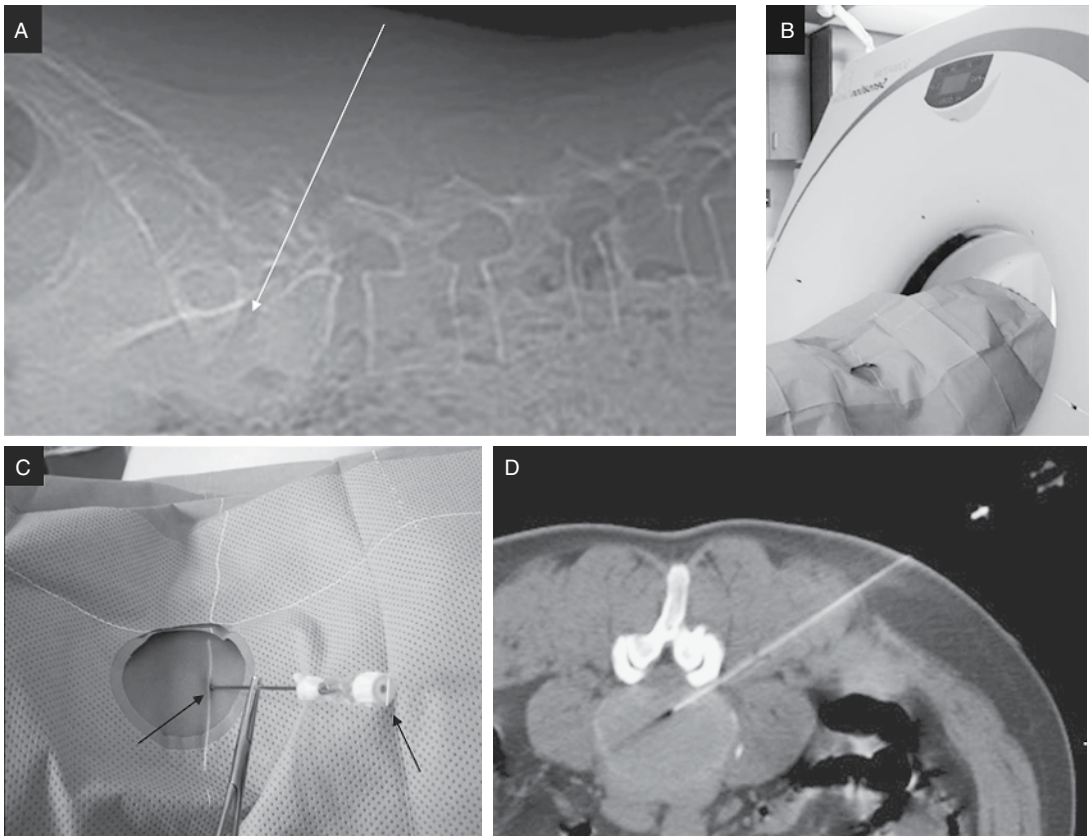

Figure 24.8 CT-guided disc access. The EE fluoroscopic concept can be translated into the needle-gantry alignment concept if CT guidance is used. The CT gantry is tilted to be parallel to the disc space to access **(A,B)**; the gantry laser beam is used as a guide to correctly angle the needle in the CC direction, exactly along the gantry, so that the laser light projects at the skin entry point, along the needle shaft, and on the top of the needle hub (black arrows on **C**). If adherence to this technique is respected, the whole length of the needle is imaged on one single slice, from the skin entry to its tip **(D)**. The RL obliquity of the access is left to the operator to "guess and check," while in the superficial soft tissues, until desired angle is found, thereafter the needle can be advanced to the target.

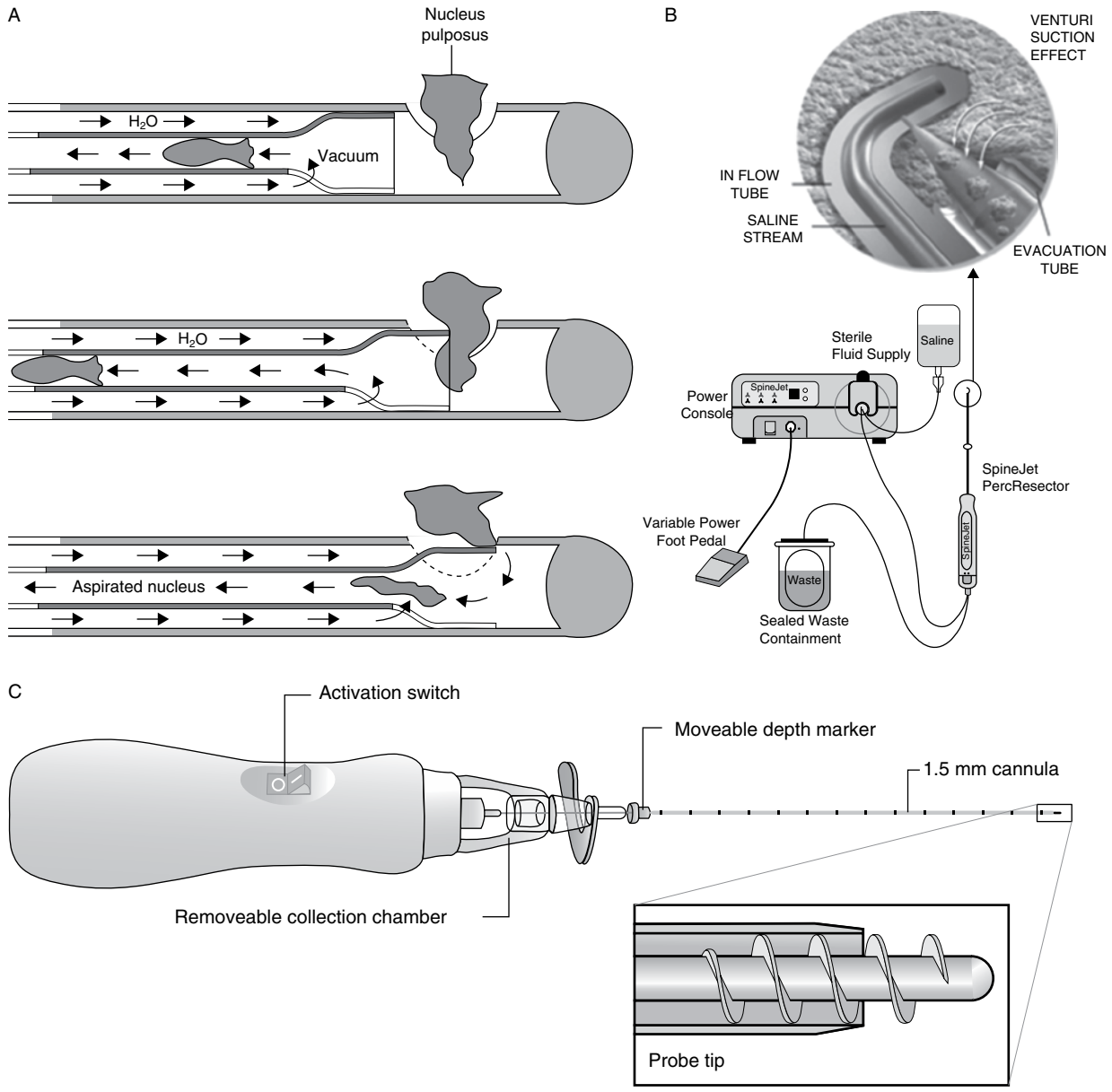

Figure 24.9 Mechanical devices. **(A)** shows the Nucleotome probe, with its blunt, rounded tip. Internal irrigation and cutting functions are incorporated. The aspirated nucleus pulposus enters the side port, and is resected by a pneumatically driven "guillotine blade," which has a reciprocal, not rotary movement. **(B)** shows the SpineJet Hydrodiscectomy system; the nucleus pulposus is fragmented by the high speed water jet, while a Venturi suction effect aspirates and removes the fragments through the evacuation port and tube. **(C)** shows the Lumbar Dekompressor and blow-up of its probe tip.

that the operator is obliged to imagine, to predict where the needle tip will be located after a certain depth of advancement, as usually happens when the needle is off-plane with the gantry, and imaged partially on multiple adjacent sections. The gantry-needle alignment greatly reduces the number of CT sections required at each CT control of the procedure (usually three to six slices are sufficient), and consequently reduce the procedure time and the radiation dose. It is imperative that the control CT views obtained intermittently during the procedure show the whole length of the inserted needle, with one slice above and one below in which no needle be visualized; if this safety condition is not respected, there is risk improper needle placement, with resultant risk of injury in a complex and delicate anatomical region such as the spine.

If the CC obliquity of the needle can be precisely controlled with the gantry-needle alignment technique, the desired RL obliquity of the needle in the CT-guided procedures is left to the experience and ability of the operator in reproducing on the patient the trajectory chosen on the

cross-sectional image of interest on the CT-consolle. Most commonly, in skilled hands, a satisfactorily RL trajectory is obtained with a maximum of three attempts of needle insertion at a point no deeper than the paravertebral muscles.

Posterolateral Oblique Approach

Not differently from the posterolateral oblique disc approach used under fluoroscopic guidance, on CT, once the correct axial obliquity of the scan is obtained, and the gantry is aligned with the disc space (i.e., on the single axial image slicing through the disc space there is no disc-endplate bone visualized), local anesthesia is administered to the skin, fascia, and paravertebral muscles, paying attention not to inject deep to the posterior articular elements, to keep a normal sensitivity of the exiting nerve root at that level. The working needle is also aligned with the gantry, obliqued as desired in the RL direction, and is inserted and advanced from a posterolateral approach to be lateral and tangent as possible to the lateral aspect of the superior articular process of the inferior vertebral level, and to course posterior to the visualized exiting nerve root. Once contact with the external annulus is reached, with no radicular pain, the needle is advanced through the annulus fibrosus and to the center of the nucleus pulposus.

Transdural Posterior Approach

If desired, using an appropriately small caliber needle (21–22 G), the disc space can be accessed from a posterior transdural approach at the lower lumbar levels, below the position of the conus medullaris. This access is feasible with straight needles when local anatomy permits an axial oblique plane parallel to the disc space passing through the interlaminar space. At certain levels, and in certain patients, the oblique plane passing through the disc space corresponds posteriorly to the bony laminae, which would clearly obstruct the needle path. The path of the needle is just lateral to midline where the spinous process is, entering the central canal through the ligamentum flavum, piercing the dura along the posterior and anterior aspect of the dural sac, and thereby entering the disc from the posterior longitudinal ligament. The nerve roots of the cauda equina easily allow this needle access, when performed gently. Although this is a potentially easy and effective access to the center of the nucleus pulposus, in selected instances, it carries additional risks of cerebrospinal fluid leak and headache, cerebrospinal fluid infection, epidural hematoma.

MODALITIES

Since the 1960s, many different techniques for percutaneous removal of the nucleus pulposus or its protruding components have been proposed. In the following section, the most diffused among them, and still present in current clinical practice, are described. They may achieve the goal in many different ways. The proposed instruments for disc decompression are chemical, mechanical, automated or not, or entail use of different types of energy, such as RF, laser, coblation, etc. The main goal of these procedures is mainly nonselective removal of the nucleus pulposus, with the aim of a global decompression and decrease of disc volume and intradiscal pressure, as previously discussed. The indications for each of these modalities are substantially identical.

Chemical

In 1959, Hirsch [49] suggested the use of proteolytic enzymes for the treatment of discal herniations. Lyman Smith, an orthopedic surgeon in Illinois, undertook a series of experimental studies in 1963 to demonstrate that intradiscal injection of a proteolytic enzyme was a possible nonsurgical treatment for disc removal. In 1964, in the *Journal of the American Medical Association*, he published his first report suggesting that the enzyme chymopapain, derived from the papaya, might prove effective in the treatment of herniated lumbar discs that have not ruptured through the encircling posterior longitudinal ligament [50]. The initially enthusiastic response to this approach has given way to a much greater degree of caution because of the reported complications of anaphylaxis and neural injuries [51–53], mostly owing to technical errors of some practitioners.

Chymopapain remains the most widely evaluated and clinically tested substance for the purpose of chemical, percutaneous treatment of disc herniations, although other enzymes such as collagenase [54] and chondroitinase ABC [55] have been proposed. Chymopapain is no longer available in the US market, although it still is in other countries.

Chymopapain is a proteolytic enzyme of vegetal origin, extracted from the latex of Carica Papaya, and has a molecular weight of 27,000 [56]. The enzymatic action of chymopapain is not highly specific and is exerted on numerous substrates such as hemoglobin, casein in milk, and on noncollagenous proteins of fibrocartilage. Like any proteolytic enzyme, chymopapain is immunogenic. Intradiscal injection of chymopapain may cause allergic reactions in humans. A small quantity of immunoreactive chymopapain appears in the plasma immediately following injection. As much as 3% of the North American population has been sensitized to papaya enzymes, due to the ingestion of chymopapain in the form of fruits or food additives. Certain subjects have developed an infraclinical sensitivity. This risk of anaphylactic reaction justifies precautions in the selection of patients, ruling out potential allergic subjects (generically hyperallergic or with previous history of anaphylactic reactions, history of allergy to papaya, or previous exposure to parenteral chymopapain, with IgE antibodies to chymopapain). In the usually administered dosage, chymopapain acts only on the proteoglycans [57–59]. Its activity takes place on the glucosaminoglycans-carrying protein. The cleavage leads to a depolymerization of the large proteoglycan molecules, and liberation of the polysaccharide groupings which lose their capacity to retain water. This hydrolytic action of chymopapain on the proteoglycans of discal tissue, well demonstrated by several experimental studies realized in vitro on normal and

pathologic human discs, and in vivo, in numerous animal species, leads to diminution of the volume of the disc and of its herniated fragment, thus reducing the compression of the nuclear material on the nerve root. Diminution of the water content can indeed relieve the hydraulic intradiscal pressure. The nonspecific proteolytic action of the chymopapain can result in severe neurologic complications [60] if misplaced outside of the disc. Adherence to a precise and proven technique is mandatory. The procedure must be performed under local anesthesia, because general anesthesia can mask early sign of an allergic reaction, and might result in possible nerve root puncture, which could in turn also lead to intrathecal injection. An 18- to 22-gauge, two-needle technique is preferable [61,62], with the 22-gauge needle advanced coaxially in the center of the disc. Injection of a small amount of contrast medium at this point can rule out epidural or intrathecal injection. The enzyme is injected slowly, in 10 to 20 minutes, to let it be accepted by the nuclear material (the positively charged enzyme will bind with the negatively charged nuclear matrix), thus reducing the risk of epidural migration. The needle is then withdrawn and the patient kept under observation for 20 to 30 minutes for possible systemic allergic reactions. The patient can be discharged from the hospital the same day.

Collagenase, an enzyme synthesized by the *Clostridium histolyticum*, splits collagen fibers, particularly type-2 fibers, mainly found in the nucleus pulposus. Wittenberg, in a randomized, prospective study, observed at 5 years 72% of good results in the chymopapain group and 52% in the collagenase group [63].

Alcohol was proposed in France for chemical lysis of the nucleus pulposus and its herniating components by the group of Tournade in Colmar [64] in 1999. Alcohol is used as a lytic and necrotizing agent in many interventional procedures (ablation of tumors, vascular malformations, nerve and ganglion blocks, including the Gasserian ganglion). Alcohol produces a molecular split of proteoglycans and glucosaminoglycans. The main advantage over chymopapain is the absence of allergic reactions.

Recently, Théron [65] proposed, for the same purpose, alcohol linked to a more viscous agent, a gel of ethylcellulose, commercially called Discogel, opacified with tungsten powder. The aim by combining the alcohol with a viscouse material is better control over the diffusion of the lytic agent, thus limiting the risk of damaging adjacent structures. The agent remains injectable through a small-bore needle, and injection can be followed under fluoroscopy, much like contrast medium for discography.

Mechanical

In 1975, Hijikata [66], in Japan, published his results with a series of patients who underwent lumbar discectomy performed percutaneously. Rather than relying on enzymatic dissolution of the herniated disc, he used specially designed instruments placed through a 5-mm cannula inserted through the lateral annulus. A circular incision was made in the annulus, and the herniated disc material was grasped with modified pituitary-type rongeurs. In his initial published findings, Hijikata reported that approximately 80% of his patients experienced improvement after this procedure. Variations on this method have been subsequently popularized by Kambin [67,68] in Philadelphia and Suezawa [69] in Switzerland. Using Craig-type biopsy instruments under fluoroscopic control, Kambin inserted a large trocar through the lateral annulus fibrosus, grasped the herniated disc, and removed it. He reported excellent results with no significant complications in 85% of 50 patients. Suezawa used the instruments designed by Hijikata and in addition he inserted a discoscope through a contralateral approach. This was essentially a fiberoptic system used to visualize, from the contralateral side, the disc material being removed. Excellent results were reported in 67% of 47 patients, although the majority of these patients showed complicating factors, such as spinal stenosis.

In another development, Jacobson [70], a neurosurgeon in Miami, designed his own instruments and used a direct lateral approach to remove herniated discs percutaneously in more than 300 patients. With the patient under general anesthesia, a 10- to 11-mm cannula was introduced through the lateral annulus. Using his own patented instruments, Jacobson grasped and removed disc material with overall good results in terms of pain relief. Unfortunately, unacceptable injury of bowel and peripheral nerves occurred. Friedman [11] studied Jacobson's technique in cadavers and demonstrated that the anatomical variations were such, that an unacceptably high rate of morbidity and potential mortality could be expected with this technique. Friedman therefore recommended against its use.

After surveying the previous techniques and assessing their potential problems, Gary Onik working with engineers from Surgical Dynamics, Inc, designed his own instruments for lumbar discectomy in 1984 and introduced it in clinical practice in 1985 [71–74]. The technique was called "automated" percutaneous lumbar discectomy, because it involves a mechanical probe, Nucleotome, which removes the nucleus pulposus by a "suction and cutting" action (Figure 24.9A). The device is now manufactured by Clarus Medical, LLC. The probe tip, excluding the handle, is 20.2 cm long and has an outer diameter of 2.2 mm. The negative pressure for aspiration is generated by the vacuum-generating console. A vacuum is created that draws nuclear material into the side port, which is located a few millimeters proximal to the distal tip of the probe. The cutting blade for fragmentation of nucleus pulposus aspirated through the port works with a reciprocal, not rotatory motion. This type of movement is a safety feature because the "guillotine" blade is contained within the probe. Consequently, only the nuclear material that is drawn into the port can be cut. The material aspirated from the inner disc and exiting through the metallic probe is ultimately deposited into a filter in a disposable collection bottle. The extracted nucleus pulposus is thus available for quantitative and macroscopic qualitative evaluation, or even for histology examination. A sequence of devices is used for introduction of the probe inside the disc, the last one being a cannula, straight with an outer diameter of 2.8 mm, or a curved one, with an outer diameter of 3.8 mm, better suited for access to the L5-S1 disc space, when the direct path from the skin is

covered by the iliac crest. The reason for a larger diameter in the latter is that it is internally coated by a Teflon layer, which reduces friction and favors the sliding of the flexible but straight probe. APLD as proposed by Onik has lost the favor of most operators, and discussion of the reasons of that falls beyond the limits of this chapter. One of the authors (GB) of this chapter has developed a large experience with very good results, depending on a strict and wise selection of patients, and still uses it in few, very selected cases [75]. In any case, Onik's remains the percutaneous procedure that removes the largest amounts of nuclear material from within the intervertebral disc. Another advantage, when comparing APLD with physical techniques that blindly destroy the disc (such as laser, RF or coblation), is that the surgeon can verify directly and visually the quantity of disc material removed, as well as its "quality." The extracted nucleus pulposus can be observed as it passes through the transparent tubing that connects to the filter. How much nucleus is taken out and how degenerated it is, are important procedural and prognostic pieces of information. For example, viewing the quantity of removed nuclear tissue and comparing it with the amount that was anticipated to be extracted from interpreting the preoperative imaging provides critical information to determine whether the probe worked in the correct intranuclear location. Observing blood coming from the disc could suggest the presence of unexpected degeneration, or of painful granulation tissue inside the disc, or prompt arrest of the procedure so as not to damage the endplate cartilage. Another important safety feature is that, once the Nucleotome is safely within the disc, it is unable, unlike other devices, to cut its way out of the disc space to cause injury to vital structures.

The notion that Onik's proposal was meritorious is suggested by the recent resurgence of new devices designed to mechanically remove the nucleus pulposus in an "automated" mode by aspiration. Observational studies are available, together with four RCTs [75–78]. The level of evidence for clinical effectiveness of APLD, as determined based on the US Preventive Services Task Force (USPSTF) criteria [79], using five levels, ranging from I to III with three subcategories in level II, is level II-2 for short and long-term relief [80]. However, APLD does not appear to compare favorably against chymopapain injection and open discectomy [76,78].

A similar hydraulic aspiration principle is utilized by the SpineJet probe, produced by HydroCision. The disposable SpineJet probe simultaneously cuts and aspirates nucleus; a round atraumatic tip design reduces risks of annular puncture and endplate damage. The SpineJet Hydrosurgery system utilizes a reusable power console with foot pedal activation (Figure 24.9B). Both the Nucleotome and the SpineJet are fluid-based systems, that is, the inner disc material is hydrated while the probe's aspiration action is active for tissue removal: consequently they can, unlike every other system (purely mechanical, thermal, laser, etc.) efficiently ablate tissue regardless of patient's age and disc hydration. Moreover, internal irrigation with sterile saline is a vehicle for easy aspiration, to prevent accumulation of nuclear material and consequent clogging inside the probe.

The Dekompressor, proposed by the Stryker Company in 2003, is a single-use probe, introduced through a 15-mm cannula, intended for percutaneous discectomy in the lumbar, thoracic, and cervical spine. Under fluoroscopic imaging, the Dekompressor utilizes an Archimedes pump principle to remove nucleus material from the disc. The rotating screw blade is spun by a disposable rotational motor (Figure 24.9C). The single-use probe is smaller than Onik's Nucleotome, with no need for a console or other external control to make it operate properly. Unlike the Nucleotome and the SpineJet, the Dekompressor does not entail a hydration of the inner disc to favor tissue removal (purely mechanical extraction). Case series are available [81–85], although no controlled studies have to date been performed. The level of evidence for clinical effectiveness, based on USPSTF criteria [79], is level III for short- and long-term relief [86].

RF Ablation

The use of thermal energy to modulate and ablate tissue is not new. Electrical current, in one form or another, has been applied to human tissues as a surgical modality for more than 100 years. RF energy occupies a range upon the electromagnetic radiation spectrum. The frequency at which the device operates determines the absorption characteristics and tissue effects. Electrosurgical units based on standard monopolar or bipolar devices generally operate from 200 to 500 kHz and they are limitedly applied or avoided to prevent unwanted tissue destruction. Devices operating in this frequency range cause the electrode that comes in contact with the tissue to become hot, therefore acting like true heat cautery. RF in the radiowave range (between 1.7 MHz and 4.0 MHz) of the radiation spectrum emits energy that is nonthermal with optimal controlled absorption characteristics of water-rich tissues, with minimal tissue alteration. High-frequency radiosurgery, above 1500 kHz (1.5 MHz), transmits pure radiowaves to the tissue without heating the electrode. The heat for this ablation is generated by a natural resistance of the tissue, which comes in the path of the waves released through the electrode tip of the device. The cellular water in the soft tissues gets heated and when the temperature reaches 100°C, it starts boiling, and produces steam, which results in cellular molecular dissolution of individual tissue cells. The cells exposed to these waves are destroyed whereas the surrounding tissues remain unaffected. This property of radiofrequencies eliminates the possibility of undesired damage to the normal tissues, while improving the surgical precision.

The Disc-FX discectomy system is proposed by Elliquence, LLC, formerly Ellman Innovations, LLC, New York. It works in bipolar mode at 1.7 MHz. In particular, the bipolar Trigger-Flex probe is used to obtain a radiowave energy application both for removal of nucleus material and a modulation of weak collagen fibrils and sealing of annular tears (shrinking or eliminating defects in the annulus) as well as contributing to depopulating nerve fibers sensitizing the outer annulus due to its smooth thermal effect.

The two different effects in the nucleus (ablation for decompression) and in the annulus (annulus modulation)

are obtained by means of two different waveforms generated by an external source; the ablation in the nucleus is achieved using a more aggressive waveform, called "Bipolar Turbo," the modulation of the annulus using a smoother waveform "called Bipolar Hemo."

The bipolar Trigger-Flex probe is flexible and steerable (Figure 24.10A), and consequently can be oriented to operate either in the nucleus or along the posterior annulus. At the same time, the flexible probe allows a more targeted removal of the protruding nucleus, thus relieving tension on the innervated and irritated annulus.

Neither observational studies nor RCTs have been published in the literature. In our personal but limited experience of the first 30 cases, the procedure showed short-term results comparable with those of Nucleoplasty. The action of the probe on the posterior annulus and its nerve fibers should allow treatments also of patients suffering from purely discogenic back pain.

Coblation

Percutaneous plasma RF-based discectomy, with commercial name Nucleoplasty (ArthroCare, Sunnyvale, CA), is conducted by using a bipolar radio-frequency-based device, which functions via a plasma-mediated process [87–89], to perform precise removal of disc tissue. In this process, bipolar voltage pulses at 100 kHz are applied to the active electrode at the distal end of the device, which produces a strong electric field region around the electrode. The electrolytes in the surrounding conductive medium (e.g., sodium ions resident within the nucleus pulposus) respond to the electric fields, and if the voltage is sufficiently large, a localized finely focused plasma field (ionized vapor) is produced between the electrode and adjacent tissue [23,90]. The plasma field comprises a complex mixture of gas-phase radical chemically reactive and nonreactive molecules and a very small fraction of ionized particles (predominately positive ions and electrons), some of which can break molecular bonds in the adjacent tissue by energetic particle bombardment and chemical reactions. The organic molecules in the disc material (particularly long-chain molecules such as collagen) are thought to be susceptible to fragmentation by the plasma particles, resulting in their conversion into liquid and gaseous products that are subsequently desorbed from the targeted site. Water molecules (which compose a significant fraction of most types of tissue) can be fragmented into excited and groundstate hydroxyl radicals and hydrogen atoms. Both of these species are chemically active and can cleave long-chain molecules (e.g., collagen) into smaller fragments that are either more easily liquefied or gasified. Moreover, electrons emitted from the electrodes at the distal end of

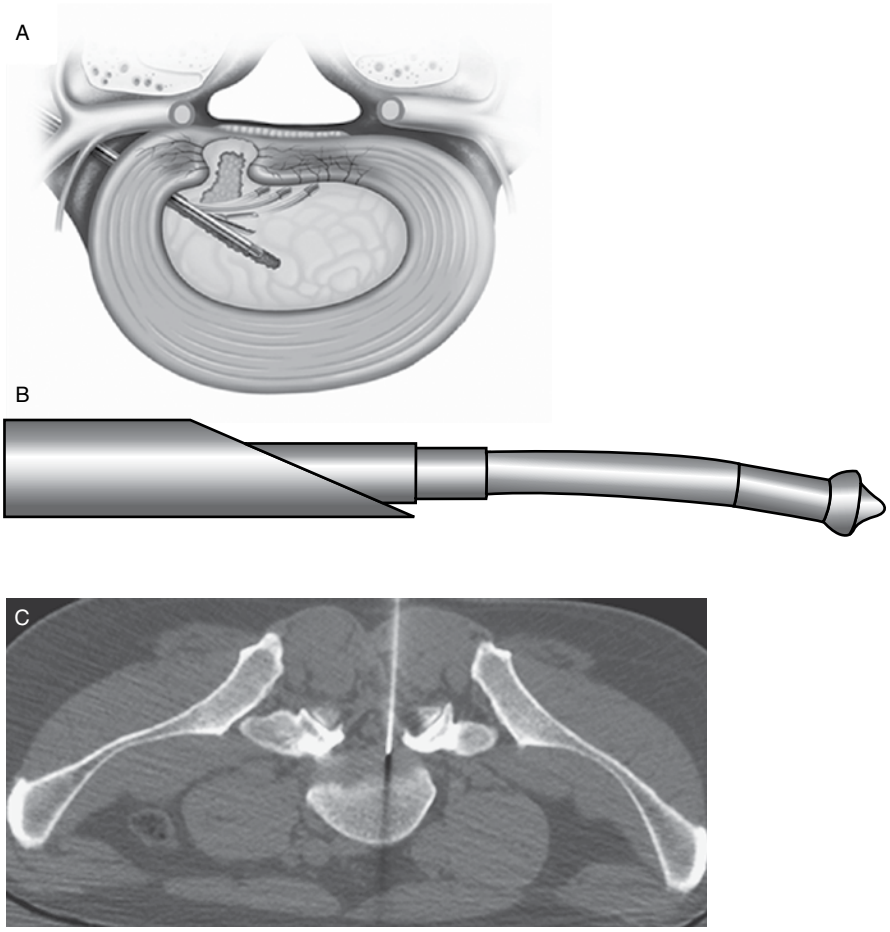

Figure 24.10 Thermal-radiofrequency-laser devices. **(A)** represents a schematic drawing of the proposed action of the Disc-Fx; the steerable probe allows topographic nuclear material removal at the site of internal annular disruption, and thermal modulation of the nerve fibers in the posterior annulus, using a less aggressive waveform. **(B)** shows the active tip of the Arthrocare spine wand, introduced in the nucleus pulposus through a beveled 17 G needle; the slightly curved tip allows the creation of multiple coblation channels upon rotating the proble at multiple passes. **(C)** shows the CT-guided coaxial insertion of the laser fiber through a 21G cannula into the extruded disc fragment, through a translaminar and transdural paraspinous approach.

the device when the voltage is applied can develop sufficiently high energies not only to cause the water molecules to fragment but also to directly dissociate the chemical bonds in the nearby targeted tissue structures (in this case disc tissue) into smaller fragments [89]. The net result is a reduction of soft tissue volume and effective excision of the soft tissues within the nucleus. Small channels are created in the nucleus, and they are thermally treated, producing a zone of thermal coagulation. Thus, Nucleoplasty combines coagulation and controlled tissue ablation (patented Coblation technology). The plasma radio-frequency-based process has been reported to have minimal histopathologic effect on tissues immediately adjacent to the treated site [23,90], particularly annulus, endplates, neural elements, and nerve roots [90]. The temperature is kept below 70°C to minimize tissue damage and avoid tissue charring. Because of the mechanism of action on hydrated nuclear components, tissue ablation and consequently intradiscal decompression are supposedly higher in younger patients and in hydrated, nonadvancedly degenerated discs [23]. Actually, an exclusion criterion for lumbar Nucleoplasty must be considered a disc height less than 50% [91]. The coblation probe is introduced through a very thin, 17 G cannula (Figure 24.10B), probably the narrower among nonchemical procedures, laser excepted. This also allows for bending of the cannula and probe, for access to difficult L5-S1 levels. A large experience is available and since 2000 many tenths thousands of patients have been treated using coblation technique, both at lumbar and cervical levels [47,92–96]. Many observational studies are available but not RCTs. The level of evidence for clinical effectiveness, based on USPSTF criteria [79], is level II-3 in managing predominantly lower limb radicular pain, with no evidence for axial back pain [91,97].

Laser

Percutaneous laser disc decompression (PLDD) was introduced by Choy et al. [21,98] in 1986. By 2002, more than 35,000 PLDDs had been performed [99]. The term laser is an acronym standing for Light Amplification by the Stimulated Emission of Radiation. Laser is a form of light, and light is made up of electromagnetic energy. Laser energy is formed by energizing an active lasing medium. With the introduction of energy from an outside source, the atoms absorb the energy causing the electrons to rise to a higher excited state. When the electron returns to the normal, nonexcited state, the energy initially absorbed is given off as a photon, and the photon bundle has unique properties characteristic to that particular medium. Lasers are generally classified according to the medium they use to produce the laser light. Solid-state, gas, liquid, and semiconductor are all common types of lasers. The radiant energy of the laser beam can be transformed into heat energy that produces medical and surgical effects in tissue, such as coagulation, vaporization, or cutting. The total power output of a laser is measured in watts, the power density, measured in watts per square centimeter, and it determines the thermal effect in the target tissue; the energy density (measured in power density X time) of joules per square centimeter indicates the total amount of energy put into a given tissue. The majority of surgical lasers fall in the invisible portion of the electromagnetic radiation spectrum. The absorption characteristic of the medium largely determines the extent of penetration in particular tissue types. Application of any laser requires the surgeon to completely understand the characteristics of the specific laser for safe and effective use. The way in which light interacts with a substance largely depends upon its wavelength. Penetration depth at a certain wavelength is mostly affected by absorption by specific molecules, such as water (the principal component of the nucleus pulposus), hematoproteins, pigments, nucleic acids, and so on. As a laser is absorbed by the tissue, several surgical effects take place: at 60°C protein denaturation and coagulation of blood vessels, near 100°C evaporation of intracellular water causing shrinkage and tissue loss, beyond this point vaporization will occur. In general, the therapeutic effect of a laser significantly depends on penetration depth, in effect determining whether tissue removal or hemostasis will be predominant. Intradiscal decompression is obtained by shrinkage of the water-rich nucleus pulposus by vaporization. The evaporation of water and the increase in temperature causes protein denaturation and subsequent renaturation, causing a structural change of nucleus pulposus, limiting its capability to attract water [21,99]. An increase of intradiscal volume of only 1.0 mL causes the intradiscal pressure to rise by as much 312 kPa or 2340 mm Hg. On the other hand, a decrease of intradiscal volume causes a disproportionately large decrease of intradiscal pressure. Other beneficial effects of the laser action are postulated, such as shrinkage of collagen fibrils with reduction of disc volume [100,101] and destruction of nociceptors in the annulus or in the granulation, vascularized tissue growing in degenerated discs. Many types of lasers have been reported in the literature for spine applications [102], including the following: Nd:YAG (whose active medium is a crystal of yttrium, aluminum, and garnet doped with neodymium ions, and whose beam is in the near infrared) at 1054 nm and at 1320 nm, KTP (a beam generated by a neodymium:YAG laser is directed through a potassium titanyl phosphate crystal to produce a beam in the green visible spectrum) at 531 nm, CO 2 at 10,600 nm, Ho:YAG (YAG doped holmium) laser at 2100 nm, diode (semiconductor) laser at 810–890–940–980 nm. The Ho:YAG laser is better suited for open or endoscopic surgery, under direct visual control, because of the more mechanical effects and risk of endplate damage from energy scattering [103–105]. Yeung, Casper, Chiu, and Knight further report utility of Holmium laser to remodel extradiscal bony architecture during endoscopic spine surgery [105–107]. Nd:YAG at 1054 nm and KTP 532 are the most popular for intradiscal treatments [21,108–112]. KTP lasers are similar to Nd:YAG lasers in both action and effects [113,114], as are diode lasers [115]. The latter have the advantage of a much less expensive and less cumbersome power unit. Advantages of the 980 wavelength (peak absorption of water) of the diode laser are maximum absorption for well-hydrated, soft tissue like the nucleus pulposus, with great thermal effect and consequent efficient shrinking effect [115,116], with minimal thermal damage of surrounding tissues

(particularly the endplates). Absorption by hemoglobin is lower than KTP 532 nm lasers, but they maintain an acceptable haemostatic effect. Advantages of the laser discectomy are the tiny access port, the tiniest among the different percutaneous discectomy modalities. For the diode laser the size of the optic fiber is as small as 220 μm, although the best compromise is obtained with the 360-μm probe (too high concentration of the delivered energy for the 220-μm fiber). Such small fiber sizes, which fits coaxially in 21-gauge cannulas, allow also a transcanalar/transdural approach to the disc, under CT guidance (personal experience) (Figure 24.10C). Nd:YAG laser fibers have usually a diameter of 400 to 600 μm. A series of pulsed shots with a maximum of 1-second duration (usually between 0.4 and 0.6 seconds) at no more than 15 watts and at 2 seconds intervals are delivered to the nucleus pulposus. A minimal saline perfusion through the access cannula prevents the tissue temperature getting higher than 100°C. No more than 1200 joules (but better below 1000, usually between 600 and 800) as total dose are administered to the center of the nucleus pulposus, in two to three different positions of the fiber tip, always under fluoroscopic control of the proper position of the device. The most frequently described complication of PLDD is spondylodiscitis both aseptic and septic [10,117–122]. The reported frequency of discitis varies from 0% to 1.2% [117,123–126]. Aseptic discitis is the result of heat damage to either the disc or the adjacent vertebral endplates [127]. Many observational studies report on the laser disc decompression results, but no RCTs are available [4]. The level of evidence for clinical effectiveness, based on USPSTF criteria [79], is level II-2 for short- and long-term relief [36].

SAFETY AND RISKS

Percutaneous disc procedures are characterized by low invasiveness, and low risk profile; nevertheless, they present potential risks [22,74–78]. The main risks associated with these procedures are infection, bleeding, nerve root injury, thecal sac injury, disc-endplate injury, injury to the retroperitoneal structures, and colonic perforation.

General Technical Safety Principles

Comprehensive knowledge and extremely precise use of the radiologic projections and landmarks are prerequisites to perform these procedures. Thorough use of precise oblique working eye-eye fluoroscopic technique, with intermittent AP and LL verification of device position, or needle-gantry alignment in CT-guided procedures, is mandatory. Adherence to a safe technique will dramatically impact the outcome and the probability of realizing a side effect or complication. An incorrect projection means that the cannula and the intradiscal device is actually working away from the place where it is supposed to, not effectively operating on the nucleus or, worse, damaging vital or functionally important structures.

As discussed earlier in the technique paragraph, main *safety landmarks in fluoroscopy* are a line connecting the medial border of the pedicles on AP, and a line connecting the posterior vertebral walls on LL: the medial pedicular line is not to be crossed if the device tip has not entered the disc space on LL, to avoid thecal sac injury, whereas the posterior wall line is not to be crossed in LL if the device tip is not under the pedicle in AP, to avoid straying in the retroperitoneum. The device tip needs to be in the nucleus pulposus, away from the disc-endplate, on a well-profiled LL view of the disc space, to avoid mechanical or thermal injury (depending on the device used) to the cartilage of the disc-endplates.

Infection Risk

All percutaneous disc procedures harbor a significant risk of disc infection. Absolute contraindication to a disc procedure is local or significant systemic infection. We recommend absolute sterility of the procedure, with particular care in the prep and drape process, strict use of full drape, full gown, gloves, mask, and hat. In addition, we administer the patient an intravenous antibiotic 10 minutes prior to the procedure for prophylaxis, Cefazoline 1 g intravenous, or, in case of allergy to penicillin, ciprofloxacine 400 mg intravenous.

Bleeding Risk

Although there are different policies at different institutions [128], we set our threshold for a safe performance of an intradiscal procedure, to a minimum of 80,000/mm^3 platelet count, and 1.3 INR. We perform the procedure in patients treated with nonsteroidal anti-inflammatory drugs, including ASA, without special considerations. We recommend witholding the assumption of other antiaggregants before the procedure, such as clopidogrel (7 days) and ticlopidine (14 days). In case of patients treated with low-molecular-weight heparin (LMWH), we perform the procedure at least 12 hours (LMWH prophylaxis regimen) or 24 hours (LMWH therapeutic regimen) after the last dose, and we recommend to withhold the LMWH for 24 hours after the procedure. Patients treated with unfractionated SQ heparin less than 10,000 units daily can undergo spinal procedures, whereas patients treated with more than 10,000 units daily, and those who receive unfractionated intravenous heparin, can undergo a spinal procedure 4 hours after the last dose, provided that the activated partial thromboplastin time is normal, and their heparin can be restarted as early as 1 hour after the procedure.

Nerve Root Injury

With the posterolateral paravertebral access to the disc, the needle path is posteromedial to the exiting nerve root. To ensure safe disc access, the needle path has to be lateral but as tangent as possible to the superior articular process of the subjacent vertebral body, and with a sufficient degree of obliquity, to avoid the nerve root. Because the radicular pain is a very effective "safety alarm," by no means local anesthesia should be delivered to the region of the neuroforamen. Otherwise, the operator might have the false reassurance of absence of radicular pain while injuring

the nerve root. Local anesthetic should be injected to the skin and muscles only, keeping the injection superficial to the articular masses. For the same reason we strongly recommend the performance of these procedures only under moderate conscious sedation, and discourage the use of general anesthesia or heavy sedation.

Colonic Perforation

A posteriorly placed colon can reside behind the psoas muscle and the spine. For this reason, the preoperative imaging studies, both CT and MRI, must be carefully examined to exclude the presence of such an anatomical condition, because bowel in the path of the instruments could be perforated, with the risk of peritoneal or disc infection or local abscess formation.

POSTOPERATIVE CARE AND FOLLOW-UP

We routinely perform percutaneous procedures on an outpatient basis. At the conclusion of the procedure, observation is needed for about 2 hours before discharging the patient home. Prescriptions are provided for a 2-week supply of a nonsteroidal anti-inflammatory agent and for diazepam at bedtime for 10 to 15 days.

At the end of the procedure, just before needle or cannula removal, we always inject steroids in the void created inside the disc. After nucleus ablation with physical energies (coblation, RF or laser), aspiration with a 20- to 50-mL syringe is needed before steroids injection, to eliminate the space-occupying gaseous or liquid residues. Injection of steroids is helpful in reducing the risk of an aseptic discitis consequent to inadvertent damage of the endplates, also reducing the back pain in the early postdiscectomy period. Steroids have also a mild proteolytic and sclerosing action, which could help reducing the disc volume in the postoperative period. Steroids and local anesthetics can also be injected, during needle removal, in the foramen, around the compressed nerve root.

Patients are encouraged to move, stand, and walk on day 3. After percutaneous disc decompression early activity is not only possible but also useful. It is imperative to avoid muscle atrophy and general deconditioning. Repetitive forward flexion, prolonged car driving, prolonged sitting, and lifting heavy weights are nevertheless prohibited for 3 to 4 weeks. Limb pain resolution may take weeks, owing to "remodeling" of the disc and regression of inflammation at the surgical site. The concept of disc remodeling must also be clarified to the patient. After decompression, reduction of profiles of the outer disc (annulus) is not immediate, and is directly related to the residual tissue resilience, the latter depending on both the age of the patient and the pathologic conditions of the disc. Disc remodeling may be fast, taking hour or days, but also much slower, taking several weeks. Consequently, also nerve root decompression may be expected to take the same time lag, and the patient must be aware of that.

A procedure that does not result in substantial relief of pain should not be considered a failure until at least 6 weeks have passed. Progressive return to heavy activities or sports is usually possible at 4 to 6 weeks. During the convalescence phase, rehabilitation measures applied by experienced physical therapists are an important intervention if a good outcome is to be realized. This program incorporates early to advanced intervention, as well as early to advanced stabilization techniques, progressive resistive exercises with emphasis directed toward mytotomal deficits, aerobic conditioning, and education. Among the concepts that need to be learned are maintaining a positive attitude, recognizing the difference between symptoms of a residual herniation and those of a healing process, and proper biomechanics. The only noteworthy side effect is the possibility of increased back pain. Most patients with a surgical wound have pain, and that applies to percutaneous discectomy. The intradiscal wound is more prominent and painful for mechanical procedure, particularly APLD, requiring a longer time for healing. Injury to skin, muscle, fascia, and annulus will occur, while a correct operative technique usually avoids injury to the endplates. Patients are warned that they may experience new back pain for up to 3 to 4 weeks. Patients should be encouraged to maintain as much mobility as possible despite the presence of this temporary back pain.

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25 Neuromodulation for Lumbosacral Radicular Pain

Timothy Deer, Jonathan Carlson, and Patrick W. Hogan

CLINICAL PRESENTATION

Lumbar radiculopathy is defined by an objective loss of sensory and/or motor function as a result of axon loss or conduction block in axons of a spinal nerve or its roots. Symptoms include numbness and weakness in the distribution of the affected nerve. Neurologic examination and diagnostic tests such as electromyography can help confirm the neurologic abnormality. Magnetic resonance imaging (MRI) or computed tomography with myelogram should also be ordered to assess pathology and anatomic variations. It is important to underscore that radicular pain and radiculopathy are not synonymous. The former is caused by ectopic impulse generation of nociceptive afferent fibers, whereas the latter relates to a conduction block or axon loss, which is manifested by actual physical examination signs [1]. Lumbar radiculopathy has neurologic components of paresthesias, numbness, hyporeflexia, and weakness. Patient description is usually sharp, shooting, lacinating, or shock-like pain that extends the length of the affected lower limb, typically below the knee [2]. The pain can be exacerbated by coughing, sneezing, back extension, Valsalva maneuver, and a straight leg test [3]. The most common spine-related etiologies for lumbar radiculopathy are herniation of the nucleus pulposus, central spinal stenosis, and nerve entrapment in the lateral recess [4].

TREATMENT

After gathering sufficient clinical data to exclude the presence of malignancy, infectious, or congenital pathology, the provider must then determine if the radiculopathy has associated myelopathy, which may necessitate surgical referral.

If the patient's pathology does not require immediate surgical correction, then conservative treatment measures are typically implemented to manage the symptoms. If the patient's main symptom is pain or numbness with no neurologic deficits, many spine surgeons will recommend conservative measures prior to surgical intervention.

A multidisciplinary approach for conservative treatment has been shown to have the best results when treating radicular pain [7]. Treatment modalities such as physical therapy, nonsteroidal anti-inflammatory medications, neuropathic pain medication, analgesic medications, chiropractic care, and epidural steroid injections are usually implemented. If these treatments fail then frequently the patient faces a decision between surgery or continued conservative management.

However, a growing trend is emerging wherein some clinicians are recommending their patients try neuromodulation as a less-invasive and possibly more cost-effective alternative to conventional spine surgery [8]. This option may be particularly valuable in those patients who do not meet the criteria for good surgical outcomes or who experience recurrent symptoms after previous surgery whose surgical construct is stable. In some patients, the presence of an incomplete fusion is a treatment dilemma. Persistent symptoms after aggressive surgical attempts at correction may lead to the request to implant a spinal cord stimulation (SCS) device. This decision is complex and should occur after consultation of both the surgeon and implanting physician.

Occasionally, those patients who fail conservative management and undergo traditional spine surgery will continue to have pain symptoms postoperatively. These patients are often diagnosed with "failed back surgery syndrome (FBSS)" or lumbar postlaminectomy syndrome. FBSS is the most common indication for SCS in the United States.

BRIEF TIMELINE OF NEUROMODULATION

Norman Shealey published the first SCS implant in 1967. This patient was a complex patient with cancer pain who by today's selection criteria would have been a less than ideal candidate. The patient had a good outcome despite these issues, and the medical community became aware of the possibility of spinal cord implants for pain control. During the next decade, the procedure was performed mostly by neurosurgeons who implanted subdural electrodes directly onto the spinal cord. Secondary to significant complications such as spinal cord injury and cerebrospinal fluid (CSF) leak, the trend progressed toward epidural placement of the electrodes. These operations were typically "open" procedures that required laminotomy or laminectomy to place the electrodes into the epidural space.

In 1974, Dr. Dooley implanted the first *percutaneous* SCS electrodes using an epidural introducer needle. Over the next 20 years, the technology in leads and generator systems continued to improve. The years following this report have seen the development and Food and Drug Administration approval of multicontact percutaneous leads, new anchors to reduce migration, new programming constructs, rechargeable generators, miniaturization of generators, new multicolumn paddle leads, and several advances in physician education.

MECHANISM OF ACTION

The gate control theory of pain, published in 1965 by Ronald Melzack, a pain psychologist, and Patrick Wall, a neurophysiologist, provided a scientific basis for the use of electrical stimulation to treat pain by proposing that a "gate" regulates transmission of pain sensations from the dorsal horn in the spinal cord to the brain. This gate opens when small-fiber (C fibers and A-Δ) afferents are unusually active and closes when large-fiber (A-β) activity is dominant [9]. This theory can explain how rubbing injured skin may reduce pain, or how a transcutaneous electrical nerve stimulator unit can reduce pain by distributing pleasant electrical sensations through electrodes on the surface of the skin.

Selective depolarization via neuromodulation of large-fiber afferents in the dorsal columns of the spinal cord theoretically "closes the gate" for pain transmission without causing undesired motor stimulation. This model may be an oversimplification of the true mechanism, and other mechanisms of action have been proposed. In reality, the precise mechanism of action remains undefined.

INDICATIONS

An implantable neuromodulation system is indicated for SCS as an aid in the management of chronic, intractable pain of the trunk, and/or limbs. Lumbar radiculopathy pain can be unilateral or bilateral and can be associated with the following conditions:

- Epidural/perineural fibrosis
- Inoperable spinal stenosis
- FBSS
- Radicular pain syndrome or radiculopathies resulting in pain secondary to FBSS or herniated disk
- Cervical postlaminectomy pain
- Multiple back operations
- Unsuccessful disk surgery
- Degenerative disk disease/herniated disk pain refractory to conservative and surgical interventions

CONTRAINDICATIONS

- Uncorrected coagulopathies
- Current sepsis/infection with fever
- Implantable cardiac defibrillator
- Inability to control device or lack of patient cooperation
- Thoracic syrinx

RELATIVE CONTRAINDICATIONS

- Thoracic stenosis [if < 10 mm for a percutaneous lead [10]]. This is a general consideration but many feel the presence of CSF at the level of implant on MRI would suggest safety of implant at that level.
- Patients who may require serial MRI evaluations (e.g., multiple sclerosis). This issue may now be less of a restriction since Medtronic has received labeling to do brain MRIs in the presence of thoracolumbar implants. The physician should consult with the manufacturer of the indwelling or proposed device.
- Demand cardiac pacemaker. Many patients have been implanted with prior indwelling pacemakers. It is important to consult with the treating cardiologist to assure non interference with the device and to consider having the technical expert from the Cardiac company present at the time of implant to monitor any cross talk or interference between the two devices.

PATIENT SELECTION

In the early development of SCS, patient selection was less than ideal. Because of the relatively new nature of the technology, there was limited evidence in the literature from which to base selection criteria. However, in the past 20 years, there have been improvements in defining proper selection to improve outcomes.

The decision to proceed with SCS typically occurs after failure of more conservative therapies and involves consideration of both patient-related factors and the characteristics of the pain itself. The practitioner should ensure that reasonable and less-invasive treatment modalities (injections, medication management, physical therapy, etc.) have failed. It is also important to consider available surgical options or that the patient has declined available surgical intervention. To maintain this modality as an option for future patients, maximizing cost-effectiveness by following a conservative approach may be prudent.

ETIOLOGY OF THE PAIN AND LIKELIHOOD OF PAIN REDUCTION

Regarding the characteristics of the pain, the best outcomes have been observed in patients with steady, lancinating, and burning neuropathic pain. Further, patients with unilateral radicular pain in an extremity have demonstrated improved outcomes.

However, with the advent of new technology such as dual and tripolar lead configurations, there have been great strides in outcomes for patients with primarily axial pain, bilateral radicular pain, and even pain relief for patients with a wider variety of pain syndromes.

A recent article by Deer and Masone discusses those disease characteristics that predict good outcomes in SCS patients. Those patients with a *high probability* of successful pain reduction by SCS include [10]:

- Chronic cervical or lumbar radicular pain syndromes
- Complex regional pain syndrome, types 1 and 2

- Painful peripheral mononeuropathies
- Angina pectoris refractory to conventional surgical bypass and medical management
- Painful ischemic vascular disease refractory to medical management or surgical intervention

There is *moderate possibility* of successful pain reduction in patients with:

- Axial low back pain
- Pelvic pain
- Visceral pain syndromes of the abdomen
- Post-herpetic neuralgia

There is *low probability* of successful pain reduction in patients with:

- Neuropathic pain following spinal cord or brain injuries, nerve root avulsions
- Iatrogenic nerve root destruction
- Phantom limb pain

In the United States, the most common patient population who experience benefit from SCS is patients with "FBSS." These are patients who underwent attempted surgical correction for a disorder of the spine and suffer from persistent pain in an extremity, their axial spine, or both.

Assuming that the patient's pain complaints fit into a category wherein there is likelihood of successful pain reduction, the next step in decision making is to consider factors unique to the patient.

PATIENT FACTORS

The patient's overall mental and physical health are important considerations in successful outcomes with SCS.

Regarding the patient's physical health, it is prudent to consider their preoperative risk for general medical complications, infection (diabetics, immuno-compromised patients, patients with systemic or local infections), bleeding (genetic coagulation abnormalities, chronic oral anticoagulant therapy), and also to assess whether they have a prohibitive degree of spinal stenosis or a syrinx. Many practitioners avoid introducing SCS leads into the epidural space of patients who suffer from a significant degree of spinal stenosis. However, if there is severe spinal stenosis, a surgical paddle-type lead could be placed following a decompressive laminectomy.

Assuming the patient's physical health is conducive to SCS, the practitioner must then consider the mental health of his/her patient.

First, the practitioner must assess the patient's overall baseline cognitive function. Patients with a low level of cognitive function may be unable to comprehend aspects of the technology such as recharging their device generator and using their hand-held programmer.

After ensuring adequate physical health and cognitive ability, the patient is usually sent for a psychology screening examination. The psychology screening evaluation is required by a majority of third-party payers in the United States, and the goal is to identify psychiatric abnormalities that would predict a poor outcome.

A typical psychology screening involves assessment by a licensed psychologist, including psychological interview (pain history, medication review, pain descriptors, psychosocial history, behavioral observations, and mental status examination), possible interview of a family member, spouse, or close friend, and key psychological screening tests (Minnesota Multiphasic Personality Inventory or MMPI, McGill Pain Questionnaire, etc.).

Patient expectations should also be addressed prior to undertaking the SCS trial. The goal of the trial and implant is for pain reduction (typically at least 50%) not necessarily "curing" their pain condition. If patients are prepared adequately for what to expect in the trial, and what constitutes a successful trial, there may be improved outcomes and patient satisfaction.

Psychiatric comorbidities (acute psychosis, personality disorders, severe depression/anxiety), significant drug or alcohol addiction, and issues of secondary gain should be addressed. The patient's baseline beliefs, attitudes, and expectations for the modality will likely play a role in the success of SCS.

Poor treatment outcomes have been correlated with the most psychologically and physically distressed patients, as well as those with depression and catastrophizing attitudes [11].

Patients with severe underlying psychiatric disorders may not be able to differentiate their pain from their anxiety, depression, or other pathology. A study by Burchiel et al. [12] demonstrated that measures of depression (via the MMPI), perception of pain intensity (McGill Pain Questionnaire), and advanced age predicted the patient's pain status 3 months following SCS implant.

A study by North demonstrated that low scores on anxiety and high organic symptom scores (per Derogatis Affects Balance Scale and Wiggins scales of the MMPI) predicted success of an SCS trial leading to permanent implant. These same predictors for pain relief from the SCS trial did not hold true at 3 months after SCS implant. The author acknowledged that no psychological predictors were identified for long-term success with SCS implant, however, additional studies were underway [13].

After establishing appropriate indication and patient selection, it is reasonable to proceed with an SCS trial. In the authors' opinion, one of the key benefits to SCS in the treatment of chronic pain is the temporary test trial. The test trial affords the patient the opportunity to experience the stimulation over the course of 3 to 7 days, wherein the patient can determine if the pain is alleviated by 50%, and if so the option for permanent implant is available.

The typical outpatient SCS trial involves percutaneous placement, using a 14-gauge epidural introducer needle, of one to three SCS leads into the desired position in the epidural space. During the procedure, a test stimulation and programming session is performed to confirm "coverage" by paresthesias over the patient's painful areas.

During the procedure, the patient is typically administered a light intravenous anesthetic so that he or she is

comfortable but able to communicate adequately during the intraoperative programming phase.

After adequate coverage of the patient's painful areas is established, the introducer needle is carefully removed and the leads are anchored with tape and/or suture material. Finally, prior to discharge home, the patient is then transported to a postprocedure recovery area and another programming session is undertaken to confirm and optimize adequate coverage of the painful areas.

The patient is then discharged home, often with oral antibiotic prophylaxis, and returns in 3 to 5 days for removal of the leads. The authors try to avoid making any changes in the patients' medication regimens during the time immediately preceding and during the course of the trial period itself to minimize any confusion on the outcome.

PROCEDURAL TECHNIQUES FOR PERCUTANEOUS SCS LEAD PLACEMENT

First, written and verbal informed consent must be obtained following discussion of the risk, alternatives, and benefits of the procedure. Preoperative antibiotics, 30 minutes prior to incision, is recommended prior to the SCS trial and is considered standard of care prior to the permanent implantation [14]. Next, careful attention to patient positioning is key. One must assure proper padding of the patient's extremities to avoid iatrogenic nerve injury, even if only light sedation is administered. The patient should also have adequate padding beneath his or her abdomen to allow for optimal opening of the lower thoracic/upper lumbar interspaces to facilitate entering the epidural space. Visualization of the desired interspace can be further enhanced by caudal tilt of the C-arm.

Regarding the type and level of anesthesia to administer, it is important to note that for the SCS trial, a minimal amount of anesthesia should be administered until the leads are properly placed. The patient must have an adequate level of consciousness to indicate whether the intraoperative neuromodulation covers his or her painful areas, and if not, the leads must be repositioned. The authors recommend that the sedation be minimized to the point that the patient is able to engage in meaningful conversation with the physician.

After proper positioning, meticulous sterile technique is advisable. Some providers suggest that their patients undergo a chlorhexidine shower at home, the evening before surgery. At a minimum, the patient should be prepared and draped in the usual fashion with povidone-iodine or chlorhexidine (some providers also advocate iodophor impregnated adhesive drapes). The use of a surgical mask, cap, and gown are also employed by many physicians.

Fluoroscopic guidance is utilized throughout the procedure, particularly during lead placement. The typical fluoroscopy views are AP and lateral views. The epidural space can be entered with the introducer needles at multiple levels. Previous back surgery usually precludes epidural entry at lower lumbar levels. Entry at T12-L1 may facilitate better lead control when placing the leads at the desired level (Figure 25.1). Entry into the epidural space above the level of the conus medullaris may facilitate easier lead placement, less irritation to the conus, and therefore improved patient comfort. However, there is potentially greater risk of spinal cord injury at any level above L2.

Once the entry level is determined, the skin is typically anesthetized with local anesthetic; the authors use 1% lidocaine mixed with sodium bicarbonate and epinephrine. The sodium bicarbonate that is added should be approximately 10% of the total volume (i.e., 1 mL of sodium bicarbonate in with 9 mL of lidocaine). The sodium bicarbonate hastens the onset of topical analgesia and decreases the burning sensation of the local anesthetic. The use of epinephrine optimizes vasoconstriction thus theoretically decreasing the risk of bleeding and hematoma formation.

A 22-guage 3.5-inch spinal needle can be used to anesthetize the deeper tissue to the laminae thus decreasing the risk of bleeding and improving patient comfort. Some specialists, however, do not advocate this because of the risk of intrathecal injection. A 14-gauge modified-Tuohy or RX-Coude needle can be used to enter the epidural space with a loss-of-resistance (LOR) technique. The question of whether air or preservative-free normal saline should be used to confirm entrance into the epidural space remains a decision based on clinical judgment. The current literature has shown either no significant difference or slightly improved safety with saline in numerous literature reviews for labor epidurals [15,16]. The superior LOR

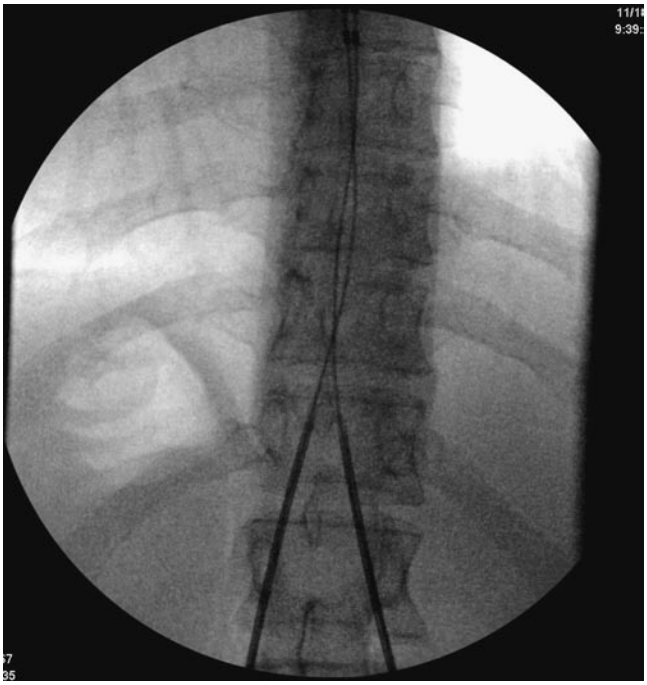

Figure 25.1 Demonstration using a modified tuohy needle to enter the epidural space at T11-T12.

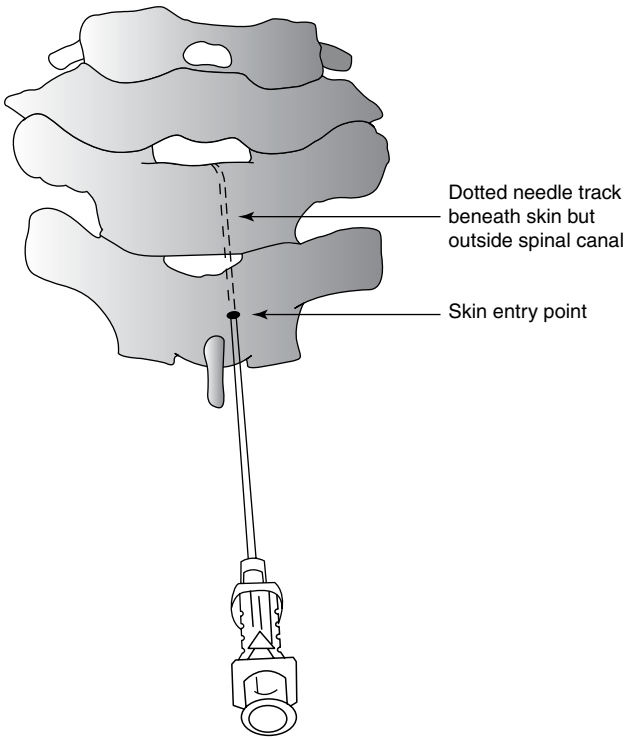

Figure 25.2 Needle entry point one and half interspace levels below the entry interspace. Courtesy of Epimed International. Used with permission from G. Racz.

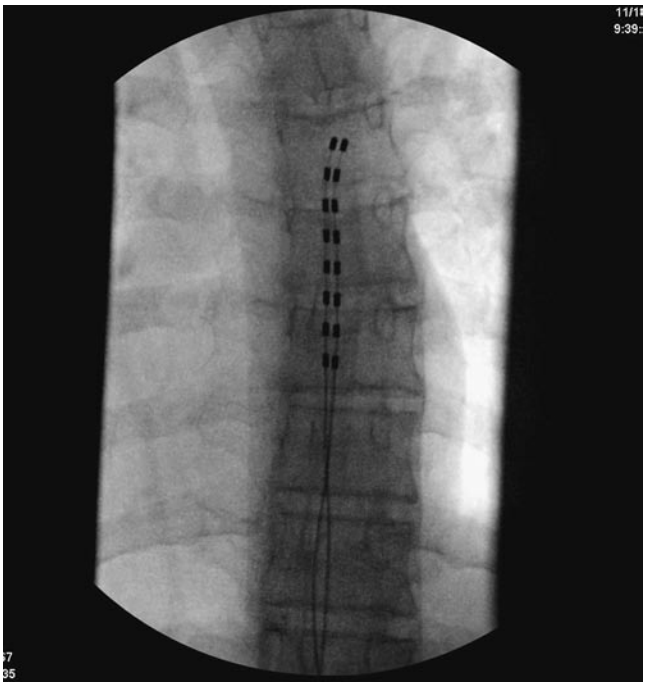

Figure 25.3 AP fluoroscopic view. Dual eight contact percutaneous leads placed in a patient with lumbar radiculopathy with symptoms of right-sided leg and foot pain.

technique has not been extensively studied in accessing the epidural space for SCS lead placement; however, some experts in pain medicine feel that the LOR with air is superior because there is less risk of current disbursement in the epidural space, thus theoretically improving the probability of a successful trial.

The needle-entry point at the skin is usually entered one and one-half to two interspaces below the epidural entry interspace, medial to the pedicle (Figures 25.1 and 25.2). The needle angle should no be greater than 30° from the skin. While using fluoroscopy in AP and lateral views, it is safest to contact laminae to gauge the needle depth prior to accessing the epidural space with the LOR technique. Some experts advocate entering the epidural space with the needle bevel down thus decreasing the risk of dural puncture and then turning the bevel up once the epidural space is entered. This has not been extensively studied and these suggestions are anecdotal in origin.

Placement of the leads should be done with fluoroscopic guidance (Figure 25.3). Neuromodulation should then be implemented and the leads placed in accordance with the patient's painful areas. The use of a single lead versus a dual lead array is at the discretion of the practitioner. A single lead array was shown to help both radicular pain and axial complaints in a few studies [17]. However, the technological advancements involving the use of dual four contact and dual eight contact leads have demonstrated better efficacy in relieving significant portions of the neuropathic axial component [18]. Placement of dual eight contact leads in a staggered array over the T7, T8, or T9 levels appears to increase the chance of capturing the axial neuropathic component of the low back [10]. If the decision is made to use dual leads, entry of the second needle on the contralateral versus ipsilateral side is at the discretion of the specialist; as the superiority of either technique has not been established. A study by Barolat et al. [19], Table 25.1, has mapped out sensory responses based on the level stimulation of the dorsal column via the epidural space.

The discussion of detailed neuromodulation programming technique extends beyond the scope of this chapter; however, it is recommended that lead location and programming be optimized at the time of the procedure. Once this is achieved, the leads should be securely anchored to prevent migration. If the percutaneous leads are placed for an SCS trial, the trial should not extend beyond 3 to 7 days to minimize the risk of infection and epidural adhesion formation. Lastly, fluoroscopic images with multiple planes should be stored to best duplicate lead placement for a permanent implant if the trial is considered clinically successful (Figure 25.4).

COMPLICATIONS

All invasive surgical, interventional spine, and interventional pain management procedures involve risks and

Table 25.1 Sensory Mapping in SCS

Location of Pain	Approximate Lumbar SCS Lead Placement
For low back and d/c the chest wall and abdomen	T7-T9, difficult to isolate without stimulating chest/abdominal wall
Buttock and thigh	T9-T11
Lateral at T11 to T12 groin hip and thigh	T9-T11 slightly lateral, also at T7-T8 of higher
Foot and Calf	T11-L1

SCS, spinal cord stimulation.

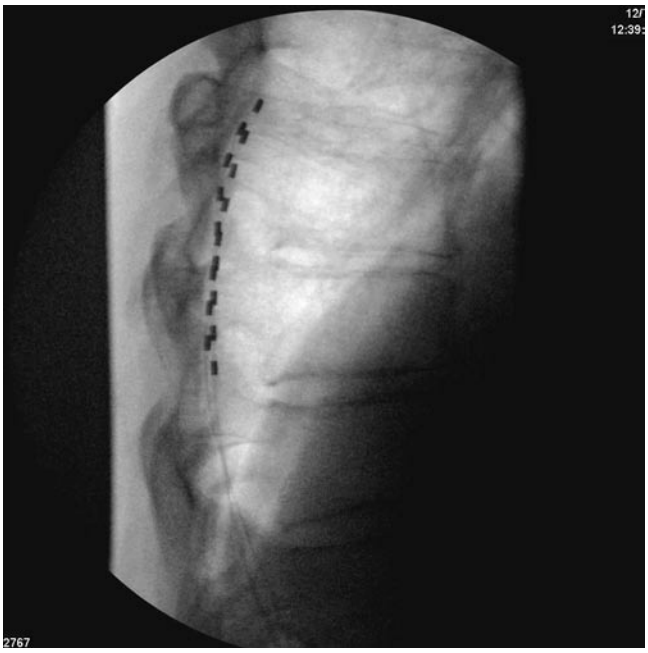

Figure 25.4 Lateral fluoroscopic view. Dual eight contact percutaneous leads placed in posterior epidural space in a patient with symptoms of right-sided thigh and distal leg pain.

potential complications which must always be weighed against the potential benefits of the treatment. Regarding SCS trial and implantation, the complications can include infection, bleeding, spinal cord injury, nerve injury, lead fracture, and lead migration.

The most common complication in the literature is lead migration, which can result in a change in the stimulation pattern and decreased analgesia. In a literature review by Monroe and Washburn, the overall incidence of lead migration was 13.5%. Their analysis included 67 articles published since 1981 that reported on 4634 patients. Lead breakage, which occurred in 7.6% of the patients, was the second most common complication [20].

The St. Jude researchers also presented results of an internal meta-analysis showing that lead migration occurred in 5.7% of 300 patients who received the company's Genesis stimulators. They also noted that the incidence of lead migration may be decreasing over time, presumably due to improved anchoring devices and techniques. Some experts advocate firmly securing the lead to the paraspinous fascia whereas others anchor the lead in the subcutaneous tissue. It is also important to leave a tension-loop in the anchoring site and generator pocket to allow for decreased tension on the lead during natural patient movement. Lead migration can also be reduced by educating the patient on the postprocedure activity restrictions such as prohibiting patients from bending and twisting for 4 weeks, until the leads heal into place. The incidence of lead fracture can also be reduced by avoidance of the midline both for entrance into the epidural space and during tunneling of the lead. The fracture usually occurs as a result of lead compression between bony surfaces. The symptoms of lead fracture include sudden loss of stimulation, and quick diagnosis can be made by verifying impedance data.

Inadvertent dural puncture is another frequent complication of SCS. The rate of this complication, even with experienced specialists, is usually estimated at 1%. Dural puncture can occur with the introducer needle itself or it can occur with advancement of the SCS lead through the introducer needle. The tip of the SCS leads is somewhat firm and can easily puncture the dura. The practitioner should have a preformulated plan for how to proceed if this complication does occur. There is insufficient data in the literature to establish a universal algorithm for management of an intraprocedural dural puncture. The decision is based upon the practitioner's clinical judgment and includes either abandonment of the procedure and rescheduling for 2 to 3 weeks later, or continuing the procedure at an alternate spinal level. The disadvantage of continuing the procedure includes the potential onset in the patient of a postdural puncture headache, which may impact the value of the trial or implant. Frequently, especially in younger patients, the headache will require epidural blood patch, which some experts argue may also increase the chance of infection during the trial or implant. However, abandoning the procedure is not without serious implications as well. For example, some patients may be forced to obtain a second authorization for the procedure from their insurance carrier and, therefore, risk denial of access to the treatment.

Another potential complication of SCS is infection. It is important to identify and optimize patients who are at increased risk for infection such as diabetics with poor glycemic control, patients on immunosuppressive medications, immunocompromised patients, patients with systemic infections, and patients with local infections near the procedure site. Further, many practitioners advocate pre- and postoperative antibiotics in addition to strict sterile technique. Early identification and aggressive treatment of superficial wound infections may prevent more extensive infection and avoidance of explantation of the device. Regarding permanent implants, the infection usually presents in the first 10 to 14 days following the procedure.

Bleeding at the generator pocket site and in the epidural space (epidural hematoma) are also serious potential complications. To minimize the risk of bleeding, it is prudent to identify those patients who are on anticoagulants

and to closely follow the accepted guidelines for perioperative management of anticoagulants for neuraxial procedures. The American Society of Regional Anesthesia published the most frequently followed guidelines for those interventional pain physicians whose primary background is anesthesiology [21].

Next, there is a risk of spinal cord and/or nerve injury during placement of the large-bore epidural introducer needle and during advancement of the SCS lead(s). It is possible to reduce this risk by ensuring an "awake" patient during the key portions of needle and lead placement (light anesthesia is acceptable and common) so that they can alert you should they experience paresthesias or unexpected pain. The use of fluoroscopy with frequent lateral images during needle advancement can help minimize inadvertent dural puncture and spinal cord injury. The typical LOR upon entrance into the epidural space is much less pronounced with the introducer needle than with the typical Tuohy needle commonly used for epidural injections. Epidural hematoma is also a risk given the large bore needle used, and practitioners should be well aware of the clinical signs and symptoms of this complication.

The generator pocket site is the source of several potential complications. First of these is seroma, which is a gathering of sterile fluid in the wound pocket. The incidence of seroma formation can be reduced by using a blunt dissection technique to minimize tissue damage, creating an appropriately sized device pocket (avoid excessively large pockets), and ensuring that at-risk patients have adequate serum albumin levels. The treatment of seromas can include the use of abdominal binders and although controversial, some experts suggest sterile needle aspiration.

The depth of implantation of the generator is another important and occasionally overlooked consideration. The generators are able to be recharged through the patient's subcutaneous tissue up to a certain depth, and therefore, the depth of subcutaneous implantation cannot exceed the manufacturer's recommendations. Practitioners should be aware of each manufacturer's criteria regarding generator depth.

Finally, surgical wound dehiscence is a more remote possible complication and unfortunately typically involves removal of the implanted hardware and reimplantation after adequate time for tissue healing.

OUTCOMES AND COST-EFFECTIVENESS

In a meta-analysis of 49 studies in which SCS systems were implanted for chronic pain patients who had more than 50% pain relief or a statistically significant reduction in Visual Analogue Score (VAS), investigators showed a long-term (> 6 months) success rate in more than 67% of the patients [22].

The medical costs of SCS compared with an alternative regimen of surgeries and other treatments in patients who respond well to SCS showed a payback period of 2.1 years or less. This includes factors such as the high initial cost of the system, periodic generator replacement, and revision [23]. A prospective study in 219 patients by Burchiel et al. indicated that patients with SCS, with whom a 1-year follow up was available, reported a significant long-term improvement in pain and quality of life. These patients had SCS placed for chronic back and lower extremity pain. These patients also showed marked improvements in pain intensity, social interactions, sleep, mobility, depression, and most aspects of daily living. This data, in combination with a low complication rate, demonstrated that SCS represents a relatively safe and effective approach in long-term pain management [24].

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26 Functional Restoration of Painful Lumbar Spinal Disorders

Michael C. Geraci, Jr. and Stuart M. McGill

OVERVIEW

The often heard statement "85% of low back pain (LBP) is of unknown etiology" [1] appears to have no scientific foundation, leads to frustration on the part of the clinician and the default diagnosis "nonspecific back pain," and despair on the part of the patient. Worse yet, it leads to patients being told that the pain is in their head, or that if left alone it will resolve in a few weeks. Interestingly, it is well documented that those who have chronic, and/or recurrent episodes of disabling back pain, are different from those who perform similar tasks and are symptom free.

Here is where the common wisdom is false. Sometimes the "bad backs" are not the weak backs when measured, but rather the stronger backs. When performing similar tasks, the "bad backs" move in a way to create more spine loading. But when measured, the "bad backs" have less torso muscle endurance than their pain-free colleagues. Those with more motion in their backs have a greater risk of future back troubles. In contrast, poor hip mobility is linked to back pain [2].

There is strong evidence that LBP has a cause (Chapter 1), that the cause can be determined (Chapter 3) and eliminated (Chapters 6–8,11,12,15,16), and that appropriate therapy is both rehabilitating and prophylactic. The way people with painful backs move and load their tissues produce nonoptimal motion patterns and motor patterns that both cause and result in inappropriate joint mobility and stability. Perturbed motion and motor patterns are both a cause and a consequence of back injury. Provocative testing can identify the patterns that cause pain, provide clues to eliminate the cause, and establish the most efficacious therapeutic exercise approach. This chapter discusses some issues linked to the assessment of patients followed with issues associated with corrective exercise. Because the spine is part of a body linkage, perturbed patterns anywhere within the linkage have the potential to disrupt the normal stress distributions causing back pain. A broad view of the interaction of the torso, hips, and lower limbs are of interest here.

THE CAUSE (AND ELIMINATION OF IT)

Although "psychosocial" variables are often attributed as a major cause of back troubles [3], only studies that neglected mechanical variables reached this conclusion. Larger studies that have measured both psychological variables and mechanical variables have found that although both are important, mechanical variables dominate the links to injury and pain [4,5]. In cases of the "failed back," too many patients are told that their LBP is in their head. However, subsequent assessment clearly determines a mechanical cause immediately. They were given a default diagnosis (i.e., "the pain is in your head") simply because they failed a treatment approach that was not matched to address the specific cause or lingering deficits. Most back troubles are the result of cumulative loading—not the culminating event that is requested on any injury report form.

For example, back pain that is exacerbated by repeated or prolonged flexion is very common in today's society. Giving this category of patient stretches such as pulling the knees to the chest may give the perception of relief (via the stimulation of erector spinae muscle stretch receptors) but this approach only guarantees more pain and stiffness the following day because the underlying tissues sustain more cumulative damage. This is also one of the indicators of discogenic pain; a flexion is a potent instigator of disc bulges [6]. Eliminating spine flexion, particularly in the morning when the discs are swollen from the osmotic superhydration of the disc that occurs with bedrest, has been proven very effective with this type of patient [7]. Many lifestyle and occupational examples are provided in the textbook "*Low Back Disorders: Evidence-based Prevention and Rehabilitation*" [8] to guide the elimination of the cause of a patient's back troubles—the physician will find that half of their effectiveness will be because of preventing the cause!

REDUCING THE RISK OF INJURY

No clinician can be fully successful without removing the cause and/or precipitating factors of painful spinal disorders in patients. This section links injury mechanisms and patient presentation so that the reader is able to better identify the causes of the back troubles and specific strategies to remove them. Controversial recommendations such as "when lifting, bend the knees and keep the back straight" rarely address the real issue, despite their popularity. Few patients are able to use this strategy in their jobs and furthermore, this is often not the best strategy. For

example, the "golfers lift" is much more joint conserving for repeated lifting of light loads from the floor given the preservation of a neutral spine posture. Using the criteria of quantified spine loads, muscle activation patterns, ensuring sufficient spine stability and the avoidance of injury mechanisms will reveal the most appropriate technique for every situation.

Spine sparing guidelines (see reference [8] for full explanation and evidence):

1. First and foremost, design work-tasks that facilitate variety.
2. During all substantive loading tasks, avoid a fully flexed or bent spine and rotate trunk using hips (preserving a neutral curve in the spine).
3. Choose a posture to minimize the reaction torque on low back as long as No. 2 is not compromised (keep the hand loads close).
4. Consider the "transmissible vector." Attempt to direct external forces (e.g., pushing and pulling) through the low back, minimizing the moment arm, which causes high torques and crushing muscle forces.
5. Use technique to minimize the actual weight of the load being handled (e.g., positioning boxes up on one corner or lifting only one end of an object such as a log).
6. Allow time for the disc nucleus to "equilibrate" and ligaments to regain stiffness after prolonged flexion (e.g., sitting or stooping) and do not immediately perform strenuous exertions.
7. Avoid lifting or spine-bending shortly after rising from bed.
8. Lightly co-contract the stabilizing musculature spine even during "light" tasks.
9. Avoid twisting and the simultaneous generation of high twisting torques.
10. Use momentum when exerting force to reduce the spine load (rather than "always lift slowly and smoothly"—this is an ill-founded recommendation for many skilled workers).
11. Avoid prolonged sitting.
12. Consider the best rest break strategies (Customize this principle for different job classifications and demands).
13. Provide protective clothing to foster joint-conserving postures.
14. Practice joint-conserving kinematics movement patterns.
15. Maintain a reasonable level of fitness.
16. Think about interactions between several of these guidelines.

ASSESSMENT AND PROVOCATIVE TESTING

Elimination of the possible "red flag" conditions that could be associated with new back pain is critically important and is a role generally very well performed. Then physical assessment usually consists of documenting the extent of motion (amount of flexion, for example) and some neural tests. How do these assist in designing the prevention and therapy strategies? The answer is they don't [9].

Each individual has a loading *tolerance*, which when exceeded will cause pain and, ultimately, tissue damage. *Capacity* is the cumulative work that an individual can perform before pain or dysfunction begin. Determining the tolerance and capacity of each individual is paramount for ensuring if therapy is matched to the patient. A patient who can walk 20 m before pain begins has a low capacity. Performing therapeutic exercise three times per week will exceed their capacity in each session. Rather, if the therapeutic exercise sessions were performed in shorter sessions but more often, exacerbating pain would be avoided. In this case, corrected walking in three shorter sessions per day, never exceeding their current tolerance and capacity, will enable the capacity to slowly grow. Then they will progress to one session per day with much more pain-free capacity.

SPINE-SPECIFIC ASSESSMENT

There are many tests that are helpful although the choice and suitability for a specific patient are based largely on their presentation and interview. For example, a patient who reports pain with a vacuuming task may be selected for the testing for compressive or shear load tolerance, which is easily performed in the office. For example, a seated patient compresses his/her spine by grabbing the side edges of the seat and pulling down (see Figure 26.1). The upright torso is stiffened with muscle activity. The test is repeated in a slouched posture. Discomfort when in the slouched posture shows a lower tolerance when the spine is flexed. This posture-modulated tolerance is powerful information for guiding the avoidance of damaging/exacerbating activity together with appropriate therapy design. In a similar way, testing whether neurological signs can be lessened through posture change is fruitful. Whether nerve mobilizing approaches have a good chance for success in sciatic patients is also revealed. Finally, recognizing aberrant motion and motor patterns that load the known pain-producing structures can be assisted with specific techniques (several of these are shown in *Low Back Disorders* [8]). An example of standing posture correction is shown in Figure 26.2. An example of aberrant movement includes instability catches observed as the patient moves through the neutral zone of spine motion that are usually better indicators than a measure of absolute motion. Working with the patient, muscular bracing patterns can usually be found to eliminate the pain. In contrast, some patterns will exacerbate and these are shown to the patient where they are coached and how to avoid the mechanism.

A critical question to ask is whether a patient reports that they have better and worse days. If so, there are some activities that help them and some that hurt them. Find out what they are and eliminate the exacerbating causes. For example, if sitting is not tolerated, avoidance of flexion with a lumbar support will help, together with organizing tasks to eliminate prolonged sitting. Specific exercises

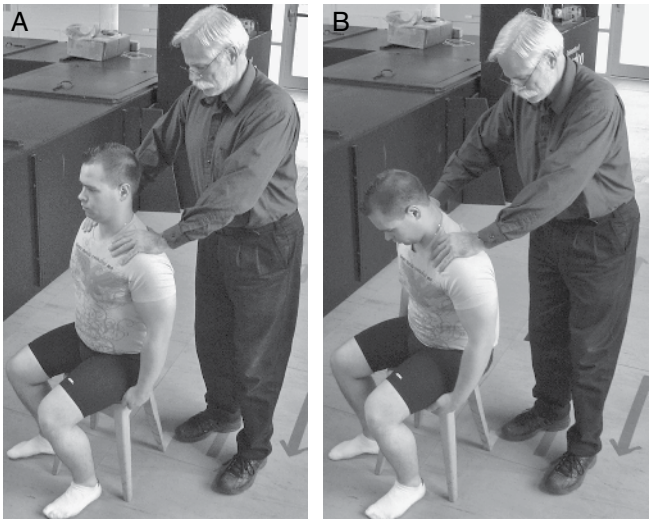

Figure 26.1 An example of provocative testing. The patient compresses the spine by grabbing the side edges of the seat and pulling up. When doing this with an upright back **(A)**, the torso is stiffened with muscle activity. The test is then repeated in a slouched posture **(B)**; discomfort in this position as compared to an upright back shows a lower tolerance when the spine is flexed (and a flexion intolerant patient).

designed to combat the cumulative stresses of sitting are then prescribed.

THE SPINE AND ITS FUNCTIONAL KINETIC CHAIN

Because many back disorders involve inappropriate movement and motor patterns, these variables are part of the treatment via corrective and therapeutic exercises. Given that the spine functions as a component of a multilinked system, disorders anywhere in the linkage have the potential to affect the spine.

Lower extremity dysfunctions, especially at the foot and ankle, as well as the hip have an influence on the lumbar spine. The foot and ankle connection, in particular, the subtalar joint (STJ), acts as a torque converter transferring STJ pronation into transverse plane lumbar motion. One of the best examples of this important connection is when a right-handed golfer on his back swing experiences LBP on the right side. If the left STJ has a pronation restriction, the tibia and femur will not have their normal amount of internal rotation necessary to move the pelvis into right rotation as the pelvis rotates on the relatively stable right femur. This results in the arms on the back swing rotating excessively, to compensate, which in turn will rotate the spine from above downward causing L5 to rotate the right relative to the sacrum. The golfer will seek manual treatments to address this lumbar dysfunction that will correct, only to recur every time he swings the club. The more likely treatment that results in long-term resolution of his LBP is to identify during a kinetic chain evaluation this left STJ pronation restriction and to correct it along with treating the L5 segmental dysfunction.

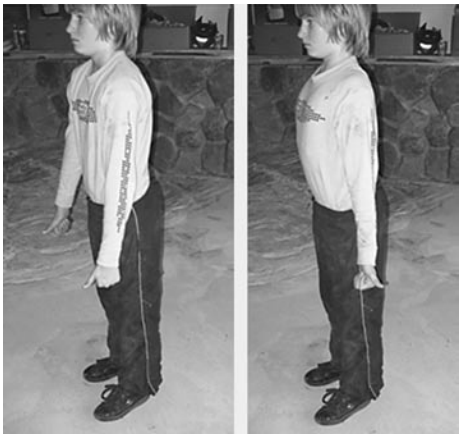

Figure 26.2 Standing posture correction could be accomplished in several ways. When directed to retract the shoulders, many patients with back pain shrug the shoulders. To prevent this, the instruction is to make a Hitchhiker's thumb and steer the arms/shoulders around in external rotation. Chin retraction also reduces the erector spine activity and removes the chronic muscle cramp from isometric contraction of the extensors without relaxants.

The foot and ankle connection to the lumbar spine is also demonstrated in a study that showed delayed electromyographic (EMG) activity in the gluteus medius on the ipsilateral side of the ankle instability [10]. When a group of runners who were able to run for 10 years injury-free were compared with a group of runners who suffered multiple injuries during the same 10-year period—the most important factors were related to the foot and ankle [11]. One of the three most important biomechanical factors identified was that in the injury-free group demonstrating a moderately rapid rate of rear foot pronation. This finding along with lower vertical impact forces and lower maximal vertical loading rate were the only findings that were responsible for the difference in the injury-free group compared to the group of runners with frequent injuries when evaluating multiple biomechanical factors.

Pathology has been shown in many muscles associated with various spine disorders. As an example, Nadler showed a statistically significant association between history of LBP and psoas muscle flexibility, leg length discrepancy and lower extremity instability [12]. This study along with others helps to support the relationship between the functional kinetic chain (FKC) and LBP. Therefore, an adequate evaluation of the FKC should be performed before treatment is initiated so that the dysfunctions of the FKC that influence LBP will also be properly addressed by treatment efforts.

EVALUATION OF GLUTEAL AND SCAPULAR MUSCLE FUNCTION

There are many different models of how to evaluate gluteal and scapular muscle function [8,13–16]. However, the approach that we will discuss is a relatively quick, easy, and practical way that evaluates these muscles allowing prescription of comprehensive and effective treatment. The global evaluation, looking at the big picture, begins

Table 26.1 The Six Basic Functional Tests

1. Squats: two- and one-legged
2. Balance and reach or step-downs
3. Core range of motion
4. Eccentric control of core
5. Scapular reaction
6. Unloaded foot/ankle evaluation

Theses start as a minimum with the two- and one-legged squats. If the patient is unsuccessful with these, then the balance-reach as well as the eccentric core control do not need to be tested as they also require one-legged support.

Figure 26.3 **(A)** One-legged squatting requires neutral spine and a hip hinge as well. Guard against the knee joint moving too far forward will increase patellofemoral forces and often causes anterior knee pain. Also note if the pelvis drops to the side of the unsupported leg or the trunk leans to the supported leg, stop the patient from squatting further. **(B)** The one-legged squat on the right leg shows better knee position and no compensations of the pelvis or trunk.

with six basic functional tests [13]. These are outlined in Table 26.1 (Figures 26.3 to 26.12). Each of the six tests does not need to be done. For example, if a patient fails the one-legged squat test, then the balance and reach as well as eccentric control of core will not be tested because they involve standing on one leg as well. When the patient has reached the threshold at which he/she fails a test, we can be selective about which subsequent tests we need to do and which ones we can predicate they will fail and need not perform. The squat exercise assesses the ability to maintain a neutral spine while using a hip hinge technique to activate the gluteal muscles placing less stress on the knee joints assuring they do not move too far forward over the toes. The same technique is used again for the one-legged squat, which can be done with a posterior reach and will provide better activation of the gluteus medius than reaches in other directions. Both types of squats are more easily performed with "tweaks" from above and below. For example, using "bending the bar" or "push hands" technique (Figure 26.13) better activates the latissimus dorsi muscle, which adds extensor moment and stiffness over the full lumbar spine to the sacrum to enhance lifting ability with more stability. Placing a strap around the distal thighs so the patient can externally rotate and abduct the hips against resistance better recruits the gluteal muscles.

Once we have an adequate assessment of the FKC, we can now turn our attention to the evaluation of the core and gluteal muscles. The muscles listed in Table 26.2 outline the muscles tested during nine exercises that were evaluated in a study to determine which exercises resulted in the greatest EMG activity [17]. Not surprisingly, the most activity was seen in exercises that include two of McGill's "Big 3"—the quadruped bird dog (Figure 26.14) and the side-bridge (Figure 26.15)—which are ideal for the early stages of rehabilitation where high muscle activation and low loads are placed on the spine.

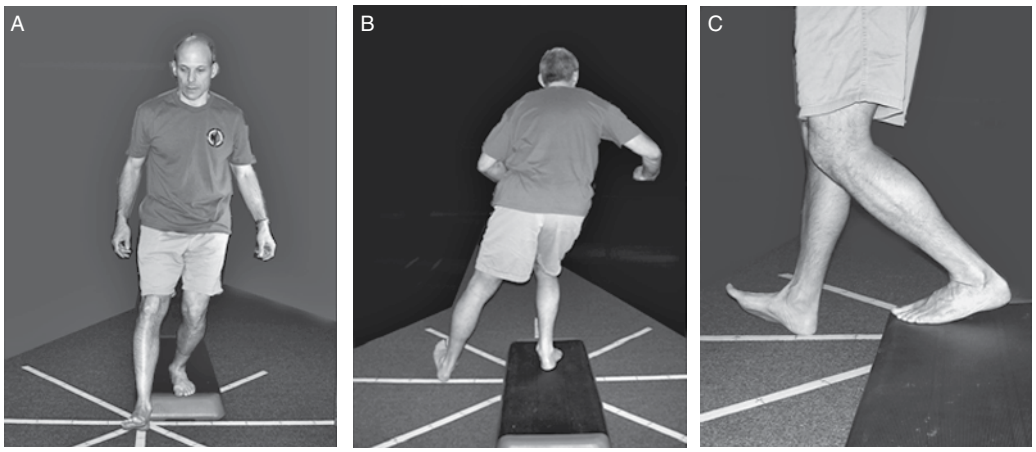

Figure 26.4 **(A)** Anterior step-down on left leg shows neutral spine, no compensation of the pelvis or trunk nor excessive forward movement of knee joint. **(B)** Medial step-down, posterior view, shows normal calcaneal eversion. **(C)** Anterior step-down on left leg, lateral view, demonstrates early heel rise, which indicates a tight soleus muscle or lack of dorsiflexion from a joint dysfunction.

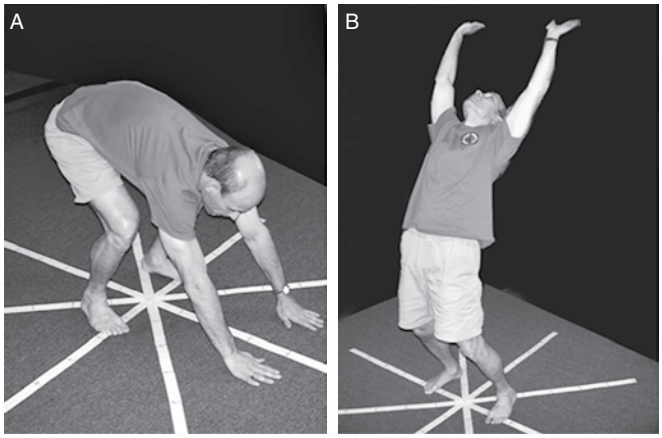

Figure 26.5 **(A)** Core sagittal plane (SP) motion with bilateral anterior upper extremity reach at floor height. This evaluates the patient's excursion at the hip, knee, and ankles. A posterior hip capsule restriction or tightness of the deep hip external rotators could restrict hip excursion. **(B)** Posterior bilateral overhead upper extremity reach evaluates hip excursion anteriorly and spine extension. Tightness of the anterior muscles such as the psoas, rectus femoris, or tensor fascia latae or a restriction of the anterior hip capsule could be responsible.

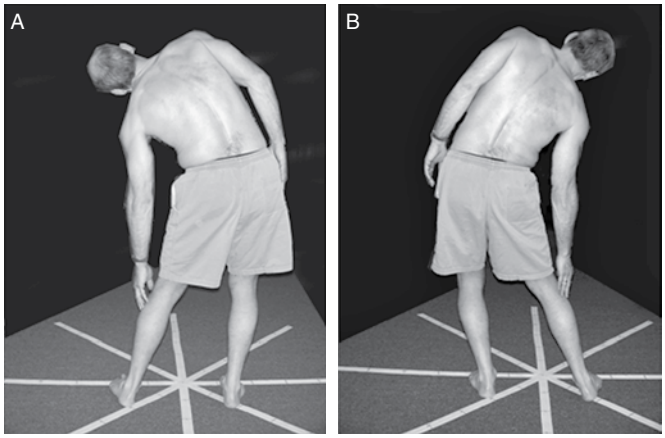

Figure 26.6 **(A)** Core frontal plane (FP) motion left side bending requires relative left hip abduction and right hip adduction. Observe for any straightening areas in the spine that may indicate a dysfunction of the paraspinal muscles or a joint dysfunction. **(B)** Right side bending here shows a straightening of the lumbar spine in the lower lumbar region.

Treatment should, therefore, begin with exercises that attack the successful positions in which a patient's muscles co-contract and place relatively low loads on the lumbar spine. If this does not occur in standing, even with tweaking from above or below, then the next position is determined by the so-called peeling back method. This is where kneeling, supine, side-lying eventually if necessary quadruped on hands and knees is attempted. This reverse neurodevelopmental approach is used by further peeled back to elbows and knees or even to a completely prone position [18,19]. Once the position of a good co-contraction of muscles is identified, the patient is trained in that position and built back up to standing.

Testing for compressive or shear load tolerance is easily performed in the office. Assessing whether lumbar spine flexion or extension provokes pain can be revealing. Testing whether neurological signs can be lessened through posture change or nerve mobilizing approaches have a good chance for success in sciatic patients is fruitful. Recognizing aberrant motion and motor patterns that load the known pain producing structures is also straight forward with specific techniques (these are shown in *Low Back Disorders* [8]).

If a patient reports that they have better and worse days, you know that there are some activities that help them and some that hurt them. Find out what they are and eliminate the causes. For example, if sitting is not tolerated, avoidance of flexion with a lumbar support will help together with rotating tasks to eliminate prolonged sitting. Specific exercises designed to combat the cumulative stresses of sitting are then prescribed.

THERAPEUTIC EXERCISE

Discussions of generic exercise for the LBP patient are not helpful and may even be harmful. For example, many Yoga and Pilates exercises may be appropriate for some but replicate the cause of dysfunction for others. The unstable back needs stability and probably mobility in the hips. The stiff back, not to be confused with a back splinted with muscle contraction, needs another approach. The older arthritic and stenotic spine needs yet another therapeutic exercise approach. But matching the exercise program to the specific patient has changed the lives of many a "failed back."

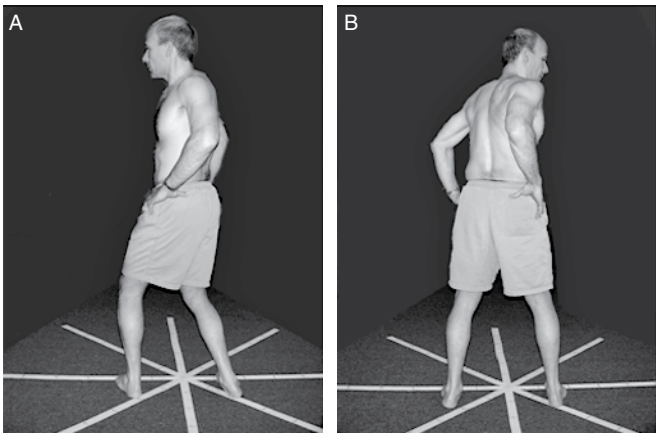

Figure 26.7 **(A)** Core transverse plane (TP) motion to the left shows a normal amount of right calcaneal eversion allowing the knee, hip, and pelvis to rotate to the left. **(B)** The TP motion to the right is clearly limited. This, in part, is due to the restricted left calcaneal eversion.

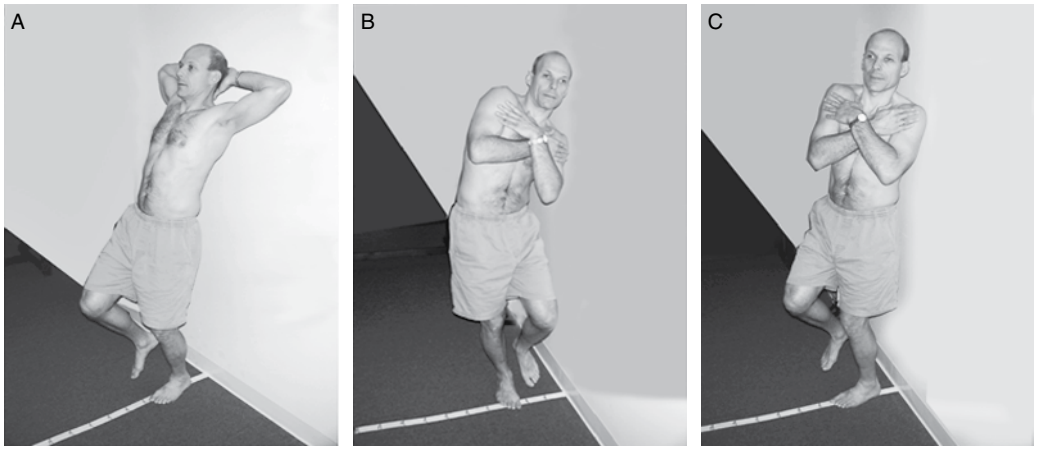

Figure 26.8 **(A)** Eccentric control of core (SP). The subject stands about 6 inches from the wall and is asked to place both hands behind their head and to gently tap their head to the wall and return to start position. **(B)** The FP eccentric control of the core starts with the subject about 7 to 12 inches away from the wall. They are asked to tap the shoulder and then return to the start position. **(C)** The TP eccentric control of the core starts the subject about 3 to 4 inches away from the wall. The subject is asked to tap each shoulder to the wall under control and return to the start position.

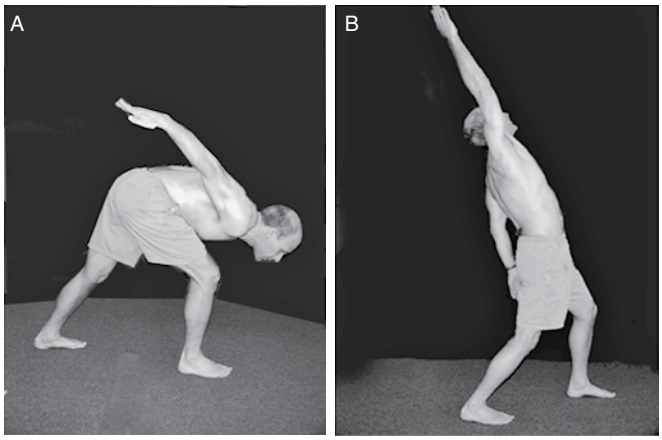

Figure 26.9 Scapular reaction (SP) evaluates the hip to scapular motion. **(A)** In the SP we first have the patient flex the hip, which helps the scapula on that side move into an anterior tilt. **(B)** If the hip does not extend fully then the scapula on the ipsilateral side will have difficulty moving into a posterior tilt.

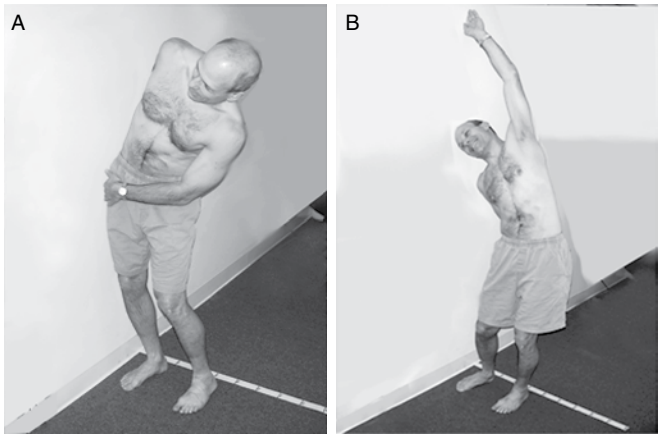

Figure 26.10 Scapular reaction (FP). **(A)** The subject is asked to tap the wall with the left hand across the body introducing left side bending of the trunk and scapular downward rotation ipsilaterally. **(B)** Then the subject takes the left arm and abducts it overhead to tap the wall. This motion introduces trunk right side bending and scapular upward rotation.

Therapeutic exercise must follow a progression. Given the perturbed motion/motor patterns that are both a cause and consequence of having back troubles, these must be addressed with corrective exercise first. Trying to strength train on perturbed patterns will guarantee chronic recurrent spine problems. Then muscle patterns that enhance spine stability must be repeated appropriately into the movement repertoire. Endurance of the muscles is then enhanced to ensure that the patterns are maintained despite challenges throughout the day. Only with this base can strengthening begin. As with all other stages, it follows a progression that is matched to the objectives of the individual. For those with athletic objectives, speed and power is then addressed. The science of therapeutic exercise design together with many examples of these five stages are documented in the textbook "*Ultimate Back Fitness and Performance*" [15].

The first stage of designing the appropriate corrective exercise emanates from the identification of any perturbed motion and motor patterns. Every exercise is considered within the working diagnostic hypothesis such that the first time the exercise is performed, it is considered a provocation test. If it is tolerated, the patient proceeds. If it is not tolerated the technique is re-examined and adjusted and/or a more tolerable variation is tried (see [20] for some examples with stabilization exercise). Many examples of corrective exercise are in my books and a couple of them are introduced here. For example, gluteal muscle activation retraining based primarily on the original work of Professor Janda has been honed in our own

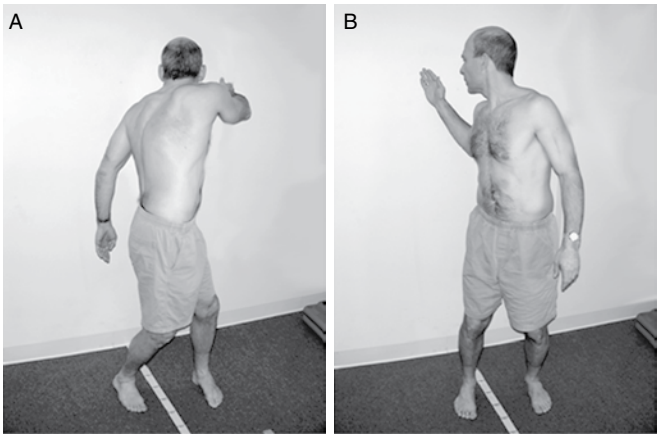

Figure 26.11 Scapular reaction (TP). **(A)** The subject is asked to rotate to the left and tap the wall behind them with the right hand at shoulder height. This requires the right STJ, knee, and hip to pronate and the scapula to protract on the ipsilateral side. **(B)** Next, the subject is asked to tap the wall behind them with the right hand requiring the left lower extremity to pronate to allow for contralateral scapular retraction.

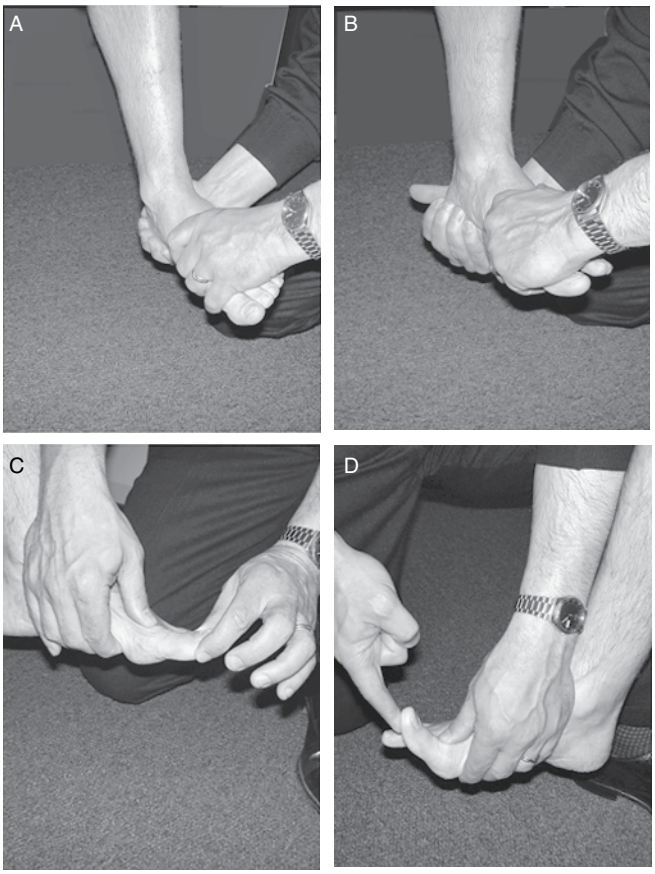

Figure 26.12 The unloaded foot-ankle evaluation. **(A)** The examiner introduces calcaneal eversion and checks the triplanar motion of the midfoot. All motions should increase with calcaneal eversion, which unlocks the midfoot. **(B)** Next, the introduction of calcaneal inversion is done with one hand and triplanar motion is introduced at the midfoot with the other hand. All motions, except inversion of the midfoot, decrease as calcaneal inversion locks up the midfoot. **(C, D)** The examiner next checks first MTP joint extension. There should be at least 65° of extension to walk normally. This unloaded foot-ankle evaluation allows one to determine if limitation of motion seen in loaded positions are from muscle tightness patterns or joint restrictions.

lab (Figure 26.16). This cannot be accomplished with traditional squat training [15]. Chronic back pain, despite the source, tends to cause hip extension using the hamstrings and subsequent back extension using the spine extensors creating unnecessary crushing loads. Gluteal muscle reintegration helps to unload the back. The next stage in the progressive algorithm is to groove patterns to ensure stability. Stability is considered at two levels—joint stability (in this case spine stability) and whole body stability. Quantification of stability proves that these two objectives are fundamentally different and need two different exercise approaches. Our observation is that the two types of stability are often confused in the clinic. We are dismayed to observe many unstable spines given gym ball exercises as a standard treatment course. Variations of our "Big 3" stabilization exercises have been quantified and selected for their ability to ensure sufficient spine stability and optimal motor patterns; they spare the spine of many injury mechanisms and pain exacerbators, and are designed to build muscle endurance (see Figures 26.14, 26.15, and 26.17). Then specific muscle group endurance is enhanced. Patient mastery of the details of exercise technique is critically important for this stage of exercise design. Success is not simply a matter of the patient performing an exercise—it is the patient performing the exercise with perfection (see Figures 26.18 and 26.19). Exercise form, subtle maneuvers to eliminate pain, pacing, duration, and other co-considerations are all extremely important. Spine stability requires that the musculature be co-contracted for substantial durations but at relatively low levels of contraction. This is an endurance and motor control challenge—not a strength challenge. For many individuals with painful low back disorders wanting to accomplish tasks of daily living pain-free, this is sufficient. In the preceding progressions, of course, strength is enhanced as are specific patterns such as the ability to squat, push/pull, lunge, and so on. But strength is not specifically trained because this requires overload and elevated risk; this is reserved for performance training. Many people, whether they have athletic objectives (such as wanting to play golf) or have demanding occupations, will fall into this category. On the other hand, many patients confuse health objectives (minimizing pain, developing joint sparing strategies) with performance objectives (which require risk) and compromise their progress with specific strength training too early. Many exercises typically prescribed to low-back patients are done so without the clinician having knowledge of the spine load and associated muscle activation levels. For this reason, we have quantified exercises in this way (see [8,21–23]) to allow evidence-based decisions when planning optimal exercise progressions.

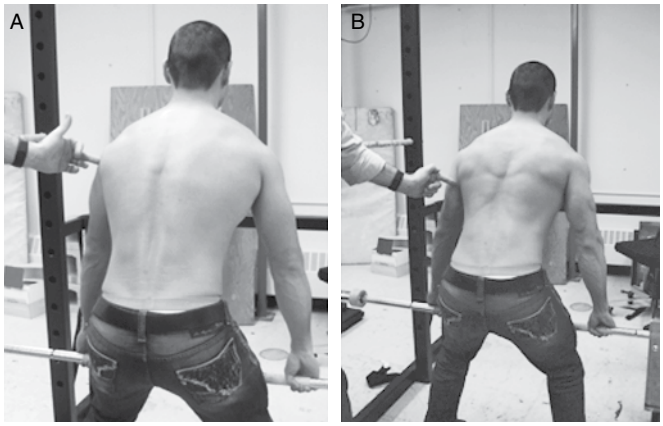

Figure 26.13 **(A)** The subject was asked to perform a squat. Note the low activation of the latissimus dorsi with this squatting technique. **(B)** Next, the subject is asked to use the "bending the bar" technique, which uses a tweak from above during the squat. In this technique, the subject is asked to attempt to "bend the bar" by bringing the elbows down and toward the back pockets. This technique activates the latissimus dorsi muscles, which add extensor moment and stiffness over the full lumbar spine to the sacrum to enhance lifting ability with more stability.

Table 26.2 Electromyographic Evidence of Core, Hip, and Thigh Muscles During Nine Rehabilitation Exercises

EMG activity of
Gluteus medius/maximus
Vastus medialis oblique/hamstrings
Longissimus thoracis
Lumbar multifidus
External oblique/rectus abdominus
Best exercises for core and gluteal muscles
Bridge-double leg
Bridge-unilateral
Prone on elbows/toes
Quadruped bird dog
Side-bridge
Exercises with less EMG activity of core and gluteal muscles
Good for endurance and vastus medialis oblique/hamstrings
Lateral step-up
Side-lying hip abduction
Dynamic skiers edge
Lunge

From ref. [17].

CAVEATS FOR THERAPEUTIC/ CORRECTIVE EXERCISE

Keep the duration of isometric exercises under 10 seconds and build endurance with repetitions, not by increasing the duration of the holds. Near infrared spectroscopy of the muscles showed us this was the way to build endurance without the muscles cramping with oxygen starvation and acid buildup.

Use the Russian descending pyramid to design sets and reps to make bigger initial gains in progress toward a pain-free back [8].

Maintain impeccable form to enhance available strength, and maintain the spine in its strongest (most tolerable) posture.

REHABILITATION EXERCISE—BIOMECHANICS AND CLINICAL PRACTICE

Rehabilitation is a staged process. My textbooks illustrate the many considerations and techniques to hone clinical skills at each stage (see list in sidebar).

Stages of therapeutic exercise:

1. Corrective exercise
2. Groove motion and motor patterns
3. Build whole body and joint stability
4. Increase endurance
   And for occupational/athletic clients:
5. Build strength
6. Develop speed, power/agility

Caveats and guidelines

For example, always begin with sagittal plane challenges (least spine load, then move to frontal plane, and finally transverse plane gives the very high spine load cost).

SUMMARY: SOME FINAL CONSIDERATIONS

First and foremost, assess the cause with provocative testing and teach the patient the specific positions and likely activities that harm his or her spine. Be careful with

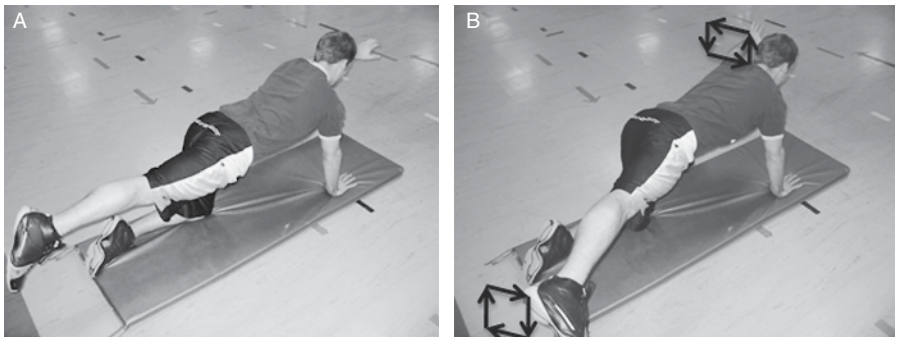

Figure 26.14 **(A)** During the bird dog, making a fist and co-contracting the arm and shoulder is a progression that enhances the contraction levels in the upper erector spinae. Drawing reciprocal squares **(B)** with the foot and hand with all of the motion about the hip and shoulder (none at the spine) engages more neuromuscular compartments in the erector spinae. An abdominal brace is helpful throughout.

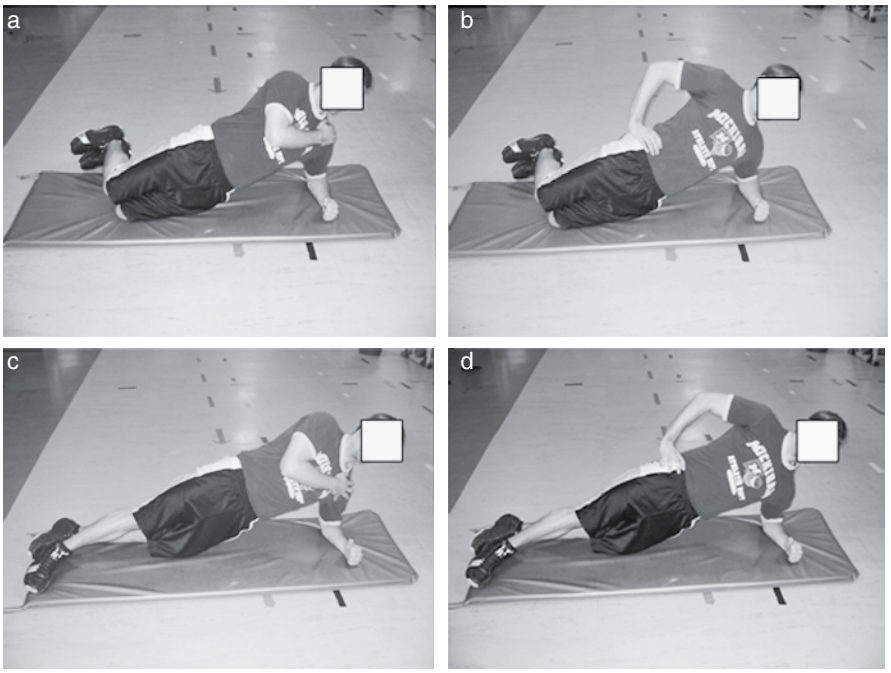

Figure 26.15 The beginner's side-bridge is held for sets of 10-second contractions before more challenging progressions are attempted. Four levels of a progression are shown **(A–D)**. Perfect spine alignment helps to make the exercise most tolerable.

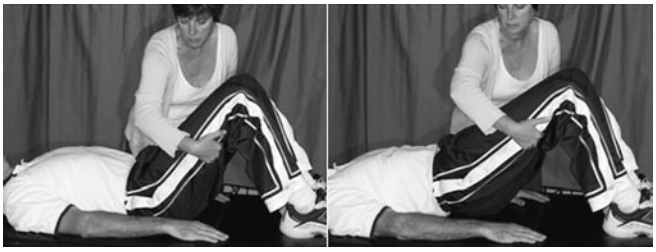

Figure 26.16 Chronic back pain tends to cause people to use their hamstring muscles, instead of their gluteals to extend the hip. This is linked to increased spine load when squatting. Performing the back bridge, squeezing the gluteal muscles, and eliminating hamstrings help to establish gluteal dominance during hip extension. Clinical cues are presented in McGill, 2009.

radiological diagnoses. They are often not associated with the tissues that are overloaded and painful, nor are they particularly good at finding tissue damage. Rather than initially attempting to find the tissue causing the pain, use an examination that synthesizes the findings of the interview process, with knowledge of injury mechanisms, and the findings from provocative testing.

Recocognize the cascade of degeneration and that some patients will be well along in the cascade with more painful tissues involved in their more complex presentation - do not dismiss them with psychosocial labels. Evidence exists demonstrating that biomechanical factors more strongly account for painful low back disorders than psychosocial variables. Address the mechanical causes of the pain and quite often the psychosocial concerns will resolve. Be careful with prescribing work hardening. Although this

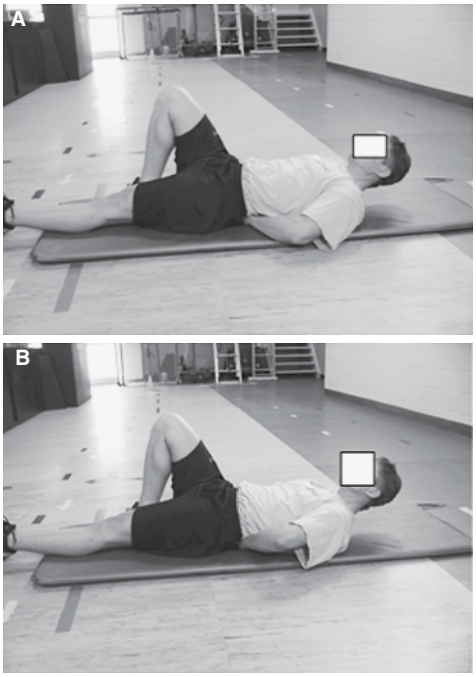

Figure 26.17 The "Big 3" stabilization exercises selected to create muscle patterns that ensure stability are the curl-up (shown here **(A)** elbows down, **(B)** elbows lifted from floor), the side-bridge, and the bird dog. Although we have quantified many variations and progressions, there are several cues for correct form. For example, during the curl-up, try and remove any motion from the lumbar spine and the cervical spine. Progression included prebracing of the abdominal wall, elevating the elbows off the floor, and breathing, to name a few. Note that there is hardly any motion.

Figure 26.18 Expert correction examples include fascial raking of the patient's obliques to intensify the brace during the curl-up. This increases the challenge to the musculature while keeping the exercise pain-free for many patients.

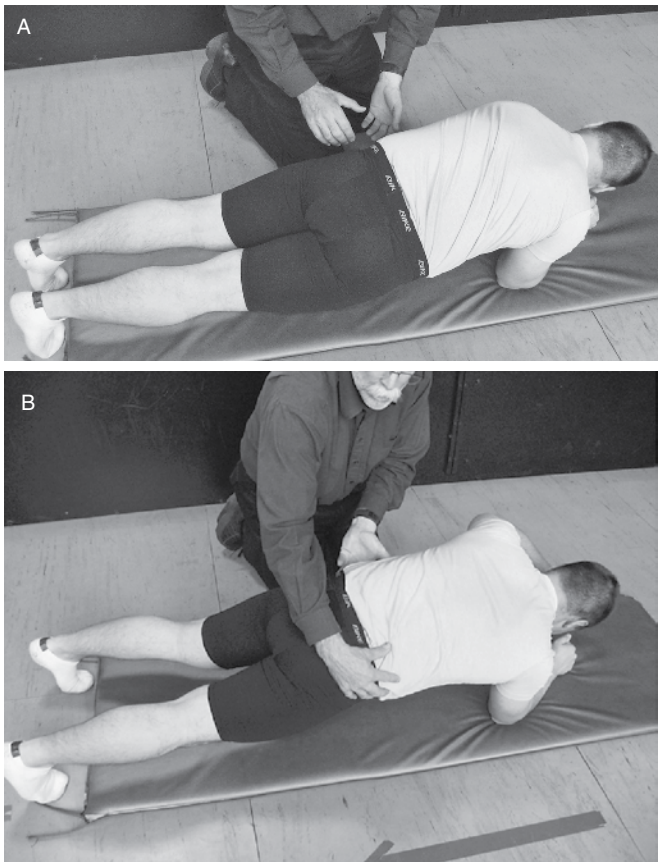

Figure 26.19 Correcting aberrant spine motion (there should be no motion between the rib cage and pelvis) during a side-bridge roll, which is one of the optimal exercises for challenging the neuromuscular compartments of the abdominal wall, quadratus lumborum, and the latissimus dorsi—all major spine stabilizers. **(A)** poor form, **(B)** corrected form being cued.

program may help some, for others their current tolerance and capacity simply will not allow them to keep up. These other individuals need a different approach to guarantee success. Prescribing muscle relaxants to a patient who stands with a torso flexed forward in a pain-induced antalgic posture will have little chance to be effective. Corrected standing will shut the muscles down.

Consider the following approach (if you are unable to perform this, then a referral to an appropriate spine expert familiar with this approach should be considered): The approach incorporates a strong biomechanical foundation, and blends expertise from various biomedical/psychosocial disciplines. First, an impression is formed from the first meeting of the patient in the waiting room—their sitting posture, how they rise from the chair, their initial gait pattern, and so on. Then a history is taken looking for possible candidate injury mechanisms, and perceived pain exacerbators and relievers. Observation continues during basic motion patterns as the examination proceeds delving further into the mechanics and nature of the symptoms. Then provocation tests are performed to either strengthen or weaken the hypothesis. Motion and motor patterns that are tolerated are identified. All of this data is used to formulate the plan for corrective exercise and the starting dosage of tolerable therapeutic exercise. The process concludes with functional screens and tests that were chosen on the basis of information obtained in the preceding process (the assessment process is well documented in *Low Back Disorders* [8]).

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27 Biomechanical Principles for Reducing Low Back Pain

Sue A. Ferguson and William S. Marras

INTRODUCTION

Biomechanics refers to the application of the laws of physics to the musculoskeletal system of the body for the purposes of quantifying the degree of load experienced by its constituent tissues. To define "safe" loading of the tissues, the load imposed upon the tissue of interest during the performance of a task is compared with the tissue tolerance. If the imposed tissue load exceeds the tolerance, the task is deemed risky. If the imposed load is below the tolerance, the task is considered safe. Traditionally, biomechanical principles have been applied to the spine and torso structures to design acceptable work conditions or to better understand the implications of surgical interventions. In this chapter, we will demonstrate how these same principles can be applied to those who have suffered a back disorder in order to better understand the potential risks associated with activities of daily living. In this context, we shall define "proper biomechanics" as the biomechanical conditions surrounding a daily living task that would be expected to reduce the risk of low back injury as well as reduce the probability of exacerbating a recurrent low back pain episode.

The Oswestry Disability Index assesses how an individual's low back pain interferes with activities of daily living such as lifting of heavy objects, personal care, traveling, as well as other activities [1]. We believe that the use of proper biomechanics during the performance of these activities minimizes the load on the spine during these activities. Reducing the load should minimize the low back pain experience and risk of exacerbating the pain, thereby reducing the effect of these tasks on disability reporting. Proper biomechanics often entails assessing the biomechanical demands of a task and altering how one approaches the task in order to minimize the load to the spine. For example, one may attempt to lift a heavy barrel in order to move it. However, instead of picking up the barrel it may be biomechanically beneficial to tip it on its side and roll it to its destination. Similarly, when travelling, use a backpack or suitcase with wheels that will allow the user to roll the backpack or suitcase instead of carrying it. This will minimize the load on the spine and reduce the risk of exacerbating an existing low back condition or having an initial low back injury. Proper biomechanics will reduce the load on the spine during the task reducing the risk of an initial injury and potentially the impact that low back pain may have on performing the task resulting in a reduced Oswestry Disability Index score. Thus, low back pain may have less effect on activities of daily living when proper biomechanics is used resulting in a lower score.

Proper biomechanics may reduce the risk of low back injury during activities of daily living; however, good ergonomic task design will minimize the risk more effectively. There is scientific evidence that tissue load is dictated primarily by the demands of the task. A field study examined 400 workers performing 200 jobs; one worker with a low back injury in the past 3 months and one healthy worker were examined on each job [2]. The risk of low back injury due to job was evaluated with a model that contained five risk factor characteristics, including frequency, moment, sagittal flexion, lateral velocity, and twisting velocity [3]. The results of the study showed that the risk of low back injury dictated by the job was the same for both the low back pain worker and the healthy worker [2]. Thus, the task requirements "drive" the physical exposure. Furthermore, all trunk motion measures evaluated coronal, sagittal, and transverse plane position; velocity and acceleration showed no significant differences between the asymptomatic population and low back injury group. These results indicate that the biomechanical requirements of a task are driven by the conditions of the task and not the particular manner in which the individual chooses to perform the task. Thus, the use of proper biomechanics is not going to reduce the risk of low back injury as much as "proper ergonomic design" of the task will reduce the risk of injury or exacerbation.

Given the significance of biomechanical loading in defining risk of low back pain development and exacerbation, the goal of this chapter will be to introduce the fundamental principles in spine biomechanics. Armed with this knowledge, it is anticipated that one could assess and design proper daily living conditions so that one could minimize the risk of experiencing low back pain events.

BIOMECHANICAL PRINCIPLES

Moment

The moment is the most important risk factor in minimizing the risk of low back injury in the workplace or in activities of daily living. The moment is defined as the weight lifted multiplied by reach distance as illustrated

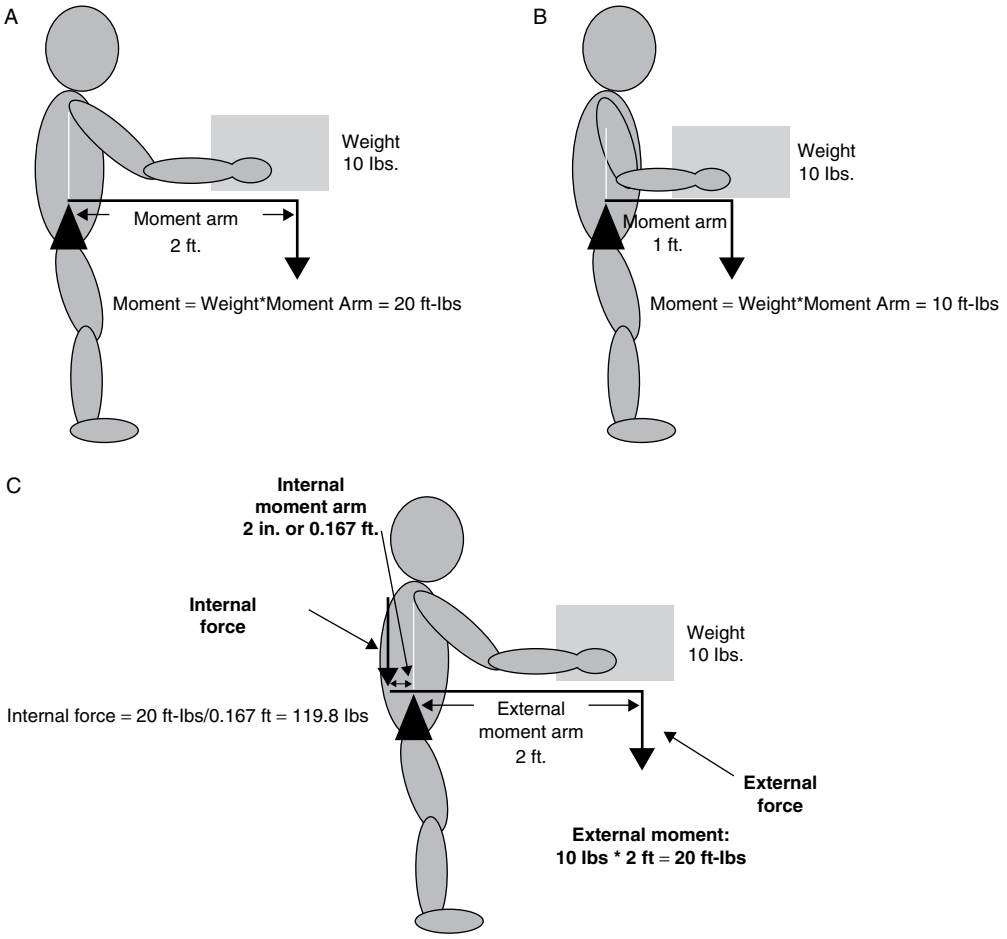

Figure 27.1 Moment with large moment arm resulting in **(A)** large spine load; **(B)** lower spine load; **(C)** internal versus external moment.

in Figure 27.1A. Hence, it is important to appreciate that the risk associated with excessive low back loading is *not* just a function of the weight of the object lifted but is really defined by how one is manipulating the weight. Figure 27.1A demonstrates that lifting a small 10 lbs weight at a reach distance of 2 ft creates a moment of 20 ft-lbs. Figure 27.1B illustrates that reducing the reach distance in half to 1 ft reduces the moment to 10 ft-lbs, thus, halving this applied load. Hence, that same weight creates a different load on the spine depending on the relative distance from the body from which it is moved.

It should also be recognized that the weight of the body segments can also impose moments about the spine. Thus, reaching outward from the body with the arms imposes a moment defined between the center of gravity of the arm and the spine. Similarly, bending forward from the waist can impose a large moment about the spine due to the mass of the torso.

Field studies have confirmed that the moment is the single most important predictor of incidence of low back pain in industry [3]. Furthermore, field studies have shown that the moment is also predictive of recurrent low back pain [4]. Thus, when designing tasks in the workplace as well as setting up activities of daily living, it is important to consider the weight of the task and the reach distance. The reach distance should be minimized to reduce the moment and subsequent risk of low back injury. Furthermore, minimizing the reach distance during activities of daily living may reduce the effect of low back pain on these activities and reduce disability scores on tasks such as lifting.

Internal and External Loading

Two types of forces can impose loads on the spine during work and activities of daily living, and one must develop an appreciation for the magnitudes of these forces to understand the risk associated with various task conditions. Figure 27.1C illustrates the two types of forces. First, external loads refer to the forces that are imposed on the spine as a direct result of the forces of gravity acting on external object being manipulated by an individual or the forces of gravity acting upon a body segment. The 10 lb weight in the previous example can be considered an external force (external moment). However, in order to maintain equilibrium, the external load must be counterbalanced by a load that is supplied by the muscles and ligaments internal to the body (internal force). Figure 27.1C shows the internal force (muscle) acting at a distance of

approximately 2 inches, which is much closer to the center line than the external load. Thus, the internal load or force is at a biomechanical disadvantage and, thus, the force must be much larger than the external load to maintain musculoskeletal equilibrium.

The previous discussion has introduced an important concept of external loading and the relationship to the loads imposed on the spine. Proper ergonomic design involves designing the task so that the internal loads are minimized. Internal loads which are supplied by the muscles may vary greatly among individuals. Laboratory studies examining spine loads in healthy and low back pain patients found that low back pain patients experienced greater spine loads compared with healthy individuals while performing the exact same task [5]. This increased spine loading was due to greater muscle co-activation in the low back pain population. In other words, low back pain patients were contracting muscles unnecessarily while performing the task [5]. This trend was true for all the lifting tasks performed but it was most apparent in the least physically demanding tasks. Thus, the external moment may be the same for all individuals but the internal loading may change as a function of the muscle co-activation patterns. It should be noted that these individual differences in spine loading due to muscle co-activation patterns are small in comparison to differences in spine loading due to task design. Thus, good ergonomic design of a task will minimize the risk of low back injury more than individual differences in muscle co-activation patterns.

Supporting the Torso to Reduce Moments

It should be recognized that supporting the torso weight with the arm while bending forward will reduce spine loads. A laboratory study examined how spine loads changed while lifting from a bin with one arm supporting the torso weight versus no support [6]. The results of the study indicated that spine loads were reduce by at least 15% in all three planes (compression, lateral shear, and anterior/posterior shear) during the one arm support condition compared with no support. The spine loads were reduced because the weight of the torso was supported by the extended arm instead of the muscle of the torso. This knowledge may be applied to any activity of daily living. Supporting the torso with one arm while bending forward will reduce spine loads and thus the risk of low back injury or exacerbation of existing conditions.

Length-Strength Relationship

The ability of muscle to generate a force is directly related to the length of muscle at the time when it is activated. The muscle can generate the most force when it is at resting length. When an individual bends forward with the back to pick up even a small weight, the muscle is elongated and has less force-generating capability compared with an upright posture. Epidemiologic studies have shown for decades that forward bending is a risk factor for occupational low back injuries [7,8,9,10].

Laboratory studies show that spine loads increased with bending. In a palletizing study examining three height levels, the spine loads (compression, anterior/posterior shear, and lateral shear) were the greatest at the bottom or floor level [11]. In addition, the spine tolerance is lowest in that posture since flexion decouples the posterior elements of the spine thereby forcing the entire load to be borne by the discs. The dangers of lifting from floor level have been further examined in both healthy and low back pain patients. Recently developed lifting guidelines based on spine loading and specifically anterior/posterior shear loading indicate that healthy individuals are at high risk of low back injury when lifting 25 lbs at floor levels [12]. This guideline was based on anterior/posterior shear loads exceeding the 1000 N threshold during lifting tasks. These same guidelines indicate that those with low back pain are at high risk of exacerbating their symptoms when lifting 15 lbs from the floor. Thus, when designing activities of daily living or workplace tasks, it is imperative to keep items up off the floor to minimize the risk of low back injury or exacerbation. Furthermore, keeping items up off the floor may reduce the effect of a low back pain condition on activities of daily living thereby potentially reducing an Oswestry disability score.

Twisting and lateral bending have also been shown to increase muscle co-activation levels resulting in increased spine loading [13,14]. The increased spine loading during twisting or lateral bending would result in increased risk of low back injury during activities of daily living or work tasks. Thus, awkward trunk motion in all directions (forward/back, side to side as well as twisting) should be avoided during work tasks or activities of daily living to minimize the risk of low back injury or exacerbation of existing conditions.

Force-Velocity Relationship

Dynamic motion of the trunk decreases the strength capability of the trunk in all three directions [15]. Thus, the faster the trunk is moving the less force the trunk is capable of producing. Thus, the tolerance level is lowered and makes one more susceptible to lumbar spine tissue disruption. Field studies have shown that lateral and twisting velocity of the trunk in combination with workplace factors predict whether or not a job will be in the high-risk group for low back pain reporting [3]. More recent field studies have also shown that lateral velocity capacity is predictive of recurrent low back pain [4]. Research has also shown that range of motion recovers first followed by velocity and acceleration [16]. In a field study examining workers with recent low back injuries returning to work full duty, it was shown that approximately 60% of those returning full duty were recovered based on range of motion and only 13% were recovered based on velocity. The lack of velocity recovery and the risk of injury increase at increased velocity in the workplace may increase the likelihood of recurrent low back injury or exacerbation.

Frequency

It has been well established in the literature that frequency of exertions (lifts per hour) can be a significant risk factor for low back injury in the workplace [3,8,10,17,18]. A

recent laboratory study has shown that frequency of loading increases spine loading as a function of time [19]. This increased spine load results from increased muscle co-activation that occurs at certain lifting frequencies. Furthermore, as the muscles fatigue, the muscle recruitment patterns change and make the spine more susceptible to instability.

Integrating Biomechanical Information

Collectively, if one can develop an appreciation for how these various biomechanical principles interact during the performance of a task, then one can better understand the biomechanical risks to the lumbar spine associated with the conditions. To appreciate the interaction between these biomechanical variables, biomechanical models are often employed.

Figure 27.2 Poor task design results in bad biomechanics and high risk of low back injury.

BIOMECHANICAL APPLICATIONS IN TASK DESIGN

Design Principles

Design guidelines for workplace and activities of daily living can be derived from the biomechanical principles discussed in the previous section. Acceptable load moments have been summarized by the American Conference of Governmental Industrial Hygenists [20]. These guidelines are for healthy individuals with no history of low back pain and indicate that 32 kg (70 lbs) directly in front of the worker and close to the body is safe. When moving loads held at 60 to 80 cm, 9 kg (20 lbs) is considered safe. In general, the concept is to move the object as close to the body as possible. These guidelines also incorporate the length/strength relationship. The acceptable weight limits change as a function of the height. Ideally, one should lift from just below waist height. As objects are lifted away from these ideal zones, the acceptable weight levels are reduced. To keep a good work design minimize travel distance and maintain a reasonable pace, which should result in a reasonable muscle velocity. It is imperative that frequency of task be considered in guidelines for tasks.

Strategies to Apply Biomechanics to Activities of Daily Living

One common activity of daily living that most individuals perform on a regular basis is grocery shopping, which may be quite physically demanding. When designing your tasks for grocery shopping keep in mind the tasks design principles. Many grocery carts are designed with a shelf on the bottom that grocery store employees often indicate are for heavy items. This shelf on the bottom creates a bad ergonomic design as shown in Figure 27.2. Placing the heavy item on the bottom shelf requires an individual to lift from near floor level bending the back. This task design increases the risk of low back injury or exacerbation of existing conditions. Good task design in Figure 27.3 would be to put the heavy item in the top of the grocery cart as shown. This eliminates the forward bending required in the other task set up, which would minimize the risk of low back injury during activities of daily living. Loading the grocery bag into your vehicle may also create a high-risk situation. Remember to minimize the reach distance when loading the grocery back into the vehicle. Finally, when unloading the grocery bags at your home do not carry all the bags once. Make multiple trips from the car to reduce the weight being carried. Thus, employing the biomechanical principles of this chapter in activities of daily living should minimize the risk of low back injury.

The Oswestry questionnaire [1] evaluates how low back pain affects traveling. Biomechanical principles can be applied to traveling in order to minimize the impact of existing low back pain on travel as well as minimize the risk of an initial injury. First, if you are carrying an item, place it in the vehicle before you get into the vehicle. When placing the item in the vehicle minimize the reach distance. To minimize the reach distance, one may place the item in the trunk rather than the back seat area. Another option may be to place an item on the passenger side. However, do not reach across from the driver's side as this will increase the reach distance. Instead, go around the vehicle and lift the item in and out of the vehicle from the passenger side door. This will minimize the reach distance. In addition to minimizing the reach distance, one should also minimize bending and twisting postures during the entering and exiting process. This can be accomplished by sitting on the seat and then bringing the legs into the vehicle together. Also when exiting the vehicle turn in the seat and place both legs on the ground and then stand up to exit the vehicle. This will minimize any twisting of the spine which may result in shear loading on the spine. Enter and exit the vehicle with no weight in your hands. Use your hands to support the body as you ingress or egress the vehicle. This will minimize spine loading.

Activities of daily living also include personal care such as washing and dressing [1]. While performing these activities, minimize forward bending, which will minimize spine loads. Another option would be to support your trunk with you arms on the sink area. Supporting

Figure 27.3 Good task design results in good biomechanics and low risk of low back injury.

the torso with the arm will reduce the muscle activity required to support the trunk, which will result in reduced internal spine loading. This will minimize spine load and reduce the risk of exacerbation or initial low back injury. Thus, applying biomechanical principles to activities of daily living and workplace tasks will reduce loads on the spine resulting in reduced risk of lumbar spine injury or exacerbation. Furthermore, the impact of an existing low back pain condition on activities of daily living may be minimized by applying biomechanical principles to these activities.

The Oswestry questionnaire examines how low back pain influences the length of time one can sit in a chair [1]. Biomechanical principles may be employed during the activity of daily living of sitting to extend the time tolerance to the activity. Many chairs have adjustable seat back angles. While sitting in a chair adjusting the seat back angle at 110° to 120° will transfer the upper body weight of the individual to the seat back and reduce the load on the spine. Transferring the load of the spine onto the chair would result in lower levels of muscle activity and in turn result in lower spine loads. Decreasing the load on the spine during sitting may increase the time of exposure to the sitting task resulting in better scores on the Oswestry disability questionnaire. Thus, applying biomechanical principles to activities of daily living may influence the scores of these disability questionnaires.

SUMMARY

These are just some examples of applying biomechanical principles to activities of daily living. Applying biomechanical principle to your specific activities of daily living will reduce the external loads on your spine and in turn minimize the internal loads on the spine. Minimizing the internal load will reduce the risk of low back injury and pain, as well as reduce the risk of exacerbating existing low back conditions. Applying these biomechanical principles to your activities of daily living will help maintain low back health and reduce the risk of disability.

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Part II
Cervical Spine

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28 Pathophysiology of Painful Cervical Spine Disorders

David G. Vivian and Paul E. Verrills

INTRODUCTION

The pathophysiology and epidemiology of neck pain has been relegated to the realms of psychogenic and degenerative disorders without due deference to science. The myth that neck pain is caused by disc degeneration (DD) continues to be promulgated [1]. To understand the pathophysiology of neck pain it is essential to realize that DD is not an age-related phenomenon, and that although it might be a painful process and lead to painful sequelae, there is currently no simple method to assess whether or not a radiologically determined degenerate disc is in fact the source of pain. The perpetuation of such a diagnostic label contributes to the morbidity of neck pain, as many patients have a belief that degeneration and spondylitis are the source of the pain, and as these are progressive conditions, they perceive that they are doomed to a life of pain. It is essential that clinicians understand that the diagnosis of degeneration, spondylitis, and osteoarthritis is invalid and alarming to our patients. Disability from pain is significantly related to fear [2], and iatrogenically induced fear can be instilled if radiologic labels are used to diagnose axial pain.

Painful cervical spine disorders fall into two separate categories: those conditions causing neck pain with or without more distant referred pain, and those causing radicular pain. To confuse matters, neck pain may coexist with radicular pain, and neck pain can have nociceptive and neuropathic pain qualities.

The sources and causes of neck pain and cervical radicular pain are different. The source of pain refers to anatomical site of pain origin. The cause of pain refers to the pathophysiological mechanism responsible for the source inducing nociception. In neck pain, the sources and causes of pain are often elusive, and the diagnostic label "idiopathic neck pain" is now considered reasonable [3] at least for non-whiplash or atraumatic neck pain. In reality, although terms such as *whiplash-associated neck pain* have arisen, such a "diagnosis" does not assist in identifying either the source or the pathophysiological mechanism, and are meaningless other than describing an event which may or may not be a contributing factor in the genesis of a particular symptom.

The management of neck pain and cervical radicular pain are fundamentally different. Diagnostic confusion can arise if these pain types coexist, or if the upper limb pain of cervical radicular pain is associated with more proximal pain, or if neck pain is associated with referred pain that extends into the arm. In each case, careful clinical assessment will assist in this triage process.

In some presentations of neck and radicular pain, the pursuit of target-specific origin of pain holds merit where pain fits with circumscribed pain patterns as described for disc, facet joint, and radicular pain. The challenge for clinicians when confronted with a more widespread pain presentation such as fibromyalgia is to be able to recognize and communicate the fact that routine investigations including magnetic resonance imaging (MRI) and specific percutaneous diagnostic procedures may be unhelpful and perhaps contraindicated.

The concept of tissue-specific origin of neck pain derives from studies on both asymptomatic normal volunteers and symptomatic patients who have undergone either pain provocation or anesthetization of potentially painful structures. Despite the fact that interpretation of these studies, particularly pain provocation, should be taken with some reservation in terms of specificity [4,5], they nevertheless provide useful information on the nature of referred pain from the various cervical spine structures. They demonstrate that segmental referral has distinct patterns; upper cervical structures are more likely to produce headache, for example, whereas lower cervical structures are more likely to produce scapular pain (Figure 28.1) [6].

PREVALENCE

Neck pain is extremely common, particularly in women, having a lifetime prevalence of 70%, a 1-year prevalence of 40%, and a point prevalence of 10% to 20% [7]. In working populations, it is very common, to the point where it can be considered almost normal! For example, in a study of 495 female kitchen workers the 3-month prevalence of any musculoskeletal pain was 87%, the most common sites being the neck (71%), low back (50%), and forearms/hands (49%) [8]. Multiple pain sites was the norm, as about 73% of the subjects had pain in at least two sites, 36% in four or more, and 10% in six to seven. In another study on office workers, the 1-year prevalence of neck pain was 45.5%, with additional demographic findings revealing that women have an almost twofold risk compared with men, that persons older than 30 years have 2.6 times more

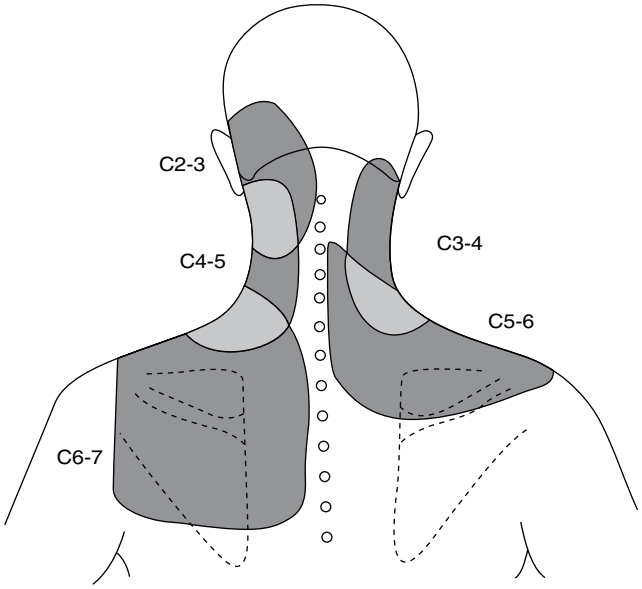

Figure 28.1 ZJ referral patterns: note that the C5/6 and C6/7 segments refer pain into the scapular region. Adapted from Ref. [6].

chance of having neck pain than younger individuals, and that being physically active decreases the likelihood of having neck pain [9]. Pain of any description is common in the workplace: in a study of 4006 workers from industrial and service companies, only 7.7% were free of regional pain of any description [10].

Neck pain also occurs in children, and it can be persistent. When 1756 schoolchildren of 9- to 12-year-old were followed over a 4-year period and questioned about the occurrence of neck pain, 24% reported no neck pain in that period, 71% had fluctuating neck pain, and 5% persistent weekly neck pain [11]. There was a trend to weekly pain in children with other musculoskeletal and/or other physical and psychologic stress symptoms at baseline [11]. Another survey of 643 adolescents (54.6% female) reported at the 14-year follow-up that females had a higher prevalence of lifetime, 1-month, and chronic neck/shoulder pain than males (50.9 vs 41.7%, 34.1 vs 23.5%, and 9.2 vs 6.2%, respectively), and that there was no relationship between neck pain and the level or intensity of physical activity or the type of sedentary activity [12].

COURSE

Neck pain is not a condition that inevitably recovers, but the overall rate of recovery is something that can be used to cautiously reassure patients, as about 40% of patients presenting with neck pain recover fully over time, and about 25% to 30% go on to have moderate to severe symptoms [13,14]. Over a 10-year follow-up period of people with neck pain, 79% of subjects reported a decrease in pain, including 43% who became pain free, but 32% reported moderate or severe residual pain [13]. Patients who had been injured and initially had severe pain were the most likely to have an unsatisfactory outcome; however, there were no other predictive clinical features [13]. Specifically, the presence or severity of pain was not related to the presence of degenerative changes, the sagittal diameter of the spinal canal, the degree of cervical lordosis, or to any changes in these measurements over the evaluation period [13]. In children, those who develop weekly neck pain tend to continue to experience it at least over a 4-year period [11], with a prevalence rate of 5% to 10% [11,12].

DEFINITIONS

Cervical Spine Pain

Neck pain (or *cervical spine pain*) is defined by the International Association for the Study of Pain as the following: *pain perceived as arising from anywhere within the region bounded superiorly by the superior nuchal line, inferiorly by an imaginary transverse line though the tip of the first thoracic spinous process, and laterally by the sagittal planes tangential to the lateral borders of the neck* [15]. It is further subclassified regionally into *suboccipital pain* (between the occiput and the spinous process of C2), *upper cervical pain*, and *lower cervical pain* [15].

Somatic Referred Pain

The fundamental mechanism for referred pain is convergence [16]. Somatic referred pain is pain evoked by the stimulation of the peripheral endings of nociceptive afferent fibers and is perceived in an ambiguous site because of the phenomenon of convergence when these afferents converge on second-order or third-order neurons in the central nervous system that happen also to receive afferents from the region to which the pain is referred [17]. Under those conditions, and in the absence of additional sensory input to clarify the situation, the brain is unable to identify the source of the pain accurately, and attributes it erroneously to the entire area subtended by the common neurons [18,19]. Ambiguity as to the source of information arises, either or both, because the painful structure is not densely innervated, and the central pathways along which the information is relayed are not highly organized somatotopically [20].

Neck pain derived from the cervical spine can theoretically arise from any innervated cervical structure, and it may be associated with referred pain. Referral pain maps have been created, demonstrating that the segmental referral patterns overlap substantially. For example, cervical spine disc stimulation by discography at any level produces local neck pain that is unilateral as often as bilateral, with referral patterns as follows: C2/3 suboccipital and facial; C3/4 suboccipital, trapezius, anterior neck, face, shoulder, interscapular, and upper limb; C4/5 shoulder, interscapular, trapezius, extremity, face, chest, and suboccipital; C5/6 trapezius, interscapular, suboccipital, anterior neck, chest, and face; C6/7 interscapular, trapezius, shoulder, extremity, and suboccipital; and C7/T1 interscapular [21] (Figure 28.2A–28.2F). As might be expected from convergence theory, interspinous ligament pain maps and

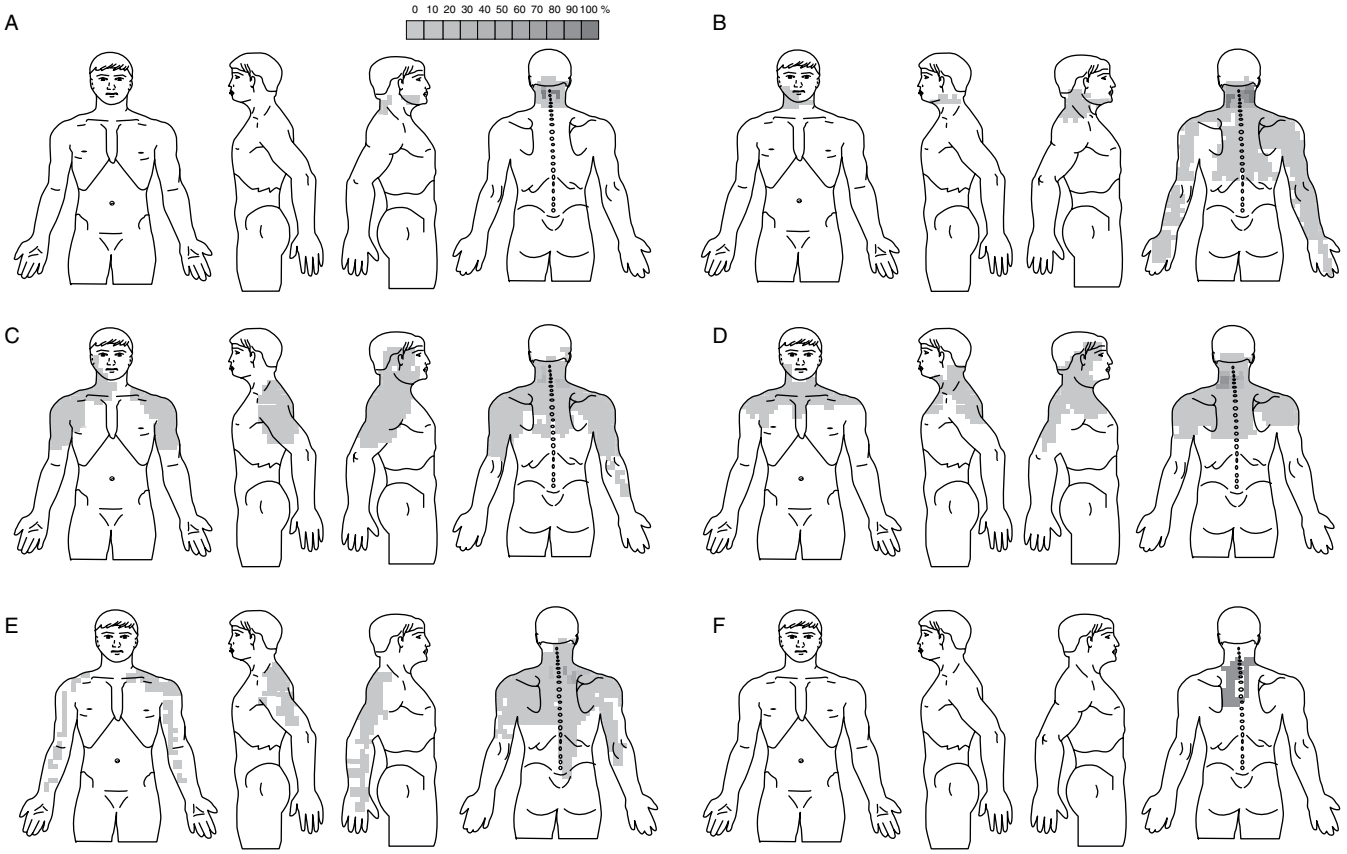

Figure 28. 2 **(A)** C2-C3 discogram pain referral map; **(B)** C3-C4 discogram pain referral map; **(C)** C4-C5 discogram pain referral map; **(D)** C5-C6 discogram pain referral map; **(E)** C6-C7 discogram pain referral map; **(F)** C7-T1 discogram pain referral map. Adapted from Ref. [21].

facet joint pain maps are similar to disc pain maps for each segmental level [6,22,23] (Figure 28.3).

Somatic referred pain is usually felt deeply, with a three-dimensional quality, and has is typically aching in quality [22]. Other adjectives variously used include gripping, boring, heavy, crampy, and lumpy [22]. Somatic referred pain from cervical structures has been recorded as extending as far as the hand in early studies [22,24], although subsequent studies have shown that, for example, facet joint pain may not extend beyond the proximal arm [25–27].

Cervicogenic headache is "pain that is perceived in the head but whose source is actually in the cervical spine or which is innervated by cervical nerve" [28]. It is therefore a type of somatic referred pain, which through the mechanism of convergence of sensory axons in the C1, C2, and C3 spinal nerves onto dorsal horn neurons also receiving afferents from the cervical trigeminal nucleus and the nucleus caudalis [29] causes pain to be potentially perceived in the distribution of the trigeminal nerve [28], particularly the ophthalmic division. Structures that might be sources of cervicogenic headache thus include the upper cervical synovial joints, the upper cervical muscles, the C2-3 disc, the vertebral and internal carotid arteries, and the dura mater of the upper spinal cord and posterior cranial fossa [30]. Additionally, as a result of headache eradication after cervical disc prolapse surgery, it has been proposed that the lower cervical roots also converge on these structures [29]. Diagnostic criteria have been specified by the International Headache Society [31] (Table 28.1).

Cervical Radicular Pain

Cervical radicular pain is a particular type of neuropathic pain caused by direct injury to a sensory nerve root or dorsal root ganglion of a cervical spinal nerve. It is characterized primarily by its location: it is felt predominantly in the upper limb and often also in the shoulder girdle. It is not infrequently accompanied by objective signs of deficit or loss of neurologic function in a segmental distribution as a result of conduction block and it can coexist with spinal or somatic referred pain [32,33]. The quality of pain tends to be deep severe aching pain, and as such it is different from the typical lancinating pain that can occur with lumbar radicular pain [33]. As cervical radicular pain is perceived in an area distant from the site of injury, it is a form of referred pain. However, it does not involve the stimulation of nerve endings, and does not involve convergence [19]. As cervical radicular pain is uncommon, with an annual prevalence rate of 0.083% in one large population study [34], deep aching arm pain is much more likely to be somatic referred pain than cervical radicular pain.

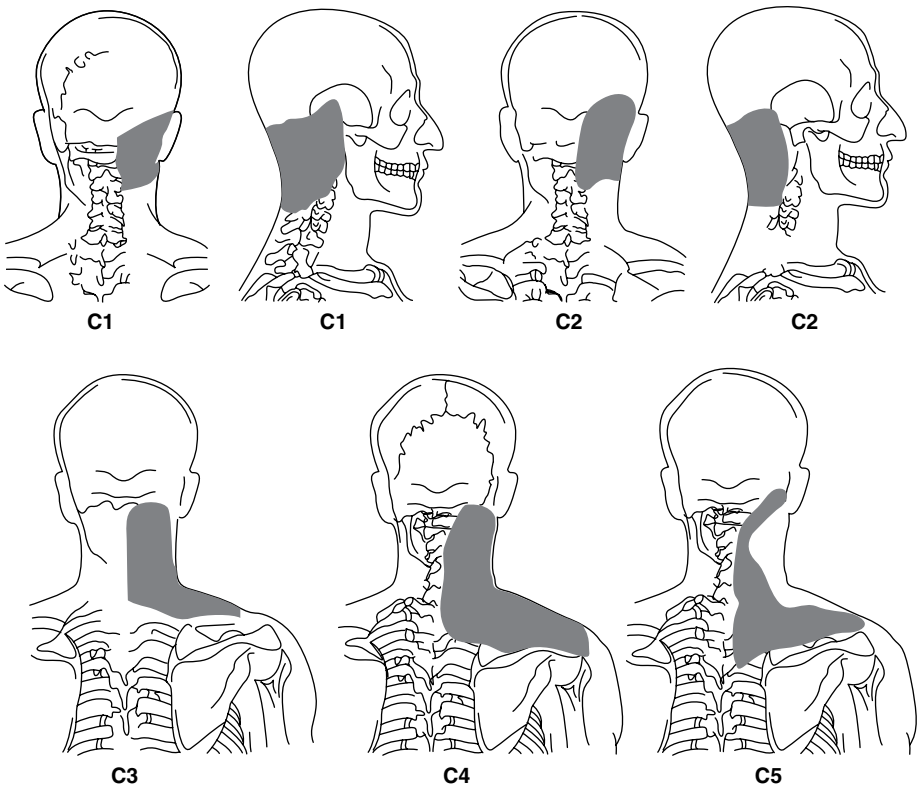

Figure 28.3 Referred pain patterns from injections of the interspinous ligaments with 6% saline using a 24-gauge needle. Adapted from Ref. [22].

Table 28.1 Diagnostic Criteria for Headache as Specified by the International Headache Society

A.	Pain referred from a source in the neck and perceived in one or more regions of the head and/or face, fulfilling criteria B and C
B.	Clinical, laboratory, and/or imaging evidence of a disorder or lesion within the cervical spine or soft tissues of the neck known to be, or generally accepted as, a valid cause of headache
C.	Evidence that the pain can be attributed to the neck disorder or lesion based on at least one of the following:
	a) Demonstration of clinical signs that implicate a source of pain in the neck
	b) Abolition of headache following diagnostic blockade of a cervical spine structure or its nerve supply using placebo and other adequate controls

With permission from Ref. [31].

Cervical radicular pain does not always correspond to dermatomal maps of sensory deficit due to cervical radiculopathy. Traditional dermatomal maps are inaccurate in any case for a variety of reasons [35], and they certainly do not coincide with dynatomal maps produced by individual nerve root stimulation [35]. Cervical radicular pain from the C6, C7, and C8 nerve roots is felt in the arm with pain extending into the forearm and hand [32] (Figure 28.4).

Neuropathic Pain

Cervical radicular pain is a subset of neuropathic pain, as it is pain caused by injury to a nerve, and typically affects the upper limb. Other neuropathic pain can be challenging to diagnose, as diagnostic criteria are still developing, and as it can present simultaneously with somatic referred or other pain [36]. The diagnosis of neuropathic pain is based primarily on history and physical examination [37]. Although neuropathic pain can have deep aching and shooting characteristics, more typical of lumbar radicular pain [33], it also attracts other descriptors such as burning, crushing, punishing, and cruel [37], and it can be associated with abnormal sensations such as formication. Because it is often unrecognized by clinicians [38], a range of neuropathic questionnaires, such as Standardized Evaluation of Pain [39], PainDETECT [40], DN4 [41,42], LANSS [43], and NPS [44], have been developed and reviewed [45]. The prevalence of neuropathic pain in the general population is about 7%, of which 70% have pain in the moderate to severe range [46].

When neuropathic pain is accompanied by clinical features of reduced or exaggerated neural function it is more easily recognized. Reduced function, which indicates nerve damage, is detected by careful clinical examination of the nervous system and can be supported where relevant with tests such as electromyography and MRI. Exaggerated function, such as hyperalgesia (increased sensitivity to noxious stimuli, defined as a shift to the left on the stimulus response curve), hyperpathia (increase response to minimal noxious stimulation), hyperesthesia (increased sensitivity to touch), and allodynia (touch or brush is perceived as painful), suggests loss of inhibition and implies nerve damage [47]. It is relevant to note that brush allodynia (brushing parallel to the skin is perceived as painful or unpleasant) is specific for loss of inhibition,

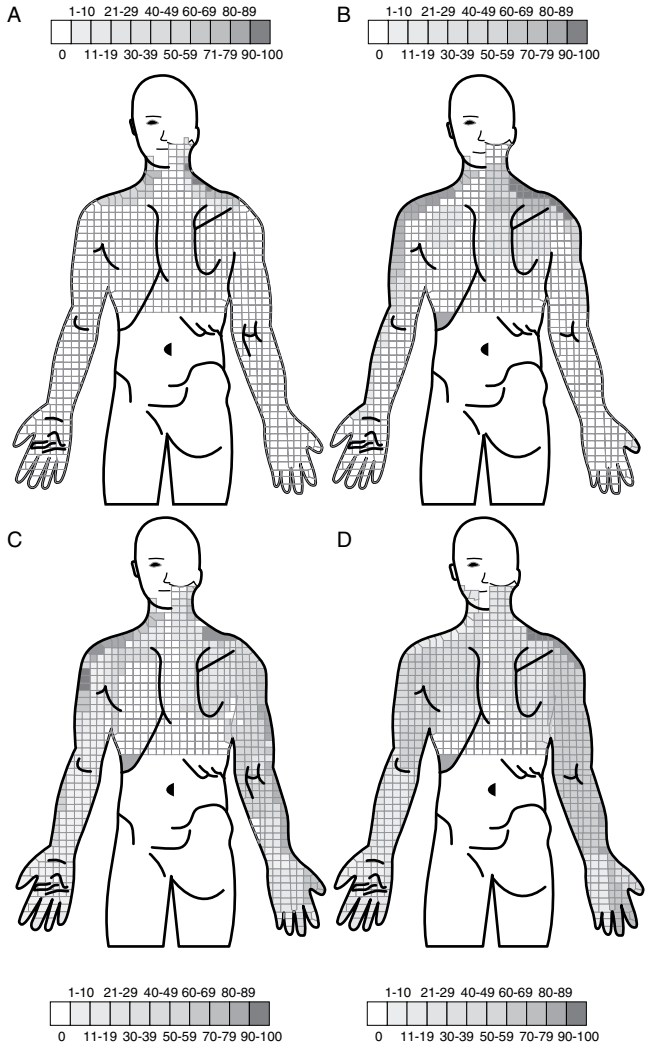

Figure 28.4 Percent occurrence of symptom provocation per bit for C4 to C7 nerve roots. **(A)** C4; **(B)** C5; **(C)** C6; **(D)** C7. Adapted from Ref. [35].

but other features, such as hyperalgesia, are found in other conditions such as fibromyalgia, which is a painful condition that should not be considered a true subset of neuropathic pain. Thus, there is a grey area in the detection of exaggerated neural function.

EPIDEMIOLOGY OF CHRONIC NECK PAIN

The major risk factors for neck pain in the general population included genetics, nicotine dependence, and stress [48] (Table 28.2).

Psychologic

Psychologic conditions have been considered to be significant in the epidemiology of neck pain, but any such assertion is not backed by the literature. General tension, including anxiety and a depressed mood [58], and a sense of inadequacy are psychosocial factors with an association with neck pain [56,59]. These associations are seen in young school children [11] and in the workplace [50,60]. However, psychologic state per se only accounts for 2% of the variance in cervical symptoms [61]. After motor vehicle accidents (MVAs), depressive and posttraumatic stress symptoms at 5 months are associated with an increased risk of moderate to severe neck discomfort at 18 months [54].

TABLE 28.2 Variables Shown to be Risk Factors for Neck Pain

Genetics [49]
Workplace stress [50]
Nicotine dependence [51]
Some ergonomic variables
• Holding the neck in a forward bent posture for a prolonged time
• Working in an uncomfortable environment [52]
• Repetitive movements
• Mental tiredness by the end of the day
• Shortage of personal
Obesity [53]
Trauma such as MVA [54]
Female sex [9,55,56]
Educational level less than 12 years [50]
More rigorous work [57]

Work Environment Factors

It should be noted that very few workers are totally free of pain in musculoskeletal regions in any case, calling into the question the incidence of neck pain at work [10]. Idiopathic neck pain at work is most associated with social factors, seemingly best summarized as work in an uncooperative or oppressive environment [62] or work with low job satisfaction [10]. However, it is also associated with high physical workload [63] and prolonged working hours [64]. Workers who have control over their job have less neck pain than other workers [65]. In workers with neck pain, prior neck pain and prior sick leave are associated with poorer prognosis, and general exercise with a better prognosis [65].

Workers who already have neck pain tend to continue to experience it, with at 5 to 6 years only a 36% chance of being symptom free [66]. Exposure to at least two of the tasks manual handling, working with hands above shoulder level, and working with vibrating tools increases the odds of neck pain, whereas sedentary workers generally have a greater chance of being symptoms free [66].

Factors shown not to be an etiologic risk factor for work-related neck pain include pathoanatomical entities such as facet joint spondylosis [56] and DD [56]. Other variables not associated with neck pain include marital status [61], having children [61], economic status [61], living conditions [61], exercise [61], workload at home [61], activities outside work [61], smoking (see later comments on dependence) [50], whole-body vibration [67], and ergonomic variables in general [68] (Table 28.3).

Some individual ergonomic variables, however, do have a correlation with neck pain; these include holding the neck in a forward bent posture for a prolonged time (odds ratio [OR] = 2), sitting for a prolonged time (OR = 2),

TABLE 28.3 Variables Shown not to be an Etiologic Risk Factor for Work-Related Neck Pain

Facet joint spondylosis [56]
Disc degeneration [56]
Marital status [61]
Having children [61]
Economic status [61]
Living conditions [61]
Exercise [61]
Workload at home [61]
Activities outside work [61]
Smoking (see later comments on dependence) [50]
Whole-body vibration [67]
Ergonomic variables in general [68]

making the same movements (OR = 1.6), mental tiredness at the end of the workday (OR = 2), and shortage of personnel (OR = 1.7), each with 95% confidence limits (CI) above 1.0 [9], and working in an uncomfortable posture [52]. Other factors associated [62] with neck pain include psychosocial work place factors [10], previous injury [50], MVAs [54], female gender [56], educational level less than 12 years [50], some occupations (clerical, industrial, and agricultural) [50], and occupational factors such as physical or mental stress at work [50] and working with machines [57]. In office workers, risk factors are female sex (not due to sex-specific genetic factors) [9,55] and high stress, and protective factors are increased neck mobility and frequent exercise [60].

Sickness absence from work due to neck pain is related to work-related neck flexion, neck rotation, low decision authority, and medium skill discretion, and tends to be related to high job demands, low skill discretion, and low job security [69]. It should be noted that this may be a reflection of general health trends which show that general health is poorest in low income unemployed in both men and women and the trend is maintained for those employees not in fixed-term employment [70].

Trauma

MVAs are associated with neck pain. In Auckland, where there is a no fault compensation system, 18.7% of people who were involved in a MVA reported neck pain or stiffness after 5 and 18 months, and of these, 70% had limitations of work and recreation [54].

Posture

Three postural abnormalities (elevation of one shoulder, elevation of one hip, and deviation of the spine from the midline of the body) have been shown to have no association with low back, mid back, or neck pain over a 25-year period [71].

Genetics

The overall genetic contribution to neck pain is considered to be 39% [49]. There is a significant genetic factor in the genesis of neck pain in younger population [72,73] but this effect may diminish, with one study suggesting that by 70 years the effect is minimal [72]. The genetic effect is significantly greater in women [49,72]. Furthermore, there is a primarily genetically determined moderate association between spinal pain including neck pain and symptoms of anxiety and depression [74].

Age

The prevalence of neck pain peaks in the middle age groups [48,75]; in the elderly, pain is less prevalent but more long-lasting [75]. However, in a survey of people aged 100 years or older, 23% of women and 19% of men had experienced neck pain during the past month [76].

Obesity

Chronic neck, back, limb, head, abdominal, and pelvic pain is strongly associated with obesity (OR = 2.0, 95% CI = 1.27–3.26) and severe obesity (OR = 4.1, 95% CI = 1.57–10.82) [53].

Smoking and Nicotine Dependence

Chronic neck and back pain are both more prevalent in current smokers and in those diagnosed with lifetime as well as current nicotine dependence [51]. However, although there is no significant incremental relation between current chronic neck pain and being a current smoker, there is a significant association with lifetime and current nicotine dependence [51].

CERVICAL SPINE DEGENERATION

Aging is not the same as degeneration, but both are substantially underpinned by genetic mechanisms. Put simply, degeneration is accelerated aging, but the actual difference is difficult to assess in clinical practice. Both are detected in late stages on imaging studies. Histologic aging and degeneration occur years before later detection by various imaging techniques. Cervical spine degeneration is characterized macroscopically by synovial joint cartilage loss and intradiscal tears; facet joint osteophytes appear about 20 years after cartilage loss and paradiscal osteophytes also about 20 years after disc tears; vertebral end-plate irregularity appears about 15 years after disc tears [77]. Note, however, that if the spine is subject to trauma, end-plate fracture may well precede and precipitate DD [78]. Although MRI seems to detect end-stage DD, it is less able to detect it in earlier stages and thus, in early to middle stage DD, a pristine MRI, with current technology [79], does not preclude an underlying degenerative process [80].

The important point to recognize about degeneration is that there is no known demonstrable relationship between radiologic degeneration and neck pain, thus rendering the diagnosis of neck pain as "degenerative" by any of its labels illegitimate. This does not mean, however, that degeneration is not painful. What it means is that if it is painful there is no simple method of establishing such a relationship.

DD and facet joint spondylitis can theoretically cause neck pain by the process of nociception: mechanical and/or chemical stimulation of nerve endings. Mechanical stimulation might arise in a disrupted disc or facet joint because of abnormal loading of normal nerve endings, or from abnormal loading of neovascularized and reinnervated tissue that can follow anulus fibrosus (AF) disruption or end-plate injury [81]. Chemical stimulation can arise from inflammatory processes which ensue after tissue disruption. Both processes can be influenced by sensitization of the somatosensory system either locally (peripheral sensitization) and centrally (central sensitization) [81].

Aging

Degeneration is not the same as aging, and neither degeneration nor aging are necessarily related to neck pain. To understand degeneration, it is instructive to consider the process of aging.

The human body undergoes progressive age-related change over a lifetime, change that is caused by genetic and environmental factors [82]. Aging is the net effect of alteration and changed balance in gene expression profiles and transcriptional changes, which are associated with numerous biologic processes, cellular responses to environmental and endogenous factors, and disease states [83,84], leading inexorably to a gradual decline of organ systems, reduced reserve capacity, and increased chance of death [85]. At a cellular level, aging is implicitly related to cell senescence. Although senescent cells are metabolically active, they do not divide [86]. Cell senescence is accompanied by a distinct set of cellular phenotypic changes caused by mechanisms such as DNA damage, chromatin instability, overexpression of oncoproteins, a variety of stress signals such as oxidative damage, and progressive telomere shortening [85]. Cell senescence is not a benign process; it is associated with disease-independent, chronic, low-grade systemic inflammation, which includes in its sequelae sarcopenia [87], and which underlies biologic mechanisms responsible for age-related inflammatory processes [88–91].

The human spinal skeleton reaches maturity when, at about 20 years, the cartilage growth plates fuse [92]. With aging there is a gradual decline in cellular efficiency, leading inexorably to generalized frailty, defined by increased stiffness and reduced activity [93]. Stiffness is caused largely by alteration in collagen biochemistry in relatively avascular structures such as cartilage, tendons, discs, and ligaments, rather than muscle [92], whereas weakness is muscle related (sarcopenia). Reduced activity is multifactorial, but, from a skeletal perspective, is strongly influenced by sarcopenia. Sarcopenia is attributable to an imbalance between protein synthesis and degradation, or between apoptosis and regeneration processes, or both [94]. As a corollary, exercise has been shown to be important in longevity and health [95,96], and in the skeleton, it certainly fights sarcopenia and probably reduces stiffness. Although aged tissue is generally stiffer and less efficient, it does not necessarily undergo substantial overall structural change. However, over the years, there is both an increased risk of tissue degeneration from a variety of mechanisms including acute and/or repetitive trauma and reduced healing capacity.

Aging-related changes that might be expected over a 10-year period as detected on MRI in middle aged people (from a sample of 233 subjects) include progression of "degenerative" changes in 81.1% (in reality, these are progression of age changes), decrease in signal intensity of disc (59.6%), progression of anterior compression of dura and spinal cord (61.4%), development of posterior disc protrusion (70.0%), and progression of foraminal stenosis (9.0%) [97].

Degeneration

How is degeneration different from aging? The subtle but important difference between aging and degeneration is that degeneration is abnormal, and is defined as aberrant, cell-mediated response to progressive structural change [98].

Taxonomy

The taxonomy of cervical spine degeneration remains nonconsensual. In this chapter, cervical spine degeneration is the generic term used for changes at whole segmental levels; spondylitis is the term used for diarthrodial joint degeneration; DD for intervertebral DD. Diarthrodial joint spondylitis, facet joint spondylitis, and cervical DD are separate entities.

Clinical Relevance

Both aging and degeneration have an impact on cervical spine mobility. There is a gradual decline in cervical spine mobility with age, arising, at least in patients with neck pain, principally in relation to age itself more than degeneration [99,100], although this is somewhat dependent on how cervical degeneration is defined [77]. In the general population, women have a slightly greater range of cervical spine movement than men [99,101], but this relates mainly to C2/3 segmental movement [99] rather than to the degenerative process. At individual segmental levels, degeneration is associated with reduced mobility [99]. However, gross mobility is usually not effected as nondegenerative adjacent segments become somewhat more mobile [99].

DD and facet joint spondylitis are fundamentally cellular constructs, which are ultimately identified clinically by various radiologic techniques. Their radiologic occurrence is only weakly related to and therefore not caused by aging [102], peaking at about 85 years [103]. Radiologic cervical DD is present in 13% of men and 5% of women during the third decade, in 85% to 90% of the population by the sixth decade and nearly 100% by the age of 70 [104]. DD occurs most commonly at C5/6, C6/7, and C4/5, respectively [105,106]. In people aged 60 to 65 without neck pain, about 95% of men and 70% of women have at least one degenerative change on their cervical spine plain x-rays [105].

Cervical spine MRI degenerative changes are present in at least 25% of the population younger than 40 years [107,108]. When cervical MRIs were repeated on a group of

people 10 years after the first MRI, progression of degenerative findings occurred in 189 subjects (81.1%) and decreased signal intensity of disc in 59.6%, and these changes were only related to age [97]. In the asymptomatic population, disc protrusion is present in about 20% of people aged 45 to 54 years, and 57% of people older than 65 years; spinal cord impingement is present in 16% of people younger than 64 years and 26% older than 65 years; and cord compression in 7% due to disc protrusion [108].

Concurrent lumbar and cervical DD and facet joint spondylitis are present in up to 80% of the older population, and, furthermore, lumbar degeneration and advancing age are associated with cervical degeneration independent of race and sex, and lumbar degeneration precedes cervical degeneration [103,109].

Thus, degeneration is a largely genetically dependent process that is detected in later stages on imaging studies. Although it might have a role to play in the development of cervical radicular pain and other compressive neurologic states, it cannot be identified in the individual patient as a cause of neck pain.

Disc Degeneration

Anatomy of the Disc

Lumbar discs consist of a central nucleus pulposus (NP) surrounded by the AF, which consists of concentric laminae of collagen fibers thickest anteriorly and laterally [110]. In contrast, cervical discs consist of a crescent-shaped anterior interosseous ligament with thick anterior collagen fibers that taper laterally toward the uncinate processes; they are essentially deficient posterolaterally where there is only a thin layer of paramedian, vertically orientated fibers [79] (Figure 28.5). The anterior longitudinal ligament covers the front of the cervical disc, and the posterior longitudinal ligament reinforces the deficient posterior AF with longitudinal and alar fibers [79].

Cervical discs have a different chemical morphology to thoracic and lumbar discs. In particular, they contain a higher collagen content in the NP and higher glycosaminoglycans content in the AF, both of which allow for the greater demands of bending and twisting movements in the cervical spine compared with other spinal regions [111].

Discs in general are mainly composed of extracellular matrix molecules and have extremely low (about 1% of volume) cell content, with the content of cells decreasing from the periphery inwards, reflecting the relative avascularity of the disc [112]. The cells are of vital importance for disc homeostasis as they regulate the synthesis and metabolism of the extracellular matrix.

As discs are innervated, they therefore have the capacity to be sources of pain [113]. The normal disc is avascular and aneural except for the outer third of the AF [114,115]. Discogenic pain is a possible consequence of DD, which follows breakdown of the extracellular matrix, and secondary ingrowth of sensory nerves [116]. Although underpinned by genetically based cell senescence, DD is also influenced by other factors particularly compression loading and torsion [117].

Cervical discs are innervated laterally by branches of the vertebral nerve and more generally by branches of the cervical sinuvertebral nerves; these nerves penetrate as deeply as the outer third of the AF [114,118]. The end-plate adjacent to the NP is also innervated [119]. In so-called discogram-positive degenerate discs, there is a more extensive and deeper innervation of the disc, with free nerve endings expressing substance P reaching into the outer part of the nucleus [113,116,120], as well as

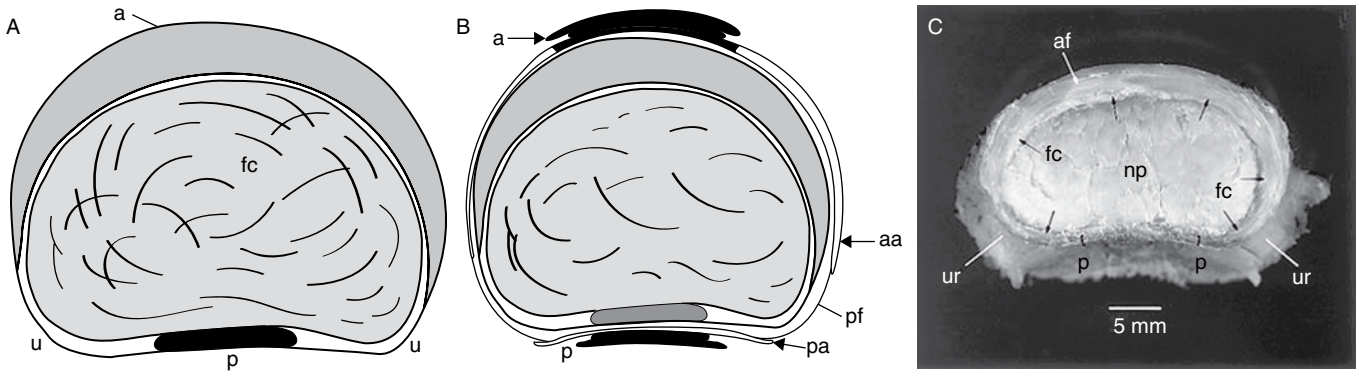

Figure 28.5 The cervical disc: "Top views of a cervical disc. **(A)** Sketch showing how the anterior AF (a) is crescentic, thick anteriorly, but tapering toward the uncinate region (u). It surrounds a central fibrocartilaginous core (fc). The posterior AF (p) is limited to paramedian longitudinal fibers. **(B)** Sketch showing how the AF is surrounded anteriorly by the three longitudinal layers of the anterior longitudinal ligament (a), laterally by the alar fibers (aa) of the anterior longitudinal ligament, posteriorly by the two longitudinal layers of the posterior longitudinal ligament (p) and its alar fibers (pa). The posterior lateral corner of the fibrocartilaginous core is covered only by periosteofascial tissue (pf). The thickness of the layers is not drawn to scale. **(C)** Photograph showing a top view of a 39-year-old disc from which all elements of the anterior and posterior longitudinal ligaments have been removed. The AF (af) is relatively thick and fibrous anteriorly, but tapers posteriorly toward the uncinate region (ur). It covers the uncinate region on the right but is deficient on the left. Deep to the fibrous AF is a fibrocartilaginous layer (fc, and arrows) that surrounds the large NP (np). Posteriorly, there is only a thin posterior AF (p) whose thickness is indicated by the brackets. Adapted from Ref. [79].

neovascularization and neuronogenesis of the adjacent end-plate and vertebral body [121]. The ingrowth of nerves in the discogram-positive disc is within a region of vascularized and innervated granulation tissue that accompanies fissures that extend from the AF to the outer part of the NP [120]. These nerves contain increased substance P-, neurofilament-, and vasointestinal peptide-immunoreactive nerves fibers, and are therefore likely to be associated with discogenic pain [120].

The Process of DD

DD is not caused by aging [122]. It may be useful to distinguish between aging and degeneration [123], but in the real world is this possible? Aging is a general process whereas degeneration tends to be more tissue or structure dependent. Both processes are determined by cell function, and, as such, are not observable or measurable by any simple technique particularly in the early stages.

Aging is associated with gradual changes in disc morphology, and is seen on MRI as disc degradation (reduction in T2-weighted signal) without other substantial changes such as disc space narrowing. DD, which, like progressive disc aging, occurs for many years at a radiologic subliminal level, is associated with substantial accelerated morphologic change. It is relevant to detect DD if it is a readily diagnosable entity, and if treatment directed at the degenerative process makes a difference to clinical outcomes.

DD is represented at a molecular level by a relative rise in the quantity of senescent cell population on the background of an overall fall in the total number of cells within the disc [86]. The percentage of senescent cells within a disc is also inversely related to the ability of other cells to proliferate, and the relative quantity of senescent and proliferating cells is independent of age [86]. DD ends with structural failure, represented macroscopically by thickened vertebral end-plates, increased cracks and fissures in the matrix, and delamination and tears in the AF [117]. The radiologic end-point of DD is intervertebral disc space narrowing and osteophyte formation [117].

Cellular function within the disc is mediated by at least five major factors: genetics, nutrition (diffusion of nutrients and oxygen across the disc matrix), cell function regulation (by interleukin-1 [IL-1], tumor necrosis factor-α [TNF-α], and TNF-β), age and senescence, and mechanical loading [124] (Figure 28.6).

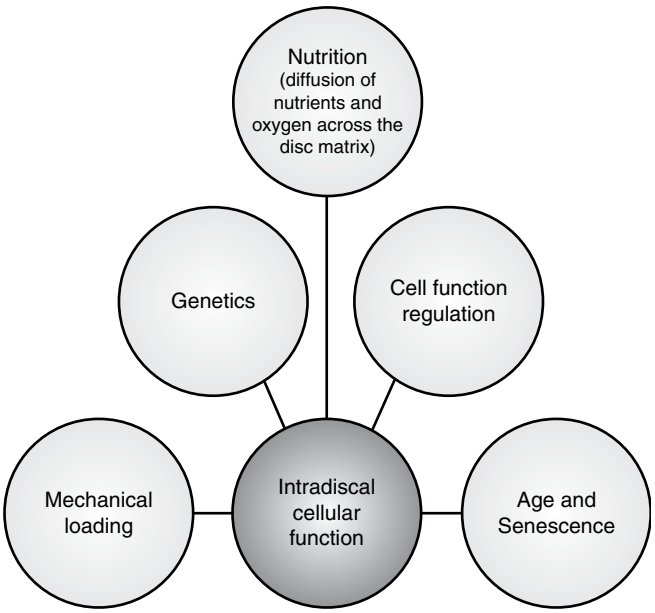

Figure 28.6 Cellular function within the disc is mediated by at least five major factors. Adapted from Ref. [124].

Genetic Factors

The genetic contribution to severe cervical DD may be as high as 80% [125], with general heritability for DD ranging from 34% to 61% in different regions of the spine [126]. The mode of inheritance of DD is multifactorial, and multiple genes, including genes coding for collagen I [127], collagen IX (COL9A2 and COL9A3), collagen XI (COL11A2) [128], IL-1, IL-6 [129], aggrecan [130], vitamin D receptor, and matrix metalloproteinase-3 (MMP-3), are thought to be associated [126], with the role of cartilage intermediate layer protein being disputed [131,132]. Furthermore, the study of genetics of DD is only in its formative years [133,134], and so far the only two factors consistently associated with DD are vitamin D receptor and collagen IX [124]. This emphasizes that DD is not primarily due to aging or to mechanically induced "wear and tear" processes [126,135].

Nutrition

Oxygen and nutrients, glucose being the principal one [136], are supplied, whereas lactic acid and other metabolic wastes are removed by diffusion (not by convective transport) either across the matrix from the outer AF vasculature or more prominently from the bone marrow via the end-plates [137,138]. The nutrient supply can fail because of changes in blood supply, for example, by general arterial sclerosis, by sclerosis of subchondral bone or end-plate calcification, and subsequent to increased cellular demand [137]. Smoking appears to be a likely factor in reduced diffusion [139], but exercise and motion are likely to promote increased diffusion [140]. Nutrient supply significantly contributes to the number of viable cells [136], and any nutritional deficit reduces call numbers, causing diminished matrix production and therefore DD [136,137]. DD itself is associated with compromise of at least lumbar posterior longitudinal ligament arterial supply, and neovascularization in the AF that pre-dates frank degeneration [141].

Cell Function Regulation

Inflammation, which itself is a bi-product of cell senescence, has a significant role in the development of DD and, as it plays a role in neuronogenesis, probably also with pain. Two cytokines, IL-1 and TNF-α, both intimately involved in inflammation, are important in the process of DD.

IL-1 appears to be a key regulator of disc cellular function. Imbalance in production of IL-1 can induce all the

changes seen in the matrix in DD [142,143]. These changes areas follows: increased gene expression for the zinc-based matrix degrading proteolytic enzymes MMP-3, MMP-13, and disintegrin and MMP with thrombospondin motifs-4 (ADAMTS-4); a decrease in the gene expression for matrix genes (aggrecan, collagen II, collagen I, and SOX6) resulting in abnormal synthesis of aggrecan and collagen II and their replacement by collagen I; apoptosis of cells; angiogenesis; and neuronogenesis [142]. As a corollary, IL-1 receptor antagonist (IL-1Ra), the inhibitor of IL-1, has been shown to reverse cellular changes of DD [143].

TNF-α is found in increased quantities in DD and may contribute to matrix degradation by increasing MMP-2 (also called gelatinase) activity [144]. Gelatinase cleaves the fragments of collagen produced by collagenase (MMP-1), which itself has a more direct action as it cleaves type II collagen [145]. This indirect action means that although TNF-α may have a subsidiary role in DD, it might play a greater role in discogenic pain through its role in neuronogenesis [146].

Leptin, which is an adipose-derived hormone that plays a significant role in energy intake and energy expenditure, has been shown to play a role in the pathogenesis of osteoarthritis by contributing to osteophyte formation [147]. In the disc it promotes the formation of cell clusters and proliferation of fibrocartilaginous tissue, and thus has a role in DD [147].

Tissue growth factor-β and the bone morphogenetic proteins appear to be anabolic in the disc, and thus there are studies underway to see if bi-products of these might alter the course of DD [124]. Indeed, these regulators of cell function are at the forefront of research into novel drugs that might prevent or delay DD and, if DD is shown to be a significant cause of neck pain, decrease discogenic pain.

Mechanical Load

In a young normal disc, any forces transmitted are dispersed evenly. However, as the matrix changes, the disc dehydrates and loses functional integrity [148]. The tissue changes of DD include increased matrix breakdown, a shift in matrix synthesis from type II to type I collagen (making the disc more fibrous [149]), decreased synthesis of aggrecan (the prime proteoglycan of the disc [145]), cell loss due to apoptosis, and cellular clustering [124]. As a result, the disc becomes more homogeneous; the NP dehydrates [150]; and fissures develop [149]. The AF collagen thickens and becomes fibrillated, and cracks can appear [151–154].

Dehydration contributes to decreased tension in the AF collagen fibers and consequent enthesis shock loading during normal movement, causing microtrauma [124], which in turn can damage adjacent bone and end-plate, leading to nutritional compromise and the promotion of neovascularization and neuronogenesis [121,155]. As the disc further loses integrity and height, the facet joints and other structures undergo increased loading and altered motion, and are rendered liable to traumatic effects [124,156].

Subliminal microtrauma is considered to induce DD by such mechanisms. More overt trauma is likely to play a greater role in DD, and it is probable that injury to the end-plate is pivotal [117]. Severe trauma can produce, within 24 hours of injury, death of the cells in the disc and consequent severe long-term implications [157].

Most of the research regarding vertebral body, end-plate, and disc response to mechanical loads pertains to the lumbar spine. In the cervical spine, the extent to which mechanical factors such as compressive loading, shear stress, and vibration play in the pathogenesis of DD is unclear.

An interesting study on cervical spine radiologic changes in case-matched Nepalese porters aged 40 to 50 years who carry loads on their heads, a process that would seem to cause microtrauma, found an overall prevalence of radiologic cervical degeneration of (58%), with a significantly lower prevalence in the porter group (the OR was found to be 0.23 [0.10, 0.53]) [158]. Such a finding suggests that repeated microtrauma might be a preventative factor in DD, possibly owing to changes at the end-plate leading to increased diffusion.

Aging and Senescence

Cellular senescence is a characteristic of most normal cells [159]. Senescent cells are highly represented in the aging human intervertebral disc. As senescent cells cannot divide the aging disc is less able to generate new cells lost to necrosis or apoptosis [160]. There are two types of cell senescence, replicative senescence (RA) and stress-induced premature senescence (SIPS). Senescence arises from progressive telomere shortening and dysfunction subsequent to incomplete DNA replication [161]. The telomeres are the repeated DNA motifs at the ends of chromosomes that protect the whole chromosome during cell division, acting in the same way as the aglet protects a shoe lace and stops it unraveling [162]. The telomeres progressively shorten with each cell division in the absence of telomerase [163], the enzyme which replenishes telomeres [164]. Telomere depletion is primarily genetic (RA), but SIPS can arise without abnormal cell division as a result of a variety of stresses and signaling imbalances [165], including exposure to non-challenge stress [166], cytokines, and oxidative stress [124]. In both RA and SIPS of aging and chronic disease, normally functioning cells are replaced by senescent cells [167].

Facet Joint Spondylitis

In the cervical spine, facet joint articular cartilage degeneration is universal from about the start of the sixth decade, with a weak trend to increasing degeneration with age [102]. The extent of degeneration is similar through all spinal levels, which is in contrast to the lumbar spine where the lower facet joints undergo more degeneration [102]. Additionally, cervical facet joint degeneration is throughout the joint, which is also in contrast to the lumbar spine, where certain parts of individual facet joints undergo greater degeneration [102].

It has been traditional, but incorrect, to use such terms as cervical degenerative disease, cervical DD, and facet joint spondylitis as diagnostic terms for a patient presenting with neck pain. These terms describe mid to late stage degenerative change seen on imaging techniques.

Although there may be a link between degeneration and neck pain, there is at present no reliable or valid method to detect this relationship [107,168]. Thus, cervical imaging is not indicated in the assessment of neck pain unless there is clinical suspicion of a red-flag disorder [169]. On the other hand, degeneration is a significant contributor to neck stiffness [77,99,170–172], and, more importantly, contributes to the clinical manifestations of cervical radicular pain, cervical radiculopathy, and myelopathy.

NECK PAIN

Any innervated structure in the neck is theoretically capable of producing neck pain with or without referred pain. Such pain may be uncommonly associated with a component of neuropathic pain, which, in the absence of other nervous system pathologies, is likely to arise from changes in the somatosensory system.

Incorrect Diagnostic Labels

Various diagnostic labels for neck pain such as soft tissue injury, whiplash, strain, sprain, tendinitis, fibromyalgia, psychogenic, spondylosis, and degeneration have arisen for presentations of neck pain. The most inappropriate are those in which radiologic changes are cited as the diagnosis. The challenge for the expert is to provide, where possible, a scientifically robust diagnostic label to a presentation of neck pain, and if this is not possible, to be able to justify any other diagnosis, particularly where the cause is idiopathic (Figure 28.7).

Diagnostic Logic

Without recourse to interventional techniques, there is no simply identifiable source of neck pain, and thus, it is typically not possible to either make a specific diagnosis or find an underlying disease process. Thus, although the pathophysiology of painful cervical disorders needs to be understood, it is essential that the clinician understands the limitations in making real sense of the science of neck pain. An understanding of the processes involved in making a provisional or differential diagnosis is important not only in developing a sense for the relevance of clinical examination and imaging, but also for explanation of the diagnosis to patients.

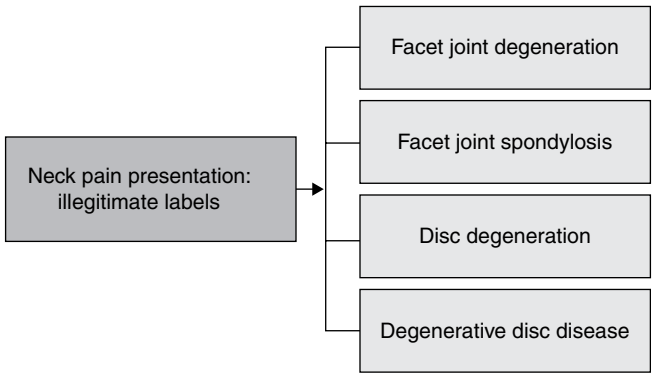

Figure 28.7 Examples of illegitimate labels for a presentation of neck pain.

Cervical spine disorders are generally separated into red-flag conditions, whiplash-associated disorders, and idiopathic neck pain [173], although the latter two are in reality difficult to separate apart from the event leading to pain generation. Trauma produces neck pain with a different but convergent spectrum of pathophysiologies, particularly anterior annular disc tears and impaction injuries of the facet joints and the lateral atlantoaxial joints [174], in comparison with non-trauma presentations. However, although such pathologies might be suspected, there is generally no method, including imaging, for detection of these laboratory detected cadaver findings [175], and the cause of pain in trauma is typically idiopathic.

Knowledge of the pathophysiology of cervical spine disorders simplifies the diagnostic algorithm for the patient presenting with pain from the cervical spine. The first issue for the clinician is to exclude serious disorders, either of pain referred to the cervical spine or of underlying severe disease. Examples of pain referred to the cervical and upper thoracic regions include acute coronary syndromes [176,177], carotid artery disease including aneurysm [178], and dissection of the vertebral artery [179]. The prevalence of local red-flag disorders, such as tumors and infections, presenting as a cervical disorder is less than 0.38% [173].

Cervical radicular pain is suspected if the pain is radicular in nature (concentrating in the upper limb) or accompanied by features of upper limb radiculopathy.

Most patients presenting with neck pain will remain within the diagnostic algorithm at this stage of clinical assessment. If diagnostic interventional techniques are not used, this is where the diagnostic process finishes in the majority of cases, with the exclusion of named specific conditions such as the extremely rare longus colli tendonitis [180,181], which is a misnomer for a condition that lacks a better name [173] (and has also been referred to as retropharangeal tendonitis [182]) and torticollis, which have their own pathophysiological processes.

As has been seen in the discussion on cervical degeneration, imaging demonstrates later stage morphologic change, but does not help with any specific diagnosis. Cervical DD and facet joint spondylitis are not legitimate diagnoses. The major anatomical suspects for the origin of neck pain are the muscles, synovial joints, and discs. The pathologic processes leading to these structures becoming painful are theorized but definitively unknown. The term *idiopathic neck pain* is therefore been used as a realistic diagnostic label for neck pain.

Idiopathic neck pain can theoretically arise from any innervated structure, including muscles, discs, and cervical spine joints. Frank muscle injuries are not seen in chronic neck pain. There is no evidence to support the theories of myofascial pain at least in the chronic pain community; tender muscles are common [183], reliability of clinical examination is poor [184–187], and histologic studies have not revealed robust pathologic changes in muscle. Thus, even if primary muscle pain exists, there is no reliable or valid method for its detection. Abnormal patterns

of muscle function exist in chronic neck pain presentations [188,189], but these are likely to be secondary effects rather than the primary source of neck pain.

What Is Idiopathic Neck Pain?

Idiopathic neck pain is any non-serious otherwise unnamed cervical spine disorder producing local neck pain with or without referred pain. It is not cervical radicular pain. It should not be used as a diagnostic label for a presentation of widespread pain disorders such as fibromyalgia or chronic widespread pain, although the widespread pain, which is thought to have origin primarily in the central nervous system, could coexist with idiopathic neck pain.

From an anatomical perspective, there are four key areas of possible pain origin: muscle, synovial joints, intervertebral discs, and alar and transverse ligaments. There is no validated literature to support muscle as a primary source of neck pain [174], or to detect it if it exists. Alar and transverse ligament injuries may occur in trauma, but not at low impact [190]. Indeed, their existence after trauma is disputed, with MRI changes being considered both normal age-related degeneration [191] and trauma related [192,193]. Thus, ligament injuries associated with major trauma are not necessarily identifiable in the absence of instability and neurologic change and in the main can be discounted as a significant or identifiable cause of neck pain. The cervical synovial joints and the intervertebral discs are considered to be the most likely sources of at least chronic neck origin pain. As previously discussed, cervical imaging does not help in discerning cervical synovial joint or disc pain.

So, in the first instance, before interventional techniques are available or performed, tissue-specific diagnosis is elusive in 100% of patients initially given the label idiopathic neck pain. A realistic opening gambit for a patient presenting with neck pain is therefore to state that the pain is idiopathic but a tissue-specific diagnosis is plausible with the aid of additional diagnostic tests.

The issue for the clinician at this stage is to determine whether or not it is worth knowing the precise anatomical source of pain. This is predicated upon the extent and stage of the presenting problem as well as the likelihood of benefit from any specific management that follows from such specific information.

A reductionist diagnostic approach requires access to medial branch blocks to diagnose facet joint pain, atlantooccipital and lateral atlantoaxial injection to diagnose pain from these synovial joints, and provocation discography to diagnose discogenic pain. It should be noted that the investigation of neck pain by discography alone or by facet joint blocks alone constitutes an inadequate approach to neck pain as it fails to identify the majority of patients whose symptoms stem from multiple elements in the three-joint complexes of the neck [5]. In one practice survey of 56 patients, both a symptomatic disc and a symptomatic facet joint were identified in the same segment in 41% of the patients: discogenic pain alone was present in only 20% of the cases and facet joint pain in 23%, and only 17% of the patients had neither a symptomatic disc nor a symptomatic facet joint at the segments studied [5]. In another pain practice, the prevalence of facet joint pain was 55%, of discogenic pain 16%, and lateral atlantoaxial joint pain 9% [194]. A diagnosis remained elusive in only 32% of those patients who completed investigations [194].

Thus, if these data are representative of all pain and/or spine practices, the real prevalence of idiopathic neck pain is around 15% to 30%, with named diagnoses occupying the other 70% to 85% as follows:

- Facet joint pain alone (20%–40%)
- Disc pain alone (10%–20%)
- Both facet joint and disc pain (30%–40%)
- Lateral atlantoaxial joint pain (<10%)
- Atlantooccipital joint pain (<10%)

Note that these figures are dependent on the criteria used for diagnosis. For example, in some centers, particularly in academic practice, the diagnostic criteria for facet joint pain are dual anesthetic blocks with total relief of pain [5]. In some centers, 50% and 80% pain relief has been used in a comparative diagnostic criteria study [195]. The looser the diagnostic criteria, the greater the prevalence of a named cause of neck pain. Also, cervical discography, as will be discussed in another chapter, has its own issues to do with validity and methodology [5,80,196–208].

Does the Pattern of Referred Pain Assist in the Diagnostic Process?

Neck pain derived from innervated cervical spine structures can present with or without referred pain. Referred pain to the head, known as cervicogenic headache, has been shown most typically to arise from the C2/3 segment [6,23,209]; referred pain to the shoulder girdle and/or the arm is most likely to arise from the C5/6 segment.

Does Clinical Examination Assist in the Diagnostic Process?

It should be noted that there is no aspect of clinical examination that assists in converting a diagnosis of idiopathic neck pain to a named condition. The existing evidence base shows that there are few if any particular clinical signs, or combination of signs, that lead to a valid or reliable diagnosis in anatomical or pathologic terms. It is mooted that an absence of clinical signs, such as no tenderness to palpation and no pain on movement of the examined structure, might alert the examiner to reconsider referred pain to the spine from visceral disorders or a red-flag condition [210]. Indeed, this might be the most important reason to examine the spine [210].

Does the Type of Pain Assist in the Diagnosis?

Regional pain is considered to be of two types, nociceptive and neuropathic, and the two may coexist. Referred pain is pain perceived in a region innervated by nerves other than those innervating the source of the pain [18]. Referred pain from a cervical spine structure (somatic referred pain) is nociceptive pain that arises from any local innervated structure [211]. Regional and referred pain can

be considered to have neuropathic qualities if there are pain descriptors such as burning, shooting, stabbing, lancinating, and searing. Neuropathic pain is considered to arise from central nervous system processes that amplify pain, and also include complex regional pain syndrome that can occur, for example, after neck surgery or spinal cord injury [212].

The presence or absence of neuropathic pain does not add to the anatomical diagnosis. It might assist in the consideration of the pathophysiology of the nociception pathway and it might alter the methods applied in the management of a neck pain presentation.

Serious Disorders

It is reassuring that the prevalence of red-flag disorders, such as tumors and infections, presenting as a cervical disorder is less than 0.38% (upper end of 95% CI) [173], and that these disorders are detectable on imaging [213]. MRI and bone scanning have the highest sensitivity for red-flag detection, but as MRI has the highest specificity for tumors, and additionally offers the further advantage of assessing other soft tissue morphology, it is the investigation of choice [214]. As a corollary, in the absence of significant trauma, plain x-ray examination is of no value to screen for such red-flag disorders or, as has been discussed earlier, to establish a "diagnosis" of entities such as "cervical spondylosis" [169,215].

PATHOPHYSIOLOGY OF DISCOGENIC PAIN

Discs, or the adjacent end-plate, can become painful as a result of the ingrowth of pain sensitive nerve fibers into these damaged tissues. As discussed earlier in this Chapter in the section on DD, some discs undergo accelerated changes consistent with DD, resulting in alteration of the structure of the disc, where neurogenesis and neovascularization accompany fissures extending from the now fibrous NP to the outer part of the AF [120]. These nerves contain increased substance P-, neurofilament-, and vasointestinal peptide-immunoreactive nerves fibers, and are therefore likely to be associated with nociception [120].

Neurogenesis is stimulated by neoangiogenesis; endothelial cells from these vessels synthesize neurotrophins, which are primarily involved with neuronal development, function, and nociception [216]. Implicit in neurogenesis are nerve growth factor (NGF) and brain-derived nerve factor (BDNF), which are two of the four principal members of the neurotrophin group, the others being neurotrophin 3 and neurotrophin 4/5 [217]. BDNF is not only involved in the differentiation and survival of sensory neurons, but also in regulation of nociceptive function, central pain modulation, modulation of inflammatory pain hypersensitivity, and neovasularization [218].

NGF and BDNF function mainly by binding to their respective receptors, TrkA and TrkB [217], which exist in disc cells [218,219]. When NGF is released, it binds to TrkA and is then taken up by a neuron where it is trafficked back to the cell body, TrkA and Trk B do exist within the disc, and have a likely intrinsic autocrine role [217]. Other than their known role in association with the neurotrophins, their existence or role in discogenic pain states has not yet been defined [217].

Inflammation has a significant role in the development of neurogenesis within the disc. Two cytokines that are intimately involved in inflammation seem important in the process of painful DD. Probably the most important regulator of disc cell function is IL-1 [142]. IL-1 regulation breaks down in DD [217], and, as it modulates neurotrophin expression, it influences neuronogenesis. TNF-α, which modulates substance P expression, also induces sensory ingrowth into the disc [146].

Although there are mechanisms that allude to the possibility of cervical discogenic pain in the absence of trauma, there are no studies that validate this theory [174]. In trauma such as whiplash, tears of the anterior AF (in reality an avulsion of the AF from the vertebral body) are hypothesized [174]. In cadavers subject to whiplash, injuries to the cervical discs are found in 90% of subjects [220], including tears in the AF [221].

So, it seems logical to assume that discs can be a source of neck pain. However, it is not yet possible to determine if a cervical disc is a major source of pain using MRI or other imaging techniques, particularly as MRI findings do not correlate well even with advanced histologic DD [222,223]. Additionally, there is no model for cervical discogenic pain in the absence of trauma. In the lumbar spine discography is used as the tool to determine if a disc is painful, and it has its advocates [224,225] and critics [226]. Cervical discography is more in its infancy [227], and although it has its advocates [228], it cannot be performed in a similar manner to lumbar discography. This is because the posterior AF is deficient, and as transverse fissures are a normal component of the young adult cervical disc [79]. Injection into the cervical disc normally leaks out through these fissures posteriorly into the spinal canal.

Lumbar discography is predicated upon various biomedical features including the following: (1) lumbar degenerative changes do not correlate with either positive pain response from discography or pain prevalence in general; (2) grade 3 fissures correlate strongly with pain reproduction; (3) these grade 3 fissures are not age related; (4) discs established by provocation discography with internal disc disruption (IDD) have abnormal stress profilometry [229]; (5) altered NP pressure can arise experimentally from end-plate fatigue failure [230], which has been demonstrated to occur with loads that are consistent with moderately heavy work activities [231]; (6) the biologic features of IDD have been reproduced in live animal experiments [232]; and (7) the process of fibrosis is distinctly different in discs with IDD compared with control discs [233].

In contrast, there is little or no biomedical framework underpinning cervical discography; the most important differences areas follows: (1) cervical disc morphology is very different to that of the lumbar disc; (2) morphologic correlates with pain and age have not been established for cervical discography; and (3) that contrast and other injectable material leaks from normal young adult cervical discs thus rendering cervical discography an inappropriate test. There is no known mechanism by which a cervical disc

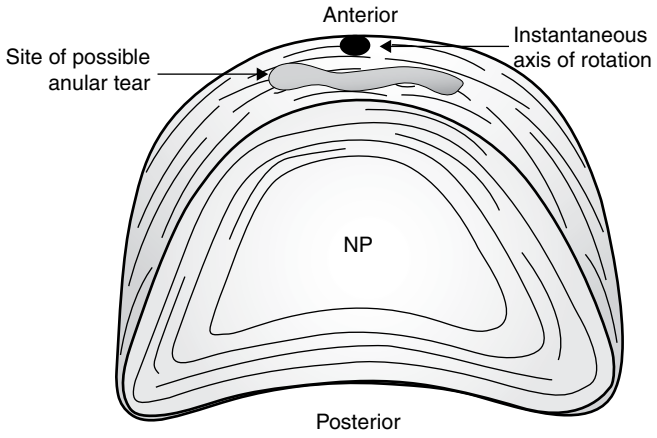

Figure 28.8 Site of posttraumatic anular tear.

could become painful in the absence of trauma. In trauma, on the other hand, rim lesions occur in the anterior AF [175] (Figure 28.8). If these were to be accessed by a needle, it would have to be introduced from an anterolateral approach avoiding the large vessels and other relevant structures in the cervical spine, probably under the guidance provided by computed tomography fluoroscopy.

PATHOPHYSIOLOGY OF CERVICAL SYNOVIAL JOINT PAIN

The cervical synovial joints, namely the facet joints, the atlantooccipital joints, and the atlantoaxial joints, are all potential sources of pain. However, osteoarthritis in these joints occurs in both the symptomatic and asymptomatic population [234,235]. Also, as with other joints, osteoarthritis, especially in the upper cervical spine, is not truly represented on radiography [236]. The method for detection of these joints as a source of pain is anesthetic injection of the joint or its nerve supply under imaging control. When used, at least after trauma, it is evident that there is a significant prevalence of facet joint pain within the neck pain cohort [25].

Again, apart from in trauma, there are no studies that demonstrate how facet joints become painful, but this is not dissimilar to the pain of all joints. In cadavers subject to whiplash, tears of facet joint capsules were commonly found [220,221].

PATHOPHYSIOLOGY OF CERVICAL RADICULAR PAIN

Cervical radicular pain is pain derived from direct compression, inflammation, or ischemic vascular compromise of the axons of the cervical spinal nerve or its roots [32]. The combination of transient compression and chemical irritation appears to be the most potent cause of persistent neuronal activity [237]. Direct nerve compression, which can be caused by space occupying lesions such as disc herniation, tumor, cyst, and osteophyte formation, does not cause pain unless it involves the dorsal root ganglion [238]. Inflammation can result from inflammatory cervical disc material lying adjacent to a nerve [239,240]; disc herniation is more common in discs with advanced cell senescence, particularly when clustering occurs [241].

How Should Neck Pain be Explained to Patients?

The reductionist approach to diagnosis makes explanation of neck pain at least logical. An example of such an explanation follows:

I have ascertained that your major concern is neck pain, which is somewhat worse on the right side and from where it spreads down to the right shoulder. Also, when worse you get a headache that spreads from the right upper neck through to your head.

After considering all these symptoms and making a physical examination I am able to reassure you that there is no serious cause for your symptoms. Furthermore, although you have been told in the past that your problem is degenerative disease, this is not the case. The x-rays and MRI do show that aging is certainly present, and that there might be some degeneration. However, most of these changes are likely to be inherited, whether they are age or degeneration related. That is, these changes are similar to your hair turning grey, baldness, skin wrinkles, etc. We know that people with those radiologic changes are no more likely to have or to develop neck pain than those without those changes. What these changes mean is that your neck may be stiffer, so perhaps you may need to exercise to keep the neck mobile. They may also be relevant if you present with another type of pain that relates to nerves being pinched (cervical radicular pain) but at present there is no indication of this, and furthermore, should such an unlikely event occur in the future, there is no known method for reducing the likelihood anyway. By the way, this pain would present with arm pain and perhaps some arm tingling or weakness; it is different pain to what you have now.

You have also described that you get some burning pain at times. This comes out of the pain system itself and is nothing to be concerned about.

Now, getting back to your pain, it comes from some structure in the neck. It can potentially come from anything that gets a nerve supply, that is, from muscles, joints, discs, etc. If you look at this model you can see how many of these structures there are. The facet joints, for example, are like the knee. They can be damaged, swell up, lock up, and be painful despite what an x-ray looks like. If we wanted to know if the facet joint was painful we would need to carefully under x-ray guidance insert some local anesthetic into the joint or onto its nerve supply and if the pain went away (or was blocked) for the time that the anesthetic was present, then this would be prima facie evidence that the joint was indeed the source of the pain.

Other structures can also cause the pain, such as the upper neck joints, C0/1 and C1/2, and these like the upper facet joints especially C2/3 can cause neck pain and headaches. The fact that your pain is in the neck and spreads to the head and shoulder but not the shoulder blade suggests that the pain is most likely to come from the C2/3 or C3/4 segmental levels. Another possible source of pain is a disc. Although pretty controversial, the only method that can be used to detect whether or not a disc is a source of pain is provocation discography,

where needles are inserted into the discs and when medicine is injected you experience your typical pain. Currently, MRI is not precise enough to tell us whether or not this pain you have comes from a disc.

I am telling you all this so you might understand the complexities of diagnosis. You understand that I think you have pain coming from one or more of these structures, but at present I do not think we need to make a more precise diagnosis. I reiterate that this condition is not serious. Additionally, although the neck pain is a significant problem for you at the present time, studies have shown that there is a reasonable chance that this pain will recover completely or to a significant extent over time.

As mentioned before, you have radiologic changes that are probably genetically acquired and are of no concern. You have also been told that the problem is muscular. It is not. The pain and tenderness in muscle is referred pain from some other source, such as a facet joint.

Also, if this pain is severe and persistent, and time plus any other treatment you might seek out does not help, then we can discuss whether or not some of these methods of making a more precise diagnosis are indicated. For now I am going to write to your family physician, and send you a copy of the letter, and say that the diagnosis is idiopathic neck pain, but that I think there is a good chance that if more thoroughly investigated, a diagnosis of right C2/3 and/or C3/4 facet joint origin pain is a distinct possibility.

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29 Algorithmic Approach to Cervical Axial Pain

David W. Chow, Gwendolen Jull, and Curtis W. Slipman

INTRODUCTION

For decades, chronic neck pain has been a clinical entity difficult to categorize and treat. This chapter will discuss a systematic categorization scheme and algorithmic approach to evaluate patients presenting with cervical axial pain who have failed to improve with a reasonable trial of conservative treatments such as physical therapy, anti-inflammatory drugs, analgesic medications, activity modifications, and relative rest. These patients have often been mistakenly diagnosed with chronic cervical muscular strains or as having myofascial pain syndrome. Muscular strains typically resolve within 2 to 3 months with conservative treatment. Fluoroscopically guided cervical spinal injections are commonly utilized to treat persistent cervical axial pain. Historical and more recent peer-reviewed literature investigating the utility of diagnostic and therapeutic cervical injections will be reviewed to provide the origins of an algorithmic approach to cervical axial pain.

HISTORICAL OVERVIEW

Fluoroscopically guided cervical spinal injections have been used in the clinical treatment of cervical axial neck pain for many decades. It is only over the past two decades that these procedures have been systematically utilized. In 1957, Smith and Nichols [1] first reported cervical discography as a diagnostic tool for the evaluation of symptomatic cervical intervertebral disc degeneration. Cloward [2] then reported the indications for diagnostic cervical discography. In 1964, Holt [3] reported that discography did not have diagnostic value. In contrast to Holt's study, Simmons and Segil [4] reported that cervical discography was a reliable diagnostic tool in the determination of a symptomatic cervical intervertebral disc. In 1986, Bogduk [5] identified the value of local anesthetic blockade of the medial branch nerves to the cervical facet joints (Figure 29.1), implicating the cervical facet joints as a pain generator for occipital headaches. A year later, Whitecloud [6] demonstrated the validity of cervical discography as a diagnostic tool. In 1988, Bogduk [7] proved that the cervical facet joints can cause neck pain and/or head pain. Cervical neck pain and/or head pain referral patterns were constructed from patients who obtained symptom relief after local anesthetic blockade of the cervical facet joints. In 1990, Dwyer [8] constructed pain referral maps of the cervical facet joints (Figure 29.2) in normal volunteers by distending the facet joint capsule with contrast injected under fluoroscopy. Aprill [9] evaluated the accuracy of the pain referral maps generated from Dwyer's study. The location of symptomatic cervical facet joints in patients with cervicogenic neck pain were predicted from the pain referral maps and then tested with local anesthetic blockade of the suspected cervical facet joints (Figure 29.3). Barnsley et al. [10] investigated the diagnostic value of comparative local anesthetic blockade of the cervical facet joints with local anesthetics of different durations of action. It was determined that false positives were present with single diagnostic cervical facet joint injections [11]. In 1994, Dreyfuss et al. [12] demonstrated that the atlanto-occipital and atlantoaxial

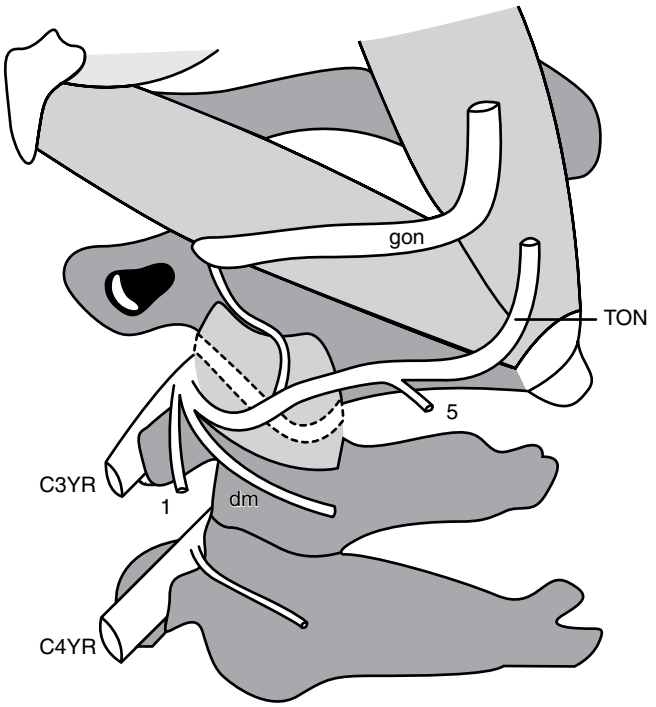

Figure 29.1 Successful local anesthetic blockade of the TON for occipital headaches implicates the cervical facet joints as a pain generator. Third Occipital Nerve (TON) lays across the C2-3 facet joint. From Ref. [5].

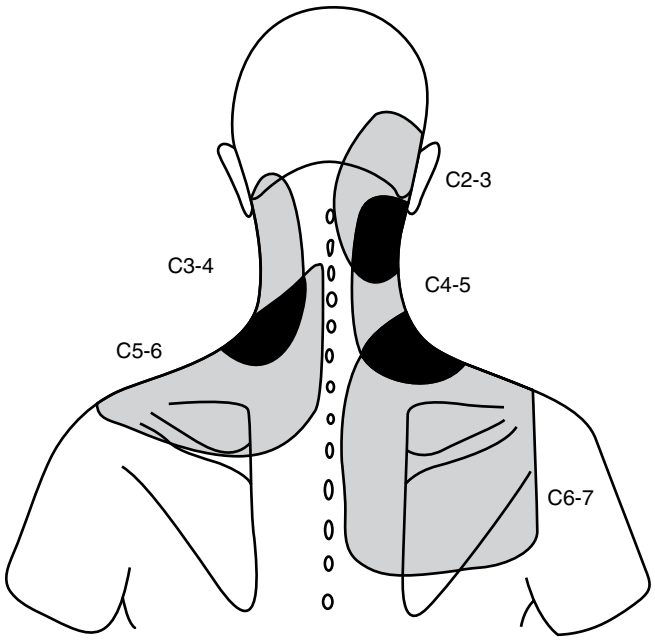

Figure 29.2 Cervical facet joint pain maps from asymptomatic volunteers. From Ref. [8].

joints may be nociceptive structures that can cause upper neck and head pain. In this study of asymptomatic volunteers, pain referral maps of the atlanto-occipital and atlantoaxial joints were constructed (Figures 29.4 and 29.5). Lord et al. [13] assessed the value of comparative local anesthetic cervical medial branch blocks in the diagnosis of cervical facet joint pain in relation to placebo-controlled cervical medial branch blocks. The prevalence of whiplash-induced chronic cervicogenic neck pain and headaches were investigated by fluoroscopically guided placebo-controlled local anesthetic injections by Barnsley [14]. In 1995, Saal [15] reported favorable outcomes for nonoperative treatment including the use of cervical epidural space steroid injections for painful cervical disc herniations and radiculopathy. In 1996, Schellhas et al. reported upon the distribution of pain referral patterns for the C3–4 to C6–7 disc levels. The study was also significant for demonstrating that magnetic resonance imaging (MRI) cannot reliably identify the source(s) of cervical discogenic pain and that significant cervical annular disc tears visualized during discography often escape MRI detection [16]. Also that same year, Lord et al. [17] reported on the effectiveness of percutaneous medial branch radiofrequency ablation for patients with chronic axial pain due to cervical facet joint syndrome (CFJS) in a double-blinded, placebo-controlled trial. In 1999, McDonald et al. [18] reported on the long-term efficacy of cervical radiofrequency neurotomy for chronic axial neck pain and demonstrated that repeat radiofrequency ablation will reinstate the same degree of pain relief if the pain returned after a successful initial procedure. In 2000, Grubb [19] reported that cervical discograms frequently identified abnormal concordantly painful fissured discs at multiple disc levels in more than 50% of the 173 patients examined, suggesting that treatment decisions based on fewer discs injected during discography should be reconsidered until more disc levels have been assessed. In 2005, Slipman et al. [20] reported upon the various cervical discogram pain referral maps (Figures 29.6–29.11) provoked during cervical discography in the first large multicenter prospective study investigating cervical discogenic pain map referral patterns. In 2008, Manchikanti [21] evaluated the effectiveness of facet joint nerve blocks using a double-blind, randomized, and controlled design confirming the existence of facet joint pain based on 80% relief with controlled local anesthetic blocks. Hence, over 50 years of work has helped lay the foundation for employing a tissue-specific diagnostic approach to optimally evaluate axial neck pain.

ALGORITHMIC APPROACH

When formulating a differential diagnosis for chronic cervical axial pain, one must have a working knowledge of the epidemiology, etiology, and pathophysiology of cervical spinal disorders that can cause axial neck pain. As in treating any medical condition, obtaining a comprehensive history and performing a thorough clinical examination is essential as it will provide information that will rank order a differential diagnosis and assist in formulating a treatment plan.

History of Presenting Complaint

Obtaining a comprehensive history is critical in providing information to organize a differential diagnosis and create a treatment plan. Determining if there is an inciting event such as a whiplash injury from a motor vehicle accident or previous trauma to the head or neck is important. Any injury such as whiplash that can cause compression, tension, shear, flexion, and extension forces at different levels is important and should be documented; no matter how remote the history of injury was, as long as the inciting event is identified as traumatic in nature. The history should be organized as traumatic versus atraumatic when attempting to formulate a differential diagnosis for cervical axial pain. A history of trauma provides the possibility that more than one structure sustained a traumatic injury. For example, cervical whiplash can injure a cervical facet joint, intervertebral disc, cervical nerve root, or a combination of these structures [14,22–30]. Cervical whiplash can injure a cervical intervertebral disc by causing a transverse tear near the anterior vertebral rim [25,26]. This transverse tear is also referred to as a "rim lesion" and is caused by distraction and shearing at the annular-endplate interface by the sudden cervical hyperextension whiplash injury [31–33]. These transverse disc tears predispose the disc to premature disc degeneration [26–28,34,35]. Cervical whiplash or trauma can also cause disc contusion or herniation, cervical nerve root shearing, facet hemarthroses, or fractures of the articular processes [14,23–29,32,36–42]. For nontraumatic cases, it is assumed that there is only one structure responsible for the painful symptoms.

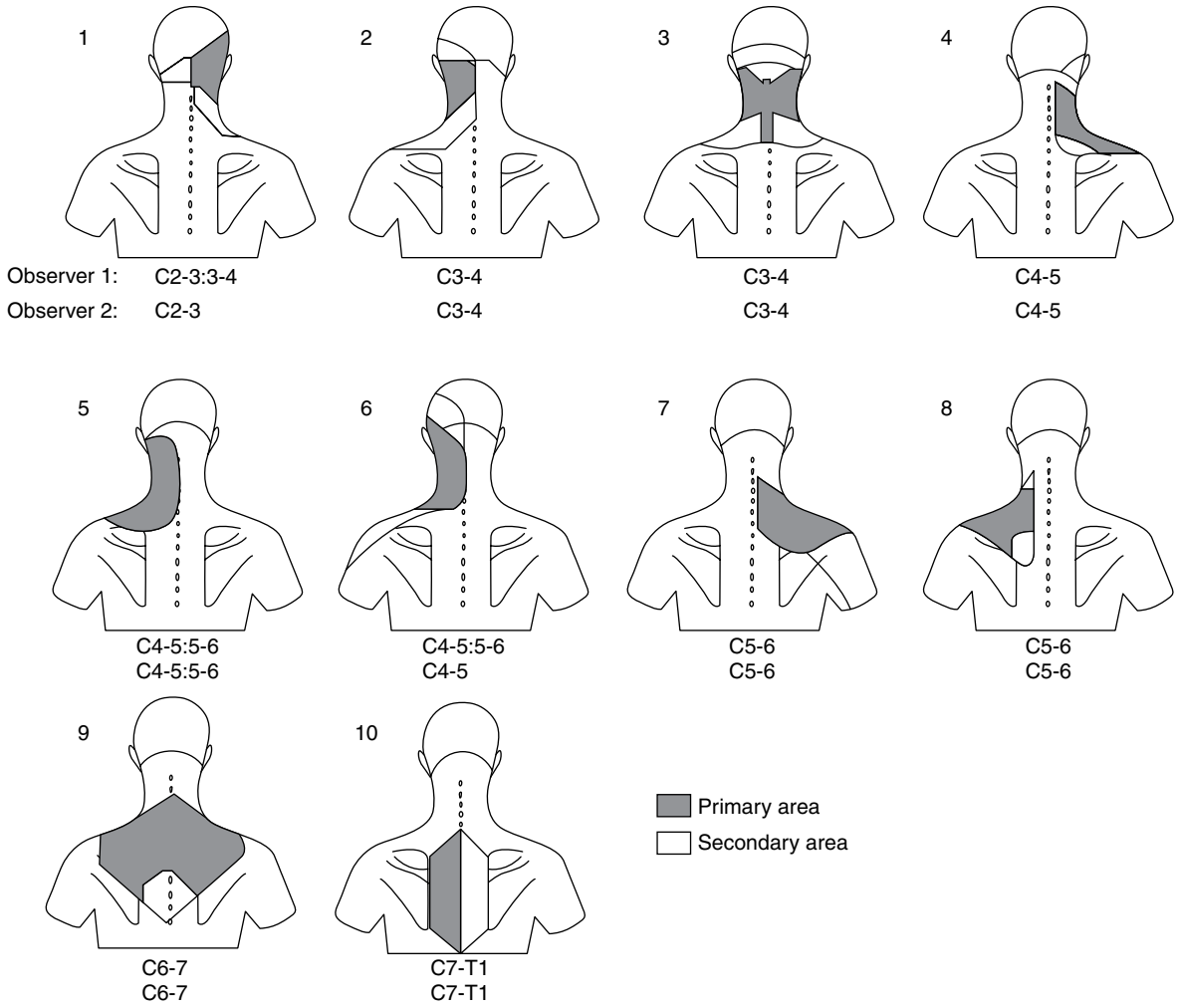

Figure 29.3 Cervical facet joint pain maps predicted and tested with diagnostic facet blocks in symptomatic patients. From Ref. [9].

Axial Versus Radicular Pain

Axial neck pain must be differentiated from cervical radicular pain as the etiologies and clinical pathways are vastly different. Axial pain includes central neck pain, occipital headache, periscapular pain, and/or interscapular pain. Chronic cervical axial pain is typically caused by CFJS and/or cervical internal disc disruption (CIDD) syndrome. CFJS typically does not refer pain below the elbow; consequently, forearm or hand symptoms would suggest nerve root or disc injury [8,9]. Cervical radicular pain is defined as upper limb symptoms greater than axial complaints. Upper back or scapular pain greater than neck pain in the patient without arm symptoms can also be radicular. Having a full understanding of the various "dynatomes" or cervical nerve root pain referral patterns is necessary to properly interpret the distribution of painful symptoms and to diagnose cervical radicular pain.

Segmentation of Pain into Quadrants

The next step in formulating a differential diagnosis is segmenting the distribution of pain into quadrants: head, neck, upper back or periscapular, and upper arm or forearm. The relative distribution of pain in these quadrants will assist in further organizing the differential diagnosis and in determining whether the pain is more likely axial or radicular. Cervical axial symptoms can be further segmented by organizing between upper versus lower neck pain and unilateral versus bilateral neck pain. Upper neck pain refers to the upper half of the neck, especially just below the occiput and often refers into the head (cervicogenic headache) via the trigeminocervical nucleus [43]. Lower neck pain refers to the lower half of the neck. For patients who have both upper and lower neck pain, it is important to determine whether their chief complaint is primarily upper neck pain or lower neck pain. Unfortunately, some patients may by unable to accurately verbalize where their primary neck pain is located. In this scenario, it is helpful to instruct patients to point with one finger indicating the location of their most intense pain.

Upper Versus Lower Cervical Pain

Upper cervical neck pain may be caused by an upper cervical facet joint (C2-3, C3-4, C1-2, C0-1) or by CIDD

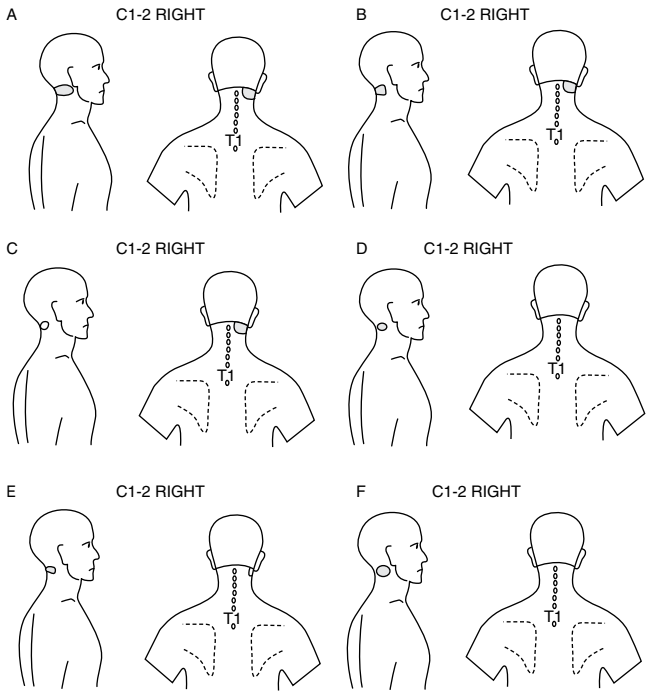

Figure 29.4 Right C1-2 facet joint pain referral maps. From ref [12]. Atlanto-occipital and lateral atlanto-axial joint pain patterns. Spine, 1994;19(10):1125–1131, with permission.

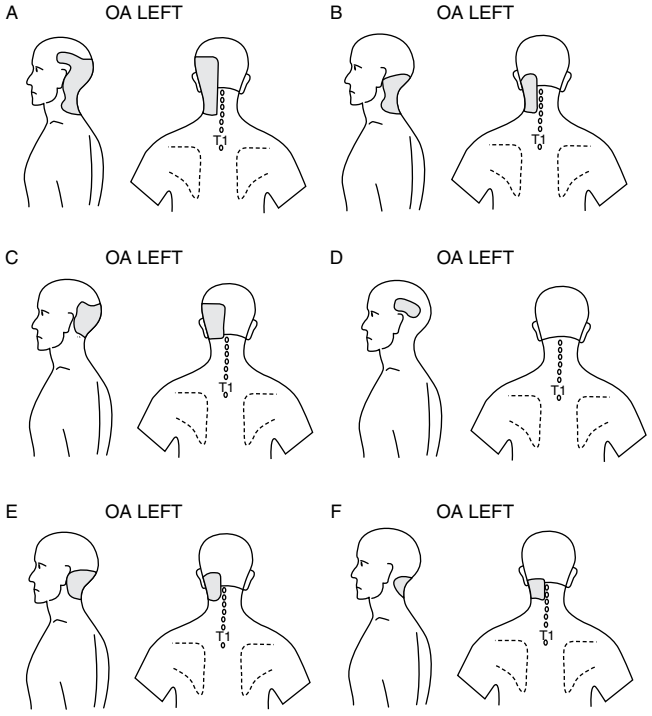

Figure 29.5 Left C1-2 facet joint pain referral maps. From Dreyfuss P, et al. Atlanto-occipital and lateral atlanto-axial joint pain patterns. *Spine*, 1994;19(10):1125–1131, with permission.

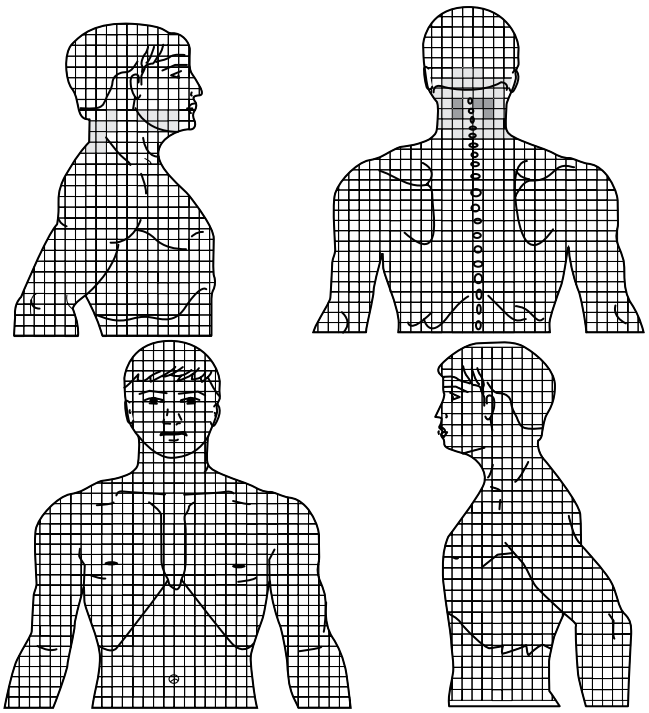

Figure 29.6 C2-3 Discogram pain referral map. From Ref. [20].

[29]. Traumatically induced upper cervical axial neck pain with occipital headaches is more suggestive of an upper cervical facet joint injury or CFJS combined with internal disc disruption (IDD) rather than CIDD alone [14,29]. Consequently, factors such as a history of trauma, unilateral symptoms, or the presence of headaches would more likely suggest upper CFJS than CIDD. Factors that would favor IDD as the cause of upper cervical axial neck pain include an atraumatic history and bilateral symptoms. Lower cervical neck pain may be caused by an intervertebral disc, lower cervical facet joint, or an injury to the fourth or fifth cervical nerve root [14,23–29,44]. In the setting of cervical whiplash or trauma, CFJS maybe more common than an injury to a cervical nerve root; however, this may be due to the dearth of epidemiologic data for traumatically induced cervical radicular pain [29,45].

Periscapular and Interscapular Pain

The differential diagnosis for painful symptoms that are mainly in the upper back, interscapular, or periscapular region include CFJS, CIDD, cervical radicular pain, and, less likely, upper thoracic IDD. If the interscapular or periscapular pain is reproduced by a nerve root provocative maneuver such as the Spurling's test, then cervical radicular pain would be ranked higher on the differential diagnosis than CFJS or CIDD. Correlating diagnostic studies such as a positive electromyography or imaging studies demonstrating lower cervical nerve root impingement would also suggest cervical radicular pain more so than cervical axial pain as the cause of the periscapular

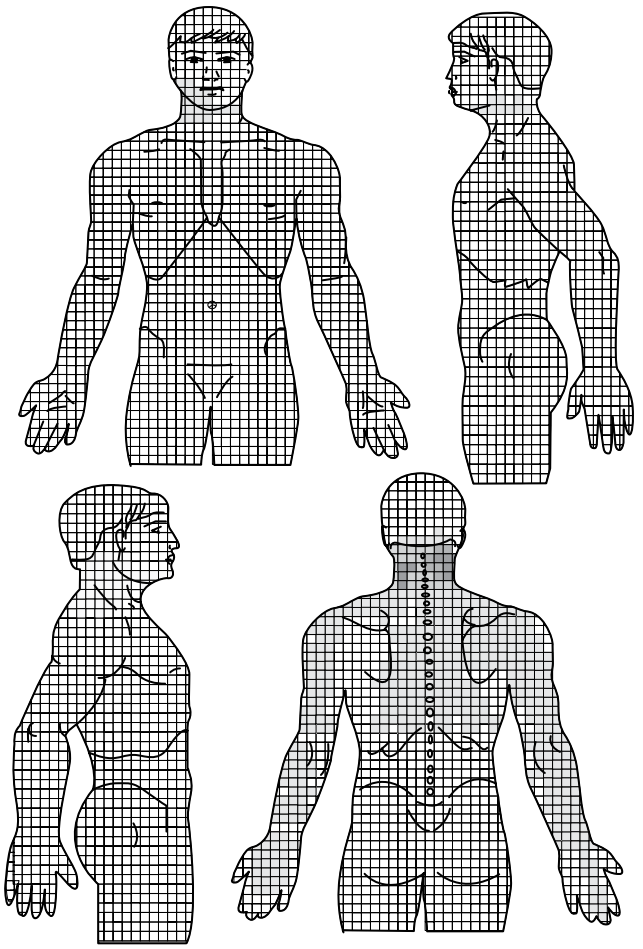

Figure 29.7 C3-4 Discogram pain referral map. From Ref. [20].

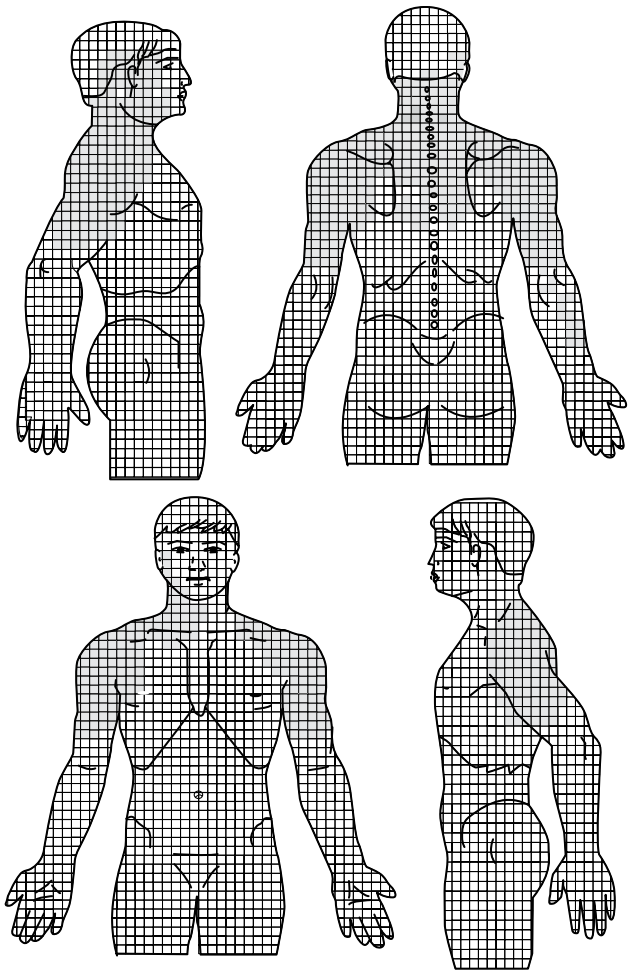

Figure 29.8 C4-5 Discogram pain referral map. From Ref. [20].

and/or interscapular painful symptoms. If nerve root tension maneuvers and diagnostic imaging studies such as a cervical spine MRI are negative for significant lower disc (C5-T1) or lower nerve root pathology, then CFJS would be more likely than CIDD or cervical radicular pain. In this scenario, one would need to investigate the lower cervical facet joints at C5-6, C6-7, and C7-T1.

Unilateral Versus Bilateral Cervical Pain

Determining whether the symptoms are unilateral or bilateral can further organize the differential diagnosis. Unilateral symptoms of cervical axial neck pain with or without occipital headaches or upper back pain are more suggestive of CFJS than CIDD, especially if there is an exquisitely tender focus overlying a facet joint. Unilateral cervical axial neck pain in the absence of a tender focus without scapular symptoms or headaches could emanate from a cervical facet joint or an intervertebral disc. In this scenario, physical examination findings of cervical extension more painful than cervical flexion may be more suggestive of CFJS than CIDD, especially in the setting of a normal cervical spine MRI. Likewise, physical examination findings of cervical flexion more painful than cervical extension with a cervical spine MRI demonstrating a cervical disc protrusion would be more suggestive of CIDD than CFJS. Bilateral cervical axial upper neck pain with or without suboccipital pain or occipital headaches is more likely to be caused by CIDD than CFJS [20]. This clinical observation is supported by the fact that bilateral cervical axial neck pain was produced in 34% to 50% of cervical intervertebral discs stimulated during cervical discography [19]. Slipman's [20] study also demonstrated that 30% to 62% of discs produced bilateral cervical axial neck pain. Dwyer's [8] study investigated pain referral patterns of cervical facet joints in normal volunteers and did not demonstrate any bilateral cervical neck pain from unilateral facet joint distention.

In general, bilateral neck or upper back pain, headaches, or symmetric upper arm symptoms are more suggestive of CIDD. Cervical discogenic pain can typically trigger bilateral neck pain that is equally painful on each side or in the perfect midline. Bilateral neck pain symptoms that are symmetric and equally painful on each side but may alternate from one side to another are still likely discogenic in nature [46]. If there is a traumatic history such as whiplash, then the law of parsimony no longer

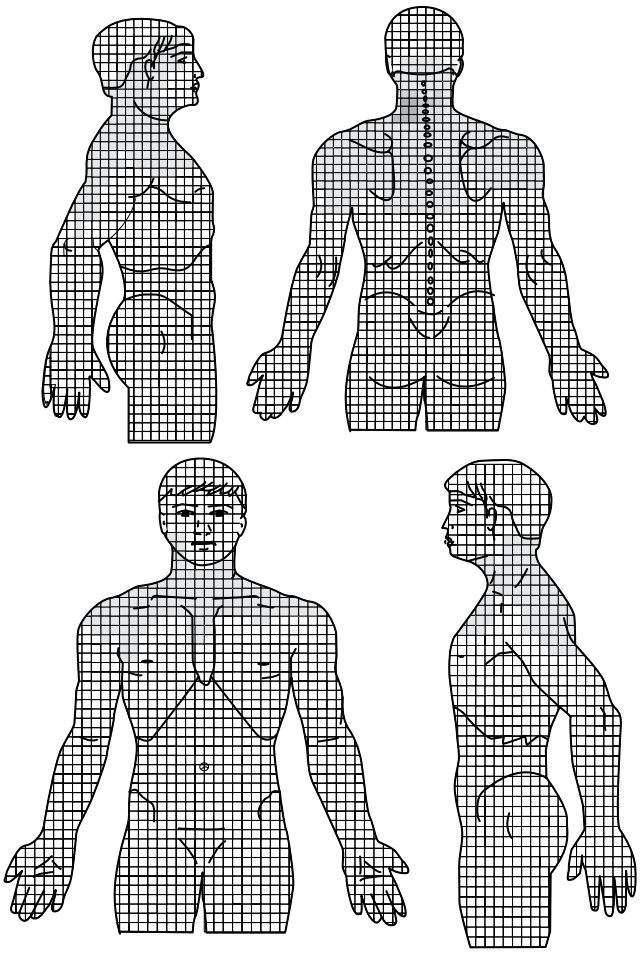

Figure 29.9 C5-6 Discogram pain referral map.

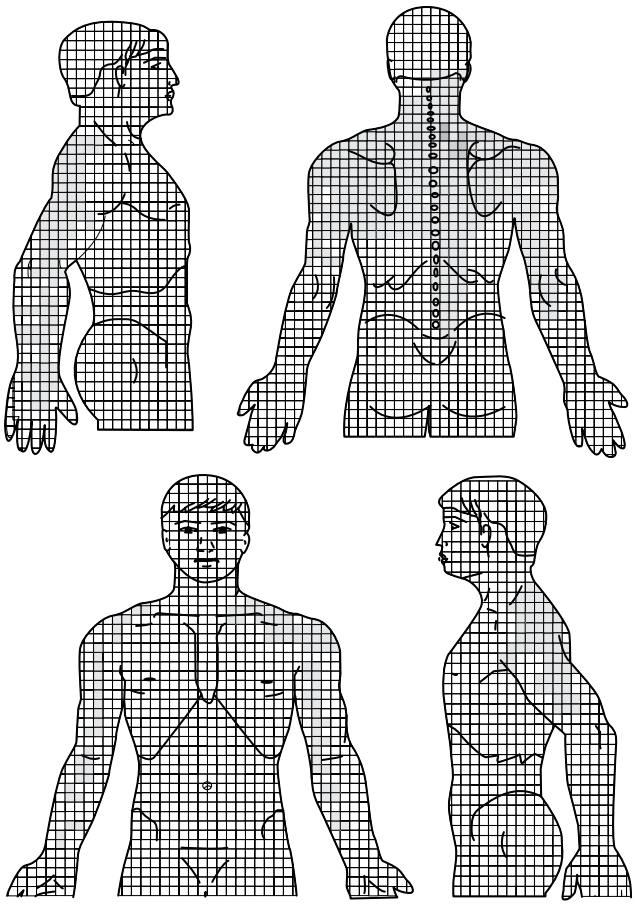

Figure 29.10 C6-7 Discogram pain referral map.

applies. In this scenario, the clinician must be cognizant that more than one structure may be injured. This can be a combination of one or more facet joints or one or more discs or a combination of facet joints and discs. If bilateral symptoms are present in the setting of a traumatic event, the clinician would need to initially evaluate for discogenic pain unless there is high clinical suspicion for CFJS based on focal facet pain that is corroborated on physical examination and without MRI evidence of a correlating cervical disc protrusion.

Physical Exam Findings

Physical exam nerve root provocative maneuvers such as the Spurling's test, which dynamically narrows the neural foramen, can also be utilized to assist in evaluating for cervical radicular pain. The Spurling's test involves cervical extension, rotation, and lateral bending to the affected side with axial compression applied to the top of the head for up to 1 minute (Figure 29.12). The Spurling's test is positive if there is a reproduction or increase in the usual radicular symptoms. If the Spurling's maneuver is positive, then cervical nerve root involvement is more likely than CFJS or CIDD syndrome. Information from a positive provocative maneuver such as the Spurling's test or nerve root tension maneuver is helpful when there are no detectable myotomal deficits or reflex changes in a patient with a nondermatomal distribution of upper limb pain. The conventional neurologic examination has acceptable diagnostic accuracy but the strongest cluster of clinical signs, which together, gave a 90% probability that the patient had a cervical radiculopathy, was collectively a positive upper limb nerve provocation test (median nerve bias), less than 60° cervical rotation range and positive Spurling and distraction tests [47].

Nerve tissues are sensitive to inflammation secondary to tissue trauma or pathology and can contribute to the pain and movement dysfunction of cervicobrachial disorders in both acute and chronic states. Neural tissues can be sensitive to movement, can become allodynic, and contribute to the pain state with or without the presence of a cervical radiculopathy [48–50]. Both basic and clinical research have demonstrated upper limb movement combinations and sequences that move and stress the upper limb peripheral nerves and the brachial plexus [51–53]. Various movement sequences are used in tests of upper limb neurodynamics to bias the movement and stress to median, radial, and ulnar nerves (Figures 29.13–29.15). Obviously other tissues can be stressed in the test sequences, analogous to the hamstring muscles in the straight leg raise test

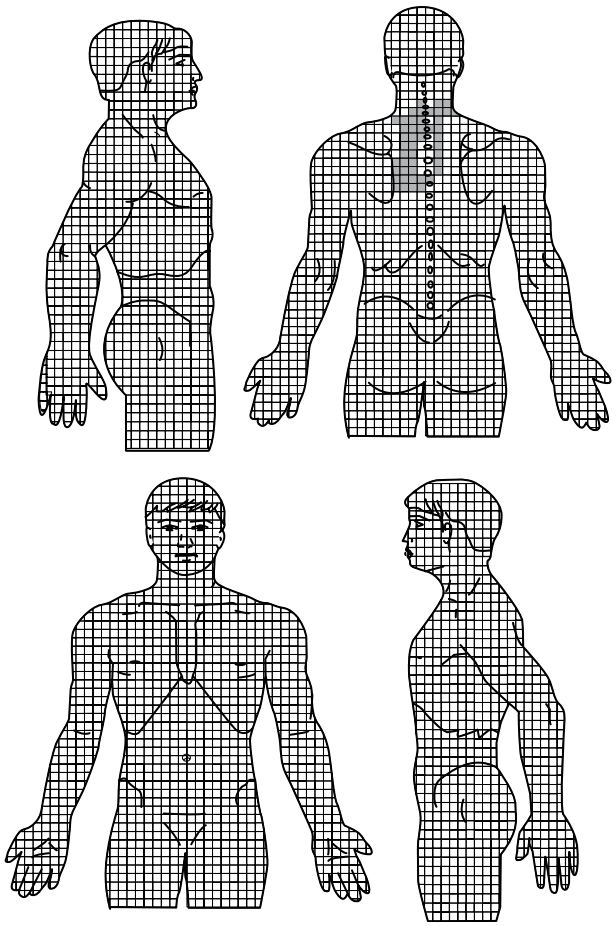

Figure 29.11 C7-T1 Discogram pain referral map.

Figure 29.12 Spurling's test: The test narrows the neural foramen through a combination of cervical extension, rotation and lateral bending to the affected side with axial compression applied to the top of the head.

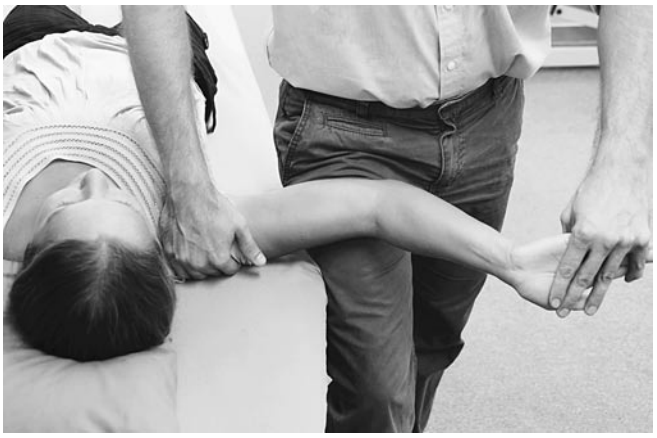

Figure 29.13 Upper limb neurodynamic test (or nerve tension test): median nerve bias

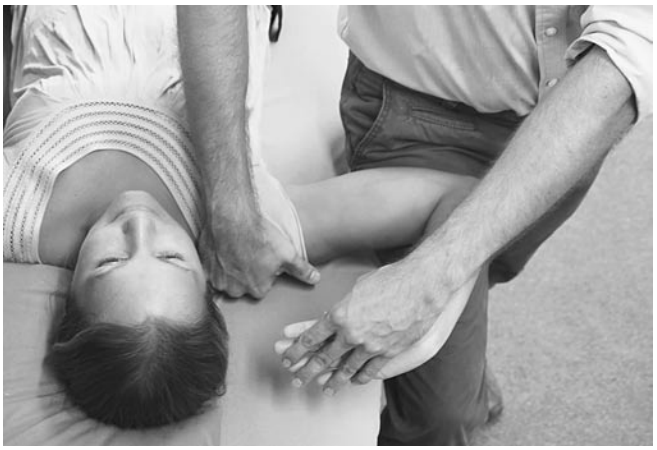

Figure 29.14 Upper limb neurodynamic test (or nerve tension test): ulnar nerve bias

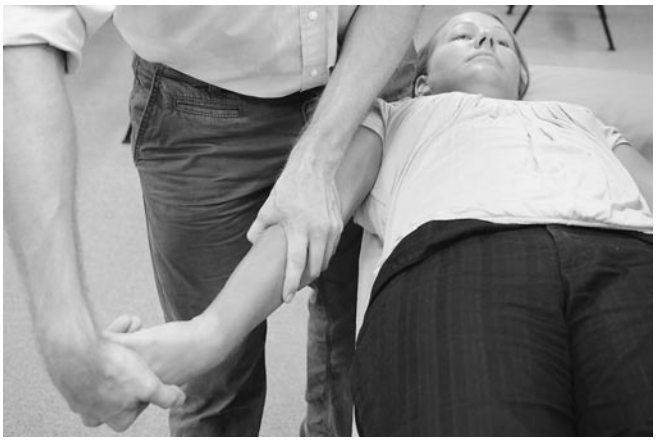

Figure 29.15 Upper limb neurodynamic test (or nerve tension test): radial nerve bias

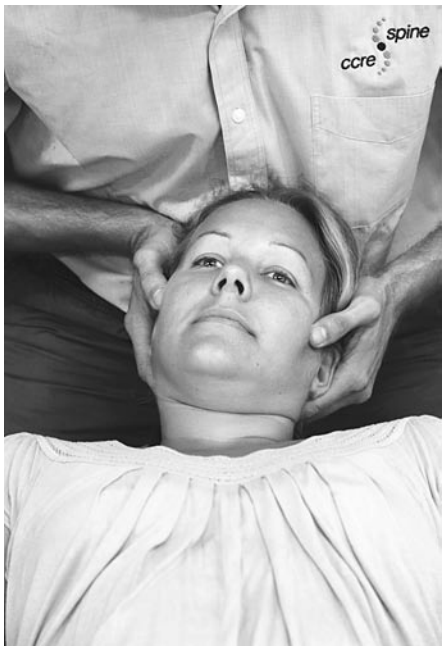

Figure 29.16 Manual examination of segmental lateral flexion movement

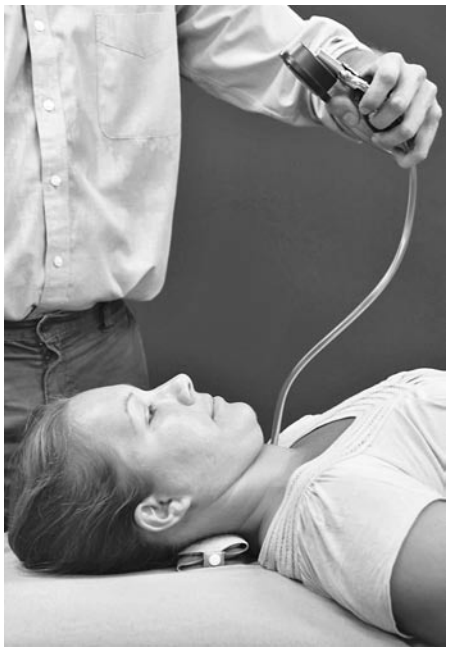

Figure 29.17 The cranio-cervical flexion test. A test of the activation and isometric endurance capacity of the deep cervical flexors (longus capitis and colli) and their interaction with the superficial cervical flexors. It is a low load test of motor control rather than a strength test.

for the low back. A positive result is gained when the final movement in the test sequence is limited in excursion; the patient's pain is reproduced and this pain can be altered with the addition of contralateral cervical lateral flexion and/or craniocervical flexion (Figures 29.16 and 29.17). In contrast, the test on the unaffected limb is classically not restricted and produces no symptoms or a tissue stretch discomfort [54]. While upper limb nerve provocation tests have been shown to have high sensitivity to detect cervical radiculopathy, their specificity is low [55]. This indicates that the tests may be positive in other conditions, for example, in peripheral nerve disorders [56]. There is also evidence that responses, on occasions, may be reflective of the presence of a more general central hypersensitivity state. This was evident in a study of patients with recalcitrant and persistent whiplash-associated disorders where in general, there was a bilateral, rather than unilateral restriction of test sequences even in patients not reporting arm pain [57]. This bilateral restriction in upper limb test sequences was argued to represent a generalized hyperalgesic response to the test in this patient group, possibly reflecting the presence of central nervous system hyperexcitability.

Patients with chronic cervical axial neck pain without radicular symptoms may likely have CFJS, especially in the setting of a normal cervical spine MRI without evidence of a focal disc protrusion. This high clinical suspicion for CFJS is further supported by physical examination findings that are suggestive of CFJS, such as more prominent cervical neck pain with cervical extension when compared to flexion and cervical paraspinal muscle tenderness with manual palpation overlying the offended cervical facet joint(s) or if the patient is able to point to the painful area corresponding to the distribution of pain reported for a particular facet joint [58]. There is better evidence, although not unequivocal, for the value of palpation for facet joint or segmental tenderness to gain some localization of a potential segmental source of pain[58–62]. While paravertebral palpation will necessarily include tenderness of paravertebral muscles, a lateral approach, shifting the bulk of the cervical extensor muscles medially, will allow more localized palpation over the cervical articular pillars to elicit facet joint pain (Figure 29.18). Tenderness elicited by a gently applied manual "springing" force over the spinous process will, in the absence of paravertebral tenderness, point more to a "discal" syndrome (Figure 29.19). Such information, in conjunction with other findings of the examination, can assist in diagnosis of the segmental source of pain and also may contribute information for the application of other diagnostic tests such as facet or nerve blocks [58]. For example, the atlantoaxial segment's contribution to axial rotation is reasonably uniquely assessed when head rotation is tested in full neck flexion, an important test in the evaluation of the cervicogenic headache patient [63–66]. Reduced range of rotation in flexion with pain would point to C1-2 joint (Figure 29.20). CFJS rarely refers pain distal to the elbow; therefore, forearm or hand symptoms suggest nerve root involvement [10,11]. Unilateral neck pain with or without headaches or upper back (scapular, periscapular) pain is more suggestive of CFJS than IDD. Patients with cervical axial neck pain and a high clinical suspicion of CFJS, who have failed a reasonable trial of nonoperative treatment, are candidates for a fluoroscopically guided diagnostic intra-articular facet joint injection or medial branch block.

Figure 29.18 Manual examination of the C2-3 facet joint. The bulk of the cervical extensor muscles are shifted medially to allow more localized palpation over the cervical articular pillars to elicit facet joint pain.

Figure 29.19 Manual examination: Pain elicited by a gently applied manual "springing" force over the spinous process will point more to a "discal" syndrome.

Diagnostic Algorithm

Imaging

Cervical flexion and extension radiographs should be ordered in patients with a whiplash injury or a history of trauma to the cervical spine. Cervical radiographs are not routinely ordered in nontraumatic cases due to its low diagnostic yield. Cervical spine MRI (see Chapters 30 and 39) is the imaging study of choice due to its high sensitivity for detecting soft tissue abnormalities such as a focal disc protrusion or actual nerve root impingement [67–71]. Cervical spine MRI is typically indicated when first-line conservative treatments such as physical therapy, medications, and activity modifications have failed. Cervical MRI is also indicated when there is acute intractable pain that prevents the patient from tolerating physical therapy and severely restricts their quality of life and activities of daily living. MRI findings of degenerative changes

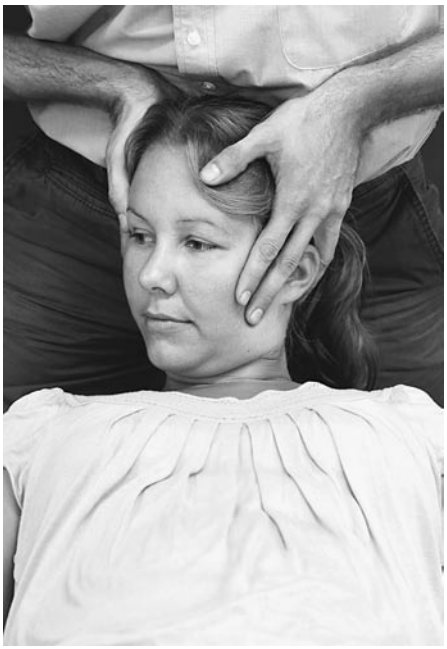

Figure 29.20 The flexion rotation test: Rotation of the head performed in preflexed position of the neck biases the movement to the C1-2 segment

involving the cervical discs, facet joints, and neuroforamina are common in both traumatic and atraumatic cases. Discogenic findings of a focal disc protrusion, annular disc tear, or a dark broad-based degenerative concentric annular disc bulge may or may not be clinically symptomatic [72–75]. However, in patients with a high clinical suspicion of CIDD, these discogenic findings on MRI can assist in determining which disc level to treat for a fluoroscopically guided cervical epidural steroid injection. Similarly, cervical facet joint arthropathy or hypertrophy on an imaging study is not diagnostic but can enter CFJS in the diagnostic algorithm if there is clinical suspicion for CFJS. Given the existence of asymptomatic imaging findings, clinical specificity is important. The clinician needs to correlate each patient's history and examination findings with the imaging study to formulate an accurate differential diagnosis that will lead to the correct clinical diagnosis.

Facet Joint Syndrome

In cases of upper neck pain with or without headaches in the setting of whiplash or trauma, diagnostic facet joint blocks are performed sequentially at C2-3, C3-4, and C1-2, until the offended joint is identified. This sequence is based from clinical experience and epidemiologic studies [14,29]. These studies identified that 50% of all patients with chronic whiplash-induced cervicogenic occipital headaches experience these symptoms due to C2-3 facet joint pain (Figure 29.21). Whiplash-induced lower neck pain emanating from a facet joint was most common at the C5-6 facet joint level [14,29]. The estimated prevalence of CFJS ranges from 36% to 67% based upon multiple studies with heterogenous populations [10,13,14,21,76–80]. Once

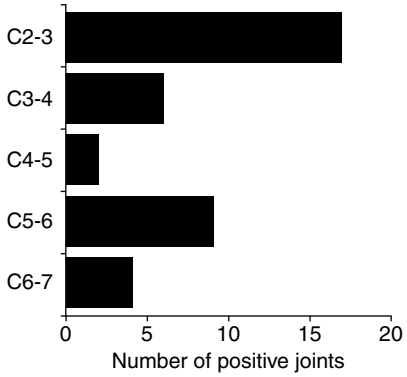

Figure 29.21 Whiplash-induced prevalence of CFJS by facet joint level. From Ref. [29].

the offending facet joint has been identified by diagnostic blocks, therapeutic steroid facet joint procedures or radiofrequency medial branch neurotomy can be performed (see Chapter 36).

Cervical Internal Disc Disruption

If the MRI demonstrates a focal disc protrusion or broad-based concentric annular disc bulge that correlates with their clinical symptoms, then a fluoroscopically guided transforaminal epidural steroid injection at the suspected symptomatic disc level is recommended. If there is high clinical suspicion for CIDD but the MRI reveals multilevel disc disease or does not demonstrate an obvious symptomatic disc level, a fluoroscopically guided epidural steroid injection can be performed at the C6-7 disc level to treat patients with lower neck pain. The epidural steroid injection can be performed at the C4-5 disc level for patients with upper neck pain. If a therapeutic effect is not realized after one or two injections, then further epidural steroid injections should be abandoned. If there is a high clinical suspicion for CIDD and the patient has failed epidural steroid injections, then the clinician must determine whether the patient's axial neck pain is intractable and whether the patient is willing to undergo cervical disc fusion or disc arthroplasty surgery to treat their axial neck pain. If the patient's axial neck pain is intractable and the patient is willing to undergo cervical disc surgery, then a diagnostic cervical discogram (see Chapter 31) would be the next treatment step to determine if there is symptomatic IDD in the cervical discs. If the discogram reveals one or two successive disc levels with symptomatic posterior annular fissures and concordant pain responses, then the patient may be a candidate for cervical disc surgery. If the discogram does not demonstrate a posterior annular disc tear with a concordant pain response, then the discogram is negative and CIDD is unlikely. In this situation, the next suspected structure in the diagnostic algorithm should be evaluated. If the discogram reveals three or more positive disc levels, two positive disc levels with an intervening normal disc, or if any concordantly painful discs are lobular, then a comprehensive chronic pain program or functional restoration program is suggested.

Diagnostic Blocks and Treatment Failure

Single diagnostic cervical facet joint blocks have an ascribed false positive rate ranging from 27% to 63% [11,13,21,76,81]. Consequently, it is possible that the initial diagnostic block may be a false-positive response. Performing a second, confirmatory diagnostic facet joint block with a different comparative local anesthetics or a third injection of a placebo control will limit false positive rates associated with single diagnostic blocks. This is important when considering the next treatment step in a patient who fails to improve with a therapeutic intervention such as a steroid facet joint procedure or medial branch neurotomy. In these instances, a confirmatory diagnostic injection using a placebo control or using two different comparative local anesthetics is recommended. This confirmatory diagnostic facet injection will assist in determining whether the patient is a true nonresponder to the therapeutic facet intervention or whether the treatment failure was due to a false-positive response from the initial single diagnostic facet block. If the confirmatory diagnostic block demonstrates a false-positive response, then the next suspected structure in the diagnostic algorithm should be addressed.

SUMMARY

Over the past two decades, the utility of cervical spinal injection procedures in the treatment of painful cervical spine conditions has been investigated by numerous research studies. Given the trend for evidence-based medicine, the most recent research studies over the past decade have been of much higher quality with improved methodology. These studies have primarily investigated disorders involving one of three primary cervical structures: zygapophyseal joint, intervertebral disc, and nerve root. An algorithmic paradigm that incorporates the use of fluoroscopically guided cervical spinal injections in the treatment of painful cervical spinal disorders is provided for the clinician to systematically formulate a differential diagnosis and treatment plan, while minimizing unnecessary injection procedures. This algorithmic process will continue to be revised and updated as new clinical information is published and incorporated into clinical practice [82].

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30 Diagnostic Imaging of Painful Cervical Discs

Yinggang Zheng, Kaixuan Liu, and Edward D. Simmons

Neck pain is a common symptom in the general population with a self-reported incidence of 213 per 1000 persons. The incidence of neck injuries during competitive sports activities ranged from 0.02 to 21 per 1000 exposures [1]. Cervical internal disc disruption (CIDD) syndrome can be a source of neck pain. CIDD is the presence of internal annular fissure, which can be categorized into inner annular fissure and major tears involving both the inner and outer layers of the annulus without apparent disc herniation.

The CIDD syndrome, commonly known as cervical discogenic pain, is caused by the leakage of nuclear pulpous through the tears, and the irritation of the nociceptors within the annular fissures. It presents as chronic axial neck pain with or without scapular or shoulder pain, which may refer into the upper arm but rarely extends down to the forearm and hand. Some patients reported tingling feelings within the upper limb without objective neurologic deficit. Many patients also experienced occipital headaches. Before establishing this diagnosis, the clinician should first rule out other disorders causing neck pain, such as a cervical facet joint or apparent cervical disc herniation with direct compression of the ipsilateral nerve root. Diagnostic studies, including plain x-ray, magnetic resonance imaging (MRI), cervical provocation discography with postdiscography computed tomography (CT), assist in affirming the diagnosis.

PLAIN FILM RADIOGRAPHY

If a patient presents with chronic neck pain similar to that caused by CIDD syndrome, anteroposterior, oblique, and lateral view, particularly dynamic motion radiographs of the cervical spine should be considered as the initial workup. X-ray studies are particularly necessary for patients with a history of trauma, inflammatory disease, spine fusion, malignancy, compromised immune system, chronic pain with failed conservative treatment, or being involved in litigation. A review study by the Neck Pain Task Force group recommends that people seeking care for neck pain should be triaged into four groups: grade I neck pain with no signs of major pathology and no or little interference with daily activities; grade II neck pain with no signs of major pathology, but with interference with daily activities; grade III neck pain with neurologic signs of nerve compression; grade IV neck pain with signs of major pathology. In the emergency room, after blunt trauma to the neck, those patients with a high risk of fracture should be further examined with plain radiography and/or CT scan [2].

It should be recognized that compared with MRI and discogram, plain films of the cervical spine have little value in directly diagnosing discogenic pain but nevertheless they can help physicians rule out other gross trauma, fracture, dislocation, and tumors. Radiographs are used to assess the stability of vertebral alignment, disc space narrowing, and facet joint arthropathy. In patients with neck pain who suffer paraspinal muscle spasms, the cervical spine alignment presents straightened with decreased cervical lordosis. As discogenic pain is more and more commonly seen in young and middle-aged patients, radiographic findings can be normal or with mild disc degeneration [3]. More substantial degenerative changes are seen in older patients. Little correlation exists between symptomatic and asymptomatic individuals and structural changes on roentgenographic examinations [4]. In patients with previous cervical bony trauma or fusion, more attention should be paid to the adjacent disc level for possible internal disc disruption from exacerbated degeneration and/or instability. In patients with normal plain x-ray results but persistent axial neck pain, additional diagnostic imaging should be considered to further evaluate a patient's chief complaint of axial neck pain.

CT, MYELOGRAPHY, AND CT MYELOGRAPHY

Because of its weak delineation of disc morphologic changes, CT alone is rarely used for diagnosing CIDD. However, CT is routinely used immediately following cervical discography to identify the location of annular fissures and leakage of contrast dye. Cervical discography is discussed in detail elsewhere in this textbook (see Chapter 31). Myelography and CT myelography, nowadays are rarely used to evaluate primary axial neck pain and have been widely replaced with MRI, are only used to resolve a suspicion of serious spinal pathology, cancer, infection, myelopathy, or progressive motor deficit.

Ruling out facet joint-originated neck pain can help establish the diagnosis of CIDD syndrome, which is difficult to surmise based solely on CT findings. Findings on CT studies alone are not able to differentiate the pain

caused by internal disc disruption from the pain of facet joint origin (see Chapter 33). Facet joint pain can be diagnosed with controlled, diagnostic, and local anesthetic blocks [5] (see Chapter 34). An immediate relief of neck pain confirms the facet as a pain generator, or a portion of the pain origin. Recent literature supports using cervical medial branch block as a preferred technique [5,6].

MAGNETIC RESONANCE IMAGING

MRI is extensively used to evaluate cervical disc morphology, the contour of the outer annulus, disc degeneration, and canal stenotic changes. It has been established previously that MRI is very useful for the diagnosis of lumbar and cervical disc herniation [7–11]. With respect to cervical discogenic pain, after other causes of neck pain have been ruled out, it is difficult to pinpoint the painful disc based solely on disc morphology on MRI [11–13]. Zheng and colleagues [11] investigated the value of MRI and discography for evaluating cervical discogenic pain. The authors retrospectively reviewed the surgical outcome of 55 patients (involving a total of 161 disc levels), who had been clinically diagnosed with cervical discogenic pain for at least 6 months and underwent an anterior cervical discectomy and fusion using the Simmons Keystone technique and who were available for a follow-up of a minimum of 2 years. All of the patients had a preoperative MRI scan, discography, and a postdiscography CT scan. In evaluating the MRI images of the cervical spine, the sagittal T2 spin echo images were used to evaluate the disc nuclear signal intensity and the status of posterior annulus as described by Horton and Parfenchuck, with some modifications [7,11,12]. The nuclear signal intensity on MRI reflects the hydration of nuclear pulpous of the disc, which can be described as white, speckled, or dark. A white disc has a hyperintense, homogenous signal; a speckled disc has a heterogenic pattern with areas of hypointense changes with two or more areas of hyperintense signal; a dark disc has a homogenous and hypointense signal. The integrity of the posterior annulus can be classified as flat, bulging, torn, or herniated. A flat posterior annulus is straight or minimally convex; a bulging annulus is a gradually convex annulus that slightly encroaches on the thecal sac in any sagittal image; a torn annulus has a discontinuity in the signal of posterior annulus; a small herniation is a disc injury in which the outer annular fibers are intact and there is a small amount of spinal canal encroachment, either focal or more circumferential on lateral view or axial view (Figure 30.1) [7,11,12].

Nuclear Signal Intensity and Neck Pain

The signal pattern of disc nucleus depends on the water content of the disc. The changes in hydration state result in different nuclear signals on MRI and usually correlate with nuclear deterioration [14,15]. The relationship between CIDD and discs with a hypointense signal or dark discs on MRI is not entirely clear [11,12]. It has been reported that the dark nuclear pattern and torn annulus have a high correlation with a positive lumbar discography [7,16]. However, the correlation is not definitive but rather suggestive if dark and torn annular patterns were seen together at one level [7]. Some physicians simply assume that the dark cervical discs are pathologic and therefore symptomatic [12]. Parfenchuck and Janssen [12] concluded that a dark or torn disc on cervical MRI may not need cervical discography because these discs are invariably the source of neck pain, but they also admitted their study was limited by the small number of dark discs included in their study. The result of Zheng's study strongly suggests that the dark cervical disc is not always symptomatic. Only 63% of the dark discs in chronic neck pain patients were positive on discography (see Chapter 31), and they accounted for almost half (49%) of the symptomatic levels in the study group [11]. This study provides valuable information to the clinician beyond cervical MRI in evaluating chronic discogenic neck pain. In the lumbar spine, the white bulging and white flat discs were found to have a 90% chance of having no pain with discography [7]. A previous study classified white cervical disc on MRI as normal. However, results of the study also showed 63% of the discs with positive discography [12]. In Zheng's study, although the authors categorized speckled

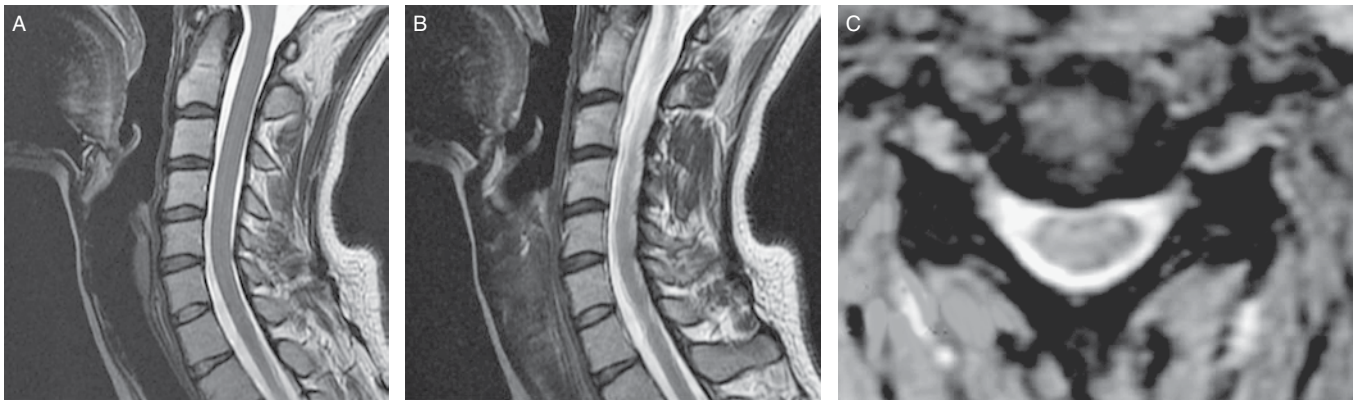

Figure 30.1 **(A)** Showing the white at C6-7, speckled at C4-5, C5-6, and dark at C2-3 C3-4 of signal intensity on MRI. Flat disc at C6-7 C7-T1, and bulging disc at C3-4, C4-5 C5-6, as well. **(B)** Showing torn disc on C3-4 and C4-5. **(C)** Showing small disc herniation.

and flat, white and bulging, and white and flat discs as probably not being pain generators, the results showed that some of these discs are the source of neck pain determined by discography. Therefore, to gain acceptable clinical results, the authors suggested not depending solely on the morphology of the disc on MRI for the selection of surgery level.

Annular Contour and Neck Pain

The status of the posterior annulus is another important aspect for evaluating the morphology of the disc on MRI. Most of the published literatures about discogenic pain are the studies of MRI and discography on the lumbar spine. Previous studies of the lumbar spine defined three categories for evaluating the posterior annulus: torn, bulging, or flat [7]. In Zheng's [11] study, the authors added into the categories a small-herniated disc where the outer annular fibers were intact and only a small amount of focal or more circumferential spinal canal encroachment was seen. The reason for adding this subtype is that the cervical spine is different from the lumbar spine [12,13,17]. The authors tried to determine whether the small-herniated disc was always a source of pain. Their results showed that positive discography was found in 59% of the small-herniated discs and torn discs, 35% of bulging discs, and 29% of flat discs, respectively. Dark and small-herniated pattern was the most commonly operated on, and 67% of the discs with this pattern had a positive discography. In decreasing order of frequency, the discs with dark and torn, speckled and small herniated, and speckle and torn patterns were also painful.

Relationship Between MRI and Cervical Discography

The knowledge of cervical disc disruption, classification, and discogram are mostly extrapolated from lumbar disc disruption and discogram [7]. Although cervical disc disruption and lumbar disc disruption share some common characteristics, because of the unique anatomy of the cervical vertebra and intervertebral disc, substantial differences exist between cervical and lumbar disc disruption and clinical implication [8,13].

Because of its ability to determine whether or not a patient has discogenic neck pain and to identify which intervertebral disc is responsible for the persistent pain, cervical discography has been the most useful diagnostic tool to directly evaluate internal disc disruption. Discography represents a functional diagnostic test because the patient's subjective response is integral in the outcome of the test. On the other hand, visual anatomic imaging can demonstrate whether or not there is an internal disc disruption and can match the result of the internal disc disruption testing with the symptomatology, for example, a disc has moderate tears and causes pain, and another disc has severe disc leaking in all directions but does not cause provocated concordant pain [14].

In general, disc disruption is proportional to the degeneration of the disc [7]. Cervical trauma can also cause or exacerbate this process. DePalma and Slipman [18] categorized the nucleogram into four categories: lobular, irregular, fissured, and ruptured (Figure 30.2).

Previous studies have proven that the presence of internal disc disruption does not always cause discogenic pain in the neck [11,13]. Internal disc disruption is more commonly seen in degenerated discs [13]. However, asymptomatic internal fissures have been seen by discography in asymptomatic individuals with normal MRI appearance of cervical discs [13]. Moreover, even in symptomatic patients, some discs with fissuring do not cause pain although some discs with fissuring do cause discogenic pain by provocative discography [11,13]. On the other hand, Schellhas et al. [13] prospectively correlated MRI imaging and discography in 10 asymptomatic subjects and 10 patients with chronic axial pain. The authors found that various degrees of annular disruption were common in both symptomatic and asymptomatic patients. The ruptures were usually posterior-lateral and full-thickness tears and leaks into the epidural space occurred in about 50% of the discs. Annular tears were identified during discography in 23 of the 24 discs judged normal based on MRI scans. Parfenchuck and Janssen's [12] study also showed that leakage illustrated either on discography or on CT discography did not correlate with whether or not a cervical disc is painful. Fissures in the annulus in the uncovertebral region occur commonly in cervical discs [8,13]. Leakage of contrast medium through such fissures results in discographic patterns that, in the lumbar region, would be considered abnormal. In the cervical region, these patterns, however, must be considered as the presence of disc degeneration rather than a pain generator in the absence of painful response during discography [8,13].

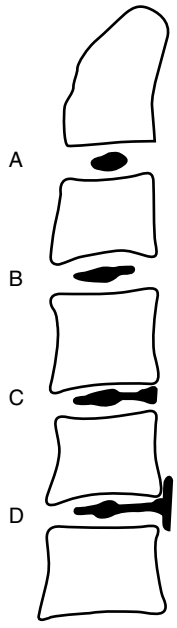

Figure 30.2 Schematic illustration of lobular **(A)**, irregular **(B)**, fissured **(C)**, and ruptured **(D)** nucleograms. Courtesy of DePalma MJ, Slipman CW. Technique and indication of cervical discography. In: Freeman MK, Morrison WB, Harwood MI, eds. Minimally Invasive Musculoskeletal Pain Medicine. New York: Informa Healthcare, 2007:235–247.

Parfenchuck and Janssen indicated MRI correlates reasonably well with provocation discography. They suggested that cervical discs abnormal on MRI are likely to be painful whereas discs normal on MRI are not likely to be painful. However, they admitted that this correlation is not absolute [12]. The diagnosis of the possible symptomatic disc level was based on the abnormal morphology of the T2 MRI scans. In Zheng and colleague's study, with respect to the disc signal, a dark disc was the most significant. A speckled disc and white disc were of less significance. With regard to the posterior annulus, a small-herniated or torn annulus was considered the most significant. The bulging annulus and flat annulus were of less significance, again in that order. Thus, the discs that were thought to be probably symptomatic were the dark and herniated, dark and torn, dark and bulging, speckled and herniated, speckled and torn, and white and herniated discs. The discs that were thought to be possibly symptomatic were the dark and flat, speckled and bulging, and white and torn discs. The speckled and flat, white and bulging, and white and flat discs were considered probably not as pain generators [11]. These possibilities of discogenic pain were finally tested using cervical discography. Parfenchuck and Janssen [12] studied the association between abnormality on MRI and pain response on discography in 101 disc levels using discography, and they concluded that MRI has sensitivity of 73%, a false-negative rate of 27%, specificity of 67%, and a false-positive rate of 33% for detecting a painful disc. With more patients and more types of disc levels being tested, Zheng and colleagues studied 55 patients in 161 disc levels, the overall MRI findings in 103 of the 161 (64%) injected levels correlated completely with the results of discography. With the sensitivity of 73.4% and the false-negative rate of 26.6%, MRI resulted in only a specificity of 49% and a false-positive rate of 51% for discriminating the painful cervical disc from asymptomatic levels [11].

SUMMARY

The CIDD syndrome presents as axial neck pain. The diagnosis of CIDD relies on ruling out other possible causes of pain via medical history and physical examination, diagnostic imaging, and x-ray–guided diagnostic injections. MRI shows the morphology of a disc, whereas discography demonstrates the pathophysiology of the probable pain generator. MRI can be used to help identify the possible painful discs but, unfortunately, it still has high false-negative and false-positive rates. Dark and small-herniated discs have a high chance of being the pain generators but are not always symptomatic. MRI is recommended to be used as a screening test to identify the levels of the possible painful discs, whereas discography can be used as an adjunctive confirmatory test. Discography can protect the levels from unnecessary interventional procedures or fusion or disc replacement surgery. The combination of clinical symptoms, MRI, and discography provides the most information for decision making and can improve the management of cervical axial pain.

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31 Provocation Discography Evaluation of Symptomatic Cervical Discs

Brian S. Trussell and Michael J. DePalma

INTRODUCTION

Perhaps a clinical vignette best encapsulates a challenging yet common presentation. A 43-year-old male presents with a 12-month history of midline mid- to lower cervical pain, which refers to bilateral upper trapezii to the tip of each acromion as well as inferiorly along the spine to the level of the scapular spine. Symptoms are exacerbated by prolonged sitting, cervical flexion, coughing, and sneezing and alleviated by lying supine without pillows under the head. Physical examination reveals intact and symmetric sensation, strength, and muscle stretch reflexes in bilateral upper and lower limbs. Spurlings and Lhermittes maneuvers are negative but reproduce mild midline axial cervical pain. Plain films and magnetic resonance imaging (MRI) revealed mild degenerative changes per report at multiple segmental levels. Despite previous treatment, including physical therapy, interlaminar and transforaminal epidural steroid injections, this patient returns with recurrent and disabling symptoms. What is the next step in the diagnostic and/or therapeutic workup? Unfortunately, this situation is encountered in many interventional spine clinics across the world.

Axial neck pain continues to be a common chief complaint seen in the clinical setting with a mean point prevalence of 7.6% (range of 5.9%–38.7%) and a lifetime prevalence of 48.5% (range of 14.2%–71.0%) [1]. Other studies have demonstrated that the prevalence of neck pain with or without upper limb pain ranges from 9% to 18% of the general population [2–5], and one out of three individuals can recall at least one incidence of neck pain in their lifetime [2]. Although there is no consensus regarding precise prevalence rates, these epidemiologic reports and systematic reviews all confirm that cervical pain complaints are ubiquitous. Neck pain is encountered more frequently in clinical practice than low back pain [6], and traumatic neck pain can become chronic in approximately 40% of patients, with 8% to 10% reporting severe pain [7]. The occurrence of neck pain increases in the workplace [8,9]. Its frequency increases with age; approximately 25% to 30% of workers younger than 30 years of age report neck stiffness, whereas 50% of workers older than 45 years of age report similar complaints [3,4,10].

The cervical intervertebral disc can become a source of chronic cervical axial pain [11–13]. Internal derangement of the intervertebral disc, internal disc disruption (IDD), was first described by Crock over 30 years ago. IDD is defined as an intervertebral disc that has lost its normal internal architecture, but maintains a preserved external contour, in the absence of nerve root compression (Figure 31.1) [14]. In traumatically induced chronic neck pain, 20% of patients suffer from cervical internal disc disruption (CIDD) and another 41% suffer from CIDD and a concomitant facet joint injury at that level [15]. Physical examination findings may be unremarkable [16], and imaging studies typically cannot discriminate symptomatic from asymptomatic cervical intervertebral discs [17–20]. Historical features such as pain referral patterns as described by Slipman et al. [21] are helpful in predicting the source disc level but are not pathognomonic. A significant proportion of patients with chronic cervical axial pain will experience persistent symptoms despite conservative care [12,22,23]. Functional

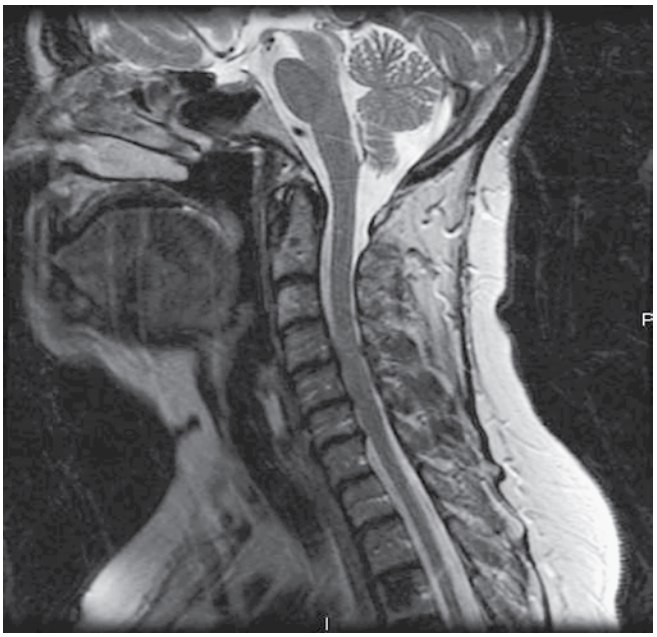

Figure 31.1 Sagittal magnetic resonance imaging of cervical spine illustrating relatively normal disc contour with mild loss of disc height and mild to moderate disc dessication of the upper three cervical discs.

diagnostic testing such as provocation discography in which subjective feedback from the patient is mandatory helps clarify the source of these chronic symptoms and guide surgical intervention [16].

In the late 1950s, Smith [24] and subsequently Cloward [25] independently developed a similar cervical disc injection technique to evaluate patients complaining of cervical axial and shoulder girdle pain. Each investigator discovered that injection of symptomatic discs reproduced the patients' axial complaints. This finding enabled the clinician to identify which segmental level should be targeted with more aggressive therapeutic intervention. Both Smith and Cloward utilized discography to select the appropriate cervical levels for their interbody fusion techniques, which are still practiced today [26,27].

In 1964, Holt's [28] study of 148 cervical intervertebral discs in 50 asymptomatic penitentiary inmates led him to conclude that "cervical discography is a painful and expensive procedure and is without diagnostic value." While we applaud Holt's effort to establish scientific validity (pro or con), we must understand that this study has enough fatal flaws to render its conclusions almost meaningless. For example, Holt completed the procedures using an irritating contrast agent without fluoroscopic guidance [28,29] utilizing an injection technique that has been described as suspect regarding mechanical performance, discometric data, and imaging results [30]. Despite Holt's disparaging claims, cervical discography has been widely studied in playing a viable role as a diagnostic tool to discriminate painfully deranged intervertebral discs from nonpainful adjacent level discs [13,31–35]. Two recent systematic reviews, Shah [36] in 2005 and Buenaventura [37] in 2007, independently concluded that the evidence is strong for the diagnostic accuracy of discography as an imaging tool and that there is moderate evidence supporting the role of discography in identifying a subset of patients with cervical discogenic pain. These studies will be discussed in detail later in this chapter.

INDICATIONS

Painful cervical intervertebral discs manifest clinically as axial neck pain sometimes associated with referred pain into the occipital, scapular, upper limb, head, and chest regions [11,21]. Therefore, indications for performing cervical discography include (1) chronic, intractable neck pain lasting at least 6 months despite medical rehabilitation and interventional spine care [33,38]; (2) cervical radicular pain, positive root tension signs, and equivocal imaging studies [34]; (3) the evaluation of vertebral levels adjacent to the levels facing impending fusion for spondylolisthesis, fractures, instability, postlaminectomy kyphosis, or myelopathy [38]; and (4) prior to therapeutic intradiscal procedures such as disc decompressive techniques [39] or annuloplasty. The treating spine specialist should thoroughly rule out cervical facet joint pain or radicular pain as the source of persistent symptomatology to increase the pretest probability of discogenic pain [40].

CONTRAINDICATIONS

Cervical discography should not be performed in the presence of certain structural spinal abnormalities. Incomplete cervical myelopathy could progress to complete tetraplegia upon disc stimulation in the presence of a large disc herniation [41]. Absolute contraindications to discography include spinal infection, bacteremia, local cellulitis or ulceration, neoplasms, central canal stenosis, uncontrolled coagulopathy [29], and symptoms of myelopathy [42]. Anticoagulant therapy and contrast dye allergy constitute relative contraindications [43]. Patients can be covered on low molecular weight heparin after stopping their Coumadin. Both decisions would need to be approved by the patient's treating medical physician. Gadolinium followed by MRI may be substituted for omnipaque contrast dye in patients allergic to the latter [44].

TECHNIQUE

Preprocedure

Prophylactic antibiotics can be administered but may only be necessary in patients with facial hair, diabetes mellitus, or mitral valve prolapse [42]. One gram of Cefazolin is typically administered intravenously within an hour prior to the procedure. If a patient is allergic to cephalosporins or penicillins, 600 mg of intravenous clindamycin can be substituted for cefazolin [45]. In addition, cefazolin (0.5 mL of 10 mg/5 mL) or clindamycin (0.5 mL of 6 mg/5 mL) may be combined with the nonionic contrast medium (300 mg I/mL) to maximize the concentration of antibiotic within the disc space where the infection is likely to occur [45].

Needle Placement

The patient is placed in the supine position with two folded sheets placed under his or her shoulders in order to position the neck in mild extension [24]. Alternatively, a shallow triangle [29] may be utilized to achieve extension of the cervical spine (Figure 31.2). The patient's head is rotated approximately 10° toward the patient's right side [24] (Figure 31.2) [29]. After the anterolateral neck is prepped with betadine or a non-iodine–based solution in patients with an allergy to iodine or betadine, and then draped with sterile towels, a segmentation count is performed using a cross-table lateral fluoroscopic view [29]. Typically, each segmental level is counted sequentially starting at the C2-3 level and continuing down to one-level caudal to the last level to be studied (Figure 31.3) [29]. Longitudinal distraction of both upper limbs may be necessary to adequately visualize C5-6 through C7-T1 as the overlying shoulder girdles can attenuate the x-ray beam obscuring the cervical bony anatomy. This initial survey will allow the physician to judge the orientation of each intervertebral disc space and adjust the required needle trajectory to place the needle tip within the nucleus.

Using a straight posteroanterior view, the targeted disc space is visualized and the right uncinate process is identified as a landmark (Figure 31.4). The fluoroscope is rotated ipsilaterally approximately 30° to 45° providing the proper

Figure 31.2 A small triangular pillow is placed under the cervical spine to place it into mild extension.

view for initial needle placement. The skin overlying the medial sternocleidomastoid muscle is anesthesized with 1% lidocaine. A 23 to 25 gauge, 2- to 3.5-inch spinal needle is then inserted at that site and advanced under intermittent fluoroscopy into the targeted intervertebral disc. The spinal needle is introduced approximately 30° to 45° obliquely from the midline and slightly inferior to the target disc [24]. Utilizing the left index finger, the carotid artery is pushed laterally and the esophagus is displaced medially away from the projected needle tract (Figure 31.5) [24,25,29].

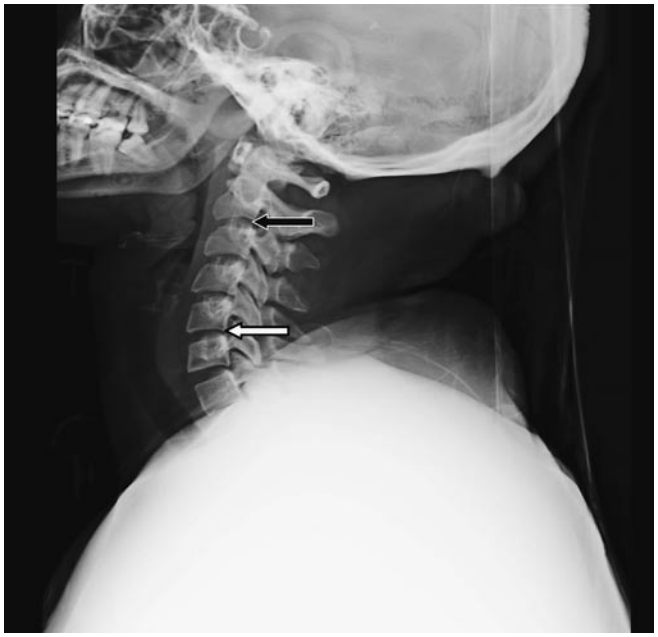

Figure 31.3 Lateral fluoroscopic view of the cervical spine obtained for segmental level count. Black arrow depicts the C2-3 disc space and the white arrow highlights the C5-6 disc space.

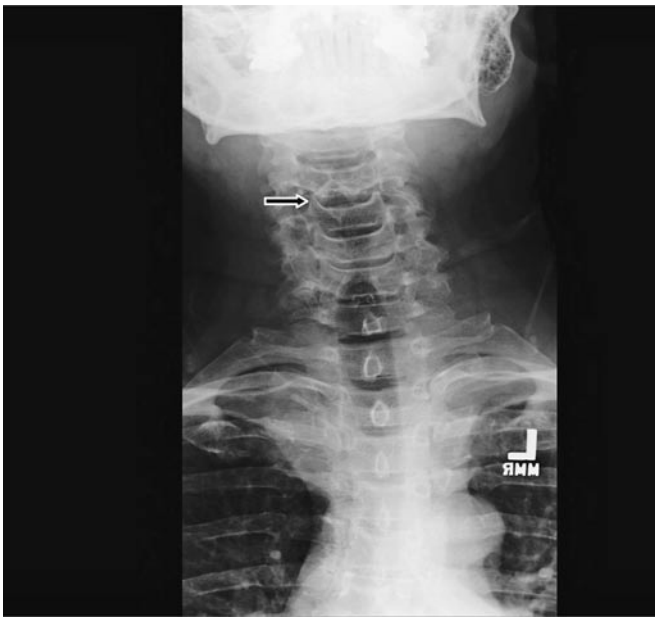

Figure 31.4 A coronal view of the cervical spine allows detection of the uncinate processes (arrow).

This step provides a safe path for accessing the disc while avoiding the great vessels, larynx, thyroid, and esophagus [46]. The carotid pulsations should be palpated by the finger tips as the carotid artery is displaced laterally, and deeper digital pressure will approximate the anterolateral surface of the spine in thin patients [25]. The 25-gauge needle is held in the dominant hand between the index finger and thumb and carefully advanced medial to the uncinate process and into the central portion of the disc [24,25,29]. The needle tip should encounter the superior edge of the caudal vertebral body. At this point, it is walked superiorly until it enters the cephalad intervertebral disc space [24]. The novice discographer should be sure to abut the

Figure 31.5 The left index finger of the examiner displaces the trachea and esophagus medially and the carotid artery laterally.

subjacent vertebral endplate with the needle tip to confirm proper depth before advancing superiorly and puncturing the annular fibers. The patient will experience a sudden, transient moment of cervical and/or shoulder girdle pain upon needle piercing of the annulus [21,29]. Occasionally, anterior spondylotic spurs can partially obstruct entry into the disc space and must be circumvented by the spinal needle. Although the medial border of the sternocleidomastoid serves as a relatively common skin surface entry mark, a more lateral approach may be required to avoid the hypopharynx at the C2–3 and C3–4 levels. In addition, a more medial entry may be necessary to avoid the apex of the lung at the C7-T1 level [29]. The spinal needle will enter the nucleus of the disc as it is directed toward the central third of the disc past the medial border of the uncinate process of the caudal vertebrae. Caution must always be exercised to avoid advancing the needle tip through the disc into the spinal cord. Needle position must be analyzed in both the posteroanterior and lateral views to ensure proper height and depth confirming needle placement within the central third in both planes.

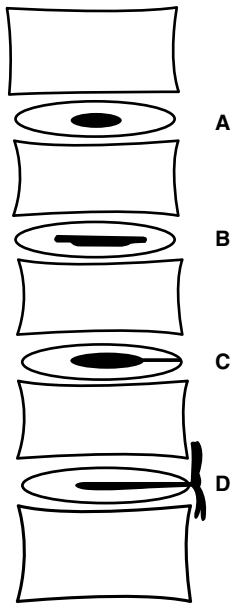

Figure 31.6 Schematic illustration of **(A)** lobular, **(B)** irregular, **(C)** fissured, and **(D)** ruptured nucleograms.

Interpretation

Following successful needle placement, nonionic contrast dye is injected under live fluoroscopy in the lateral view. Intraoperative measurements have demonstrated that intact cervical discs will maintain high intradiscal pressures upon injection of 0.2 to 0.4 mL of solution [47]. In contrast, discs that allowed posterior extension of contrast dye accommodated 1.5 mL of volume at low, wavering pressures [47]. Herniated or degenerated discs with intact outer annular fibers accepted intermediate volumes of 0.5 to 1.5 mL at sustained, but intermediate, intradiscal pressures [47]. Cervical intervertebral discs that accept more than 0.5 mL of contrast dye typically allow extravasation of dye from the posterolateral annular regions [48]. From these data, it is apparent that intact cervical intervertebral discs hold less than 0.5 mL of contrast dye at which point in time a firm endpoint is encountered [24,25,29].

Injection of contrast medium within the cervical intervertebral disc nucleus reveals the integrity of the nuclear-annular interface. This is known as a nucleogram. Similar to their lumbar counterparts, cervical nucleograms may be viewed in a variety of configurations, including spherical, disc-shaped, or lobular patterns (Figure 31.6). Extension of contrast material beyond the nuclear region into the annulus denotes annular disruption. However, escape of contrast material from the nucleus into the uncinate recesses can occur in the aged, but asymptomatic, disc. This occurrence may reflect maturation of the disc's internal architecture [29,48,49]. Disruption of the annular fibers permits extension of nuclear material into the outer third of the annulus sensitizing annular nociceptive nerve fibers [50] producing pain. A cervical intervertebral disc can be determined to be the only source of chronic neck pain if it produces the patient's symptoms while demonstrating annular disruption in the axial plane. Hence, postdiscography computed tomography (CT) must be performed to analyze the nucleogram in the axial plane.

Transverse imaging of the cervical intervertebral disc can be difficult due to the small volume of contrast injected and the sparse nuclear dispersal pattern [29]. High-resolution, thin-section CT is utilized to capture postdiscography nuclear detail not revealed by MRI or CT alone [29]. Sections are acquired at 1.5 mm slices at a gantry angle parallel to each intervertebral disc space [29].

Despite the early contention challenging cervical discography's diagnostic value [28,51], cervical discography has become a useful diagnostic tool to help guide further therapeutic intervention [13,31–35]. However, as with any diagnostic test, the pretest probability influences the test results. Errors must be minimized to improve the test's accuracy. The usefulness of cervical discography rests in its ability to determine which intervertebral disc is responsible for the patient's symptoms. Discography represents a functional diagnostic test because the patient's subjective response is integral in the outcome of the test. Conversely, visual anatomic testing such as imaging evaluations can depict diagnostic information regardless of the patient's report of symptomatology.

Discography requires the diagnostician to apply clinical significance to structural abnormalities revealed by nucleogram patterns. The patient's response to stimulation of the intervertebral disc is noted as the contrast medium is injected under lateral fluoroscopic monitoring. Typically the patient is instructed to inform the examiner whether he or she experiences any cervical pain or pressure as the contrast solution is injected. The sensation of pressure only does not indicate a symptomatic intervertebral disc. If the patient complains of pain, the examiner immediately identifies, via specific questioning, the precise location of the pain, its quality, and its severity on a scale of 0 to 10. The patient is then asked to verify if this is the exact pain in location and character and confirm that the usual

symptomatology is being provoked. Furthermore, it must also be determined whether or not the disc stimulation produced all of the patient's usual pain or just a portion of it. During the procedure for each level interrogated, be sure to record the pain location, its character, severity, and whether it was concordant or partially concordant with the patient's typical symptoms.

While monitoring the patient's response, it is also imperative to evaluate the nuclear pattern as the contrast dye is injected. In addition to the aforementioned criteria, the nulceogram pattern, the volume of dye injected to reach this pattern, and the end-point resistance are recorded at each segmental level. If the dye reaches the outer annular fibers concurrently with the patient's report of concordant pain and the severity of this pain is rated at least 7/10 or higher by the patient, that segmental disc is most likely the source of the patient's neck pain. However, the spread of the contrast pattern must also be assessed in the axial plane by postdiscography CT. Extension of the contrast to the outer annular fibers or beyond into the epidural space indicates a structurally incompetent disc and provides evidence of internal derangement responsible for the concordant pain described by the patient. Partially concordant pain would implicate that segmental level as responsible for a portion but not all the patient's symptoms. Alternatively, it may also indicate that every portion of the internal derangement was not adequately stimulated to provoke every aspect of the pain location [52]. The need to systematically and meticulously collect specific and precise information from the patient during the intervention requires that the patient remain awake and cognizant to respond to our inquiries. Therefore, sedation should be avoided unless absolutely necessary. In our experience, however, performance of provocation cervical discography without sedation is tolerable by patients undergoing the examination.

The physiologic status of the cervical intervertebral disc can also be assessed to confirm the nucleogram. The hydrodynamic biomechanics of the disc will most often corroborate what the clinician is witnessing under lateral fluoroscopy. If the cervical disc tolerates more than 0.5 mL of contrast dye, annular disruption is likely present [47]. The endpoint will indicate whether the outer annular fibers are completely disrupted. In the scenario of complete annular dysfunction, the discographer will likely observe morphologic evidence of this dysfunction as dye escapes posteriorly into the anterior epidural space. An endpoint may not be encountered if contrast volumes of 1.0 to 1.5 mL are infused. Alternatively, a herniated or degenerate cervical disc that still contains intact outer annular fibers may yield a soft yet still definable endpoint upon injection of 0.5 to 1.0 mL of contrast without evidence of epidural spread. Therefore, it is imperative that each of these data points is meticulously and correctly collected and quickly audited to assign clinical meaning to the preliminary outcome of the diagnostic intradiscal procedure.

Examination of the nuclear contrast pattern in the axial plane using postdiscography CT will validate extension of contrast material into circumferential or radial annular tears. This may not be fully appreciated during fluoroscopy. Contrast material can reside within the uncinate recesses of the posterolateral regions of the cervical intervertebral disc. On posterioanterior fluoroscopic view, this pattern appears bulbous, while on corresponding lateral views this dye pattern appears to indicate a posterior protrusion [29]. However, postdiscography CT would reveal contrast within the nucleus and uncinate recesses only. The apparent protrusion on the lateral fluoroscopic view was produced by extension of contrast material into the posterolaterally oriented uncinate recesses [29]. Observation of a relatively firm endpoint at a volume at or near 0.5 mL should suggest to the discographer the fallacy of the seemingly abnormal dye pattern on the lateral fluoroscopic view.

Intervertebral discs that do not demonstrate encroachment of the outer annular fibers by contrast material have been noted to seemingly produce cervical pain during discography. In these instances, a mildly degenerate disc with a relatively firm endpoint at close to 0.5 mL of contrast dye may be associated with partially concordant neck pain. The adjacent, caudal disc will likely reveal annular disruption and concordant symptomatology. When this scenario is encountered, we will subsequently anesthetize the painful disc that demonstrated the abnormal nucleogram with 0.5 mL of 2% xylocaine and repeat the stimulation of the cranial level after 5 minutes of time have elapsed. If no pain is reproduced upon repeat stimulation of the normal nucleogram disc after anesthetization of the abnormal level, the initially painful response of the more cranial level is classified as a false-positive response. This occurrence can theoretically be explained by a pressure-transduction phenomenon, but this postulate has not been further evaluated in a systematic manner.

Therefore, a positive level is defined as an intervertebral disc that produces severe, concordant or partially concordant neck/shoulder girdle/head pain upon injection of contrast material that reaches the outer annular fibers, at the time of pain provocation, as demonstrated during fluoroscopy and confirmed by axial postdiscography CT (see Table 31.1). Additionally, an adjacent control

Table 31.1 Diagnostic Determinants of a Positive Disc Level in Cervical Discography

Results	Concordancy of Provoked Pain	Severity of Provoked Pain	Volume Injected	Nucleogram	Endpoint	Negative Adjacent Control Disc
Positive	Concordant/partially concordant	>6/10 (7 or greater)	Typically > 0.5 mL	Fissured	None (can be soft with circumferential tear)	Yes
Negative	Nonconcordant	<7/10 (6 or less)	Typically no greater than 0.4–0.8 mL	Normal, can show diffuse spread → degeneration	Firm (can be soft with disc extrusion)	No

level must not produce pain or produce only nonconcordant pain upon stimulation. If the discogram reveals one level or two contiguous levels producing concordant pain, then the patient may be a surgical candidate provided conservative care has failed. If three or more levels are concordant, or two concordant levels are noncontiguous, or any concordantly painful discs are lobular, then the patient requires a comprehensive chronic pain modulation program.

COMPLICATIONS

Discitis, subdural empyema, spinal cord injury, vascular injury, and prevertebral abscess have all been reported as complications of cervical provocation discography [32,41,53–57]. Infection of the disc space is recognized as the most common complication associated with diagnostic cervical discography [32,42,54,56,57]. A variety of causes have been identified. The causes postulated include inadequate skin preparation [57], needle contamination [41], and contamination from esophageal contents due to improper needle placement [25]. Epidural, subdural, or retropharyngeal abscess may appear as sequelae of disc space infection or as the primary source of infection after penetration of the esophagus or hypopharynx [55]. Yet, the incidence of discitis per patient remains extremely low ranging from 0.1% to 1% with a per disc incidence of 0.15% to 0.2% [32,42,54,56,57]. The main risk factors for infectious complications appear to include beards, thick or short necks, and male gender [42]. Preprocedure prophylactic antibiotics are not guaranteed to prevent infectious complications after cervical discography [42]. Intradiscal antibiotics are most likely sufficient to protect against disc space infections related to discography and may obviate the need for systemic antibiotic prophylaxis [58].

Spinal cord compression and myelopathy are rarely seen complications of cervical discography [41]. However, these ill fortunes have occurred in a cervical spine with significant preprocedure cord compromise. Although potential intervertebral disc injury from nuclear distension has been suggested [59], possibly explaining cord involvement, there have been no subsequent experimental studies to corroborate such speculation. The overall incidence of significant complications associated with cervical provocation discography in the largest published report has been observed to be 0.6% per patient all of which were infectious [42]. We must emphasize that these low rates of serious complications related to cervical discography can only be expected in the hands of experienced and well-trained interventional spine specialists.

UTILITY OF CERVICAL PROVOCATION DISCOGRAPHY

The only viable surgical treatment for CIDD is fusion [60–62], which can be accomplished by anterior cervical discectomy and fusion or by posterior fusion. The rationale for surgery is that by fusing the bony vertebral elements, motion is eliminated, thereby reducing discogenic pain. The utility of provocation discography to determine the level(s) to fuse is controversial. Some studies have reported "good or excellent" results in 70% to 96% of patients after cervical fusion of levels determined by discography [13,32,35,63]. Siebenrock [35] performed a retrospective review of 27 patients who underwent cervical provocation discography, which led to anterior cervical fusion at 39 disc levels. Twenty-two were single-level fusions, seven were two-level fusions, and one was a three-level fusion. Overall 19 patients (73%) had good to excellent results. Six patients (23%) had a fair outcome and one (3.8%) a poor result. This study demonstrated a pain reduction of more than 75% (fair to excellent results) in 96% of the 27 patients. More good to excellent results (85.7%) were seen after two-level fusions than after one-level fusions (61.9%). Patients presenting with pain referral to the upper limbs had a more favorable outcome. According to Garvey et al. [33], 82% of 87 patients reported their self-perceived outcome as good, very good, or excellent at a mean of 4.4 years after fusion. Ninety-three percent of these patients reported more than 50% reduction in their pain rating after surgery. Interestingly, a statistically significant difference was found for patients who were treated based on a truly positive discogram.

As discussed previously in this chapter, Shah et al. [36] performed an extensive systematic review of the literature in 2005, evaluating the diagnostic accuracy of discography in the management of chronic spinal pain. Studies were scored according to the Agency for Healthcare Research and Quality (AHRQ) and Quality Assessment of Diagnostic Accuracy (QUADAS) rating scales for diagnostic testing. For inclusion, studies first had to meet at least 50% of the total possible points for each scale. The authors concluded in their review of the evidence through November 2006 that there is level II-2 (moderate) evidence supporting the role of discography in identifying a subset of patients with cervical discogenic pain. The authors defined "moderate" evidence as evidence obtained from comparative studies with nonrandomized concurrent or historical controls, single-arm trials or interrupted time series without a parallel control group. In 2007, Buenaventura et al. [37] completed another systematic review that also evaluated the diagnostic accuracy of discography with respect to chronic spinal pain. Selected studies were then subjected to two rating instruments for diagnostic accuracy studies (AHRQ and QUADAS). Evidence was classified into five levels: (1) conclusive, (2) strong, (3) moderate, (4) limited, or (5) indeterminate. The authors concluded that evidence is strong for the diagnostic accuracy of discography as an imaging tool and supported Shah's prior conclusion that there is moderate evidence supporting the role of discography in identifying a subset of patients with cervical discogenic pain. The authors concluded that discography is a useful imaging and neck pain evaluation tool in identifying a subset of patients with chronic spinal pain secondary to intervertebral disc disorders. Unfortunately, there are no well-designed, prospective, randomized clinical

controlled trials assessing cervical discography and its utility in predicting excellent surgical outcomes.

CONCLUSION

Diagnostic cervical provocation discography is a useful diagnostic intervention to aid the spine care specialist in determining how best to treat chronic cervical and shoulder girdle pain. By following strict guidelines such as identifying control disc levels, monitoring for a "concordant" pain response with a Visual Analog Scale score > 6/10, using nonionic contrast agents, and observing visual evidence of annular disruption on nucleogram and post-discography CT scan, cervical discography has low false-positive rates and is a reliable diagnostic tool. Despite controversy surrounding the utility of cervical discography, when performed correctly and meticulously under fluoroscopic guidance by experienced physicians with a knowledge of disc biomechanics and pathology, cervical discography poses minimal risk of significant complications and is a valuable adjunct in determining which segmental levels to surgically fuse.

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32 Percutaneous Treatments for Painful Cervical Discs

Steven R. M. Helper

INTRODUCTION

Chronic neck pain is common, affecting 14% of the general population [1]. The prevalence of axial neck pain originating from derangement of the cervical intervertebral discs ranges from 16% to 20% [2,3]. Treatment has traditionally been limited to either conservative medical management or open surgery. In properly selected patients [4,5], surgery, in the form of anterior interbody fusion (± anterior plating), has yielded positive clinical results in more than 70% of patients [6–13] but has the potential for long-term deterioration of surgical outcomes [14–18]. Meanwhile, cervical disc arthroplasty has primarily been studied in the setting of cervical radiculopathy and/or myelopathy [17,19–21]. Its use in the management of axial pain presumed secondary to disc degeneration is unknown. Given the prevalence of this problem and the limited treatment options, the development of alternative treatment methods is the logical advancement of care. Minimally invasive, fluoroscopically guided percutaneous procedures have emerged as a potential step in the treatment algorithm for chronic cervical discogenic pain.

Traditionally, transforaminal cervical epidural corticosteroid injections (TFCESI) have been utilized as a second treatment step, following failed conservative management. Intradiscal modulation technologies to consider, as a potential third step in the treatment algorithm of discogenic neck pain, include intradiscal radiofrequency (RF), intradiscal electrothermal therapy, percutaneous laser disc decompression (PLDD), and percutaneous plasma disc decompression (Nucleoplasty/coblation). No single approach has proven itself to be a valid and efficacious minimally invasive solution to axial neck pain of discography confirmed [22], discogenic origin [23]. The purpose of this chapter will be to discuss the feasibility of intradiscal modulation as a safe and effective treatment step in this patient population.

PATHOPHYSIOLOGY

The anatomy and physiology of the lumbar intervertebral discs have been studied extensively, and several theories have emerged regarding the pathology of mechanical and biochemical disorders of the lumbar disc [24–28]. In discussing cervical intervertebral discs, most conclusions evolve from the extrapolation of facts from the lumbar spine literature [29]. Consequently, the appropriateness of the term *cervical internal disc disruption syndrome* to describe the clinical scenario of axial discogenic neck pain, although supported, needs confirmation.

Internal disc disruption syndrome was coined by Crock [30,31] to identify the syndrome of low back pain and nonradicular referred pain in the setting of degenerative disc disease, and has evolved to encompass the entity marked by radial and circumferential tears in the annulus fibrosus associated with back greater than leg pain, radiation in a somatic referral pattern, and no focal neurologic deficit [32,33].This has been confirmed by studies showing that reproduction of discogenic low back pain is associated with tears which extend to the outer region of the annulus fibrosus [34–36]. The foundation for the development of intradiscal procedures targeting internal disc disruption has been based on the underlying histologic changes that are the hallmark of lumbar spine disease. Thus far, the development intradiscal technology in the treatment of cervical discogenic pain has largely been based upon the lumbar model. The known differences in anatomy and pathophysiology, between the cervical and lumbar discs, must be considered in the development of any intradiscal modulation procedure for the treatment of cervical discogenic neck pain [29,37–41].

Anatomy

Annulus Fibrosus

In the lumbar spine, the annulus pulposus consists of individual lamellae derived primarily of collagen type I fibers passing obliquely between vertebral bodies, with orientation of the fibers being reversed in successive lamellae. The lamellae are thicker toward the centre of the disc [42]. They are also thicker in the anterior and lateral aspect of the disc, becoming finer and tightly packed posteriorly [43]. The cervical annulus fibrosus does not consist of concentric laminae of collagen fibers with alternating obliquity. Rather, the anterior annular fibers are interwoven forming a crescentic mass of collagen thick anteriorly, which taper laterally toward the uncinate processes [41]. The annulus is essentially deficient posterolaterally. From the uncovertebral region, clefts extend to the fibrocartilaginous core

[42]. With age these clefts partially transect the fibrocartilaginous core, leaving only a posterior plug with a thin layer of overlying posterior annulus [41,44–46]. Posteriorly, a thin layer of paramedian, vertically orientated fibers, represents the annulus. The anterior longitudinal ligament covers the front of the disc, and the posterior longitudinal ligament reinforces the deficient posterior and posterolateral annulus fibrosus [41].

Nucleus Pulposus

The strength of the lumbar disc is related to the fluid and proteoglycan content of the disc. The nucleus pulposus consists of a hydrophilic proteoglycan (aggrecan) [47–49] and water gel held together loosely by an irregular network of fine collagen type II and elastin fibers. In the healthy lumbar disc, the strong osmotic properties and resultant high water content of the nucleus and inner annulus enable the tissue to act like a fluid under high pressures. Only the outermost annulus acts as a tensile restraint to the nucleus.

In the cervical spine, the nucleus pulposus is also derived from notochord [l], but this seems to be where the similarities between the cervical and lumbar discs end. At birth the nucleus makes up less than 25% of the cervical disc volume versus 50% in the lumbar spine [50]. The gelatinous nucleus begins to disappear in adolescents [37,51], and is replaced by a fibrocartilaginous core that has the consistency of a bar of soap [29,37,51]. The cervical intervertebral discs are not responsible for the same relative mechanical load as their lumbar cousins. The joints of Luschka [52] and the zygapophyseal joints [46,53] share a significant portion of the weight-bearing load of each cervical segment.

Innervation

The lumbar intervertebral disc is an innervated structure that is capable of pain generation [54–57]. The greatest number of nerve endings is found in the lateral aspect of the disc, with a slightly smaller number found in the posterior region and even less so anteriorly [43,58]. Innervation of the cervical intervertebral discs has been shown to be similar to that found in lumbar discs. The cervical sinuvertebral (SVN) nerves arise as a recurrent branch of the ventral ramus [54,59]. The SVN also receives an autonomic contribution from the vertebral nerve (sympathetic plexus) [54,59]. The SVN enters the vertebral canal to supply the disc at their level of entry as well as the more cranial disc [59,60]. Vertebral nerves contribute to the innervation of the lateral aspect of the disc [59]. Small free nerve endings and their parent nerve fibers are found throughout the annulus fibrosus [59,60]. Similar to the lumbar spine [58], study of the neural elements of the cervical intervertebral discs found most of the nerve receptors in the posterolateral region of the annulus fibrosus [60], with the highest nerve density in the middle third of the disc. The nonencapsulated nerve fibers, again found in the superficial layers of the annulus fibrosus, are hypothesized to be the substrate for primary cervical disc pain and for the pain response of provocation cervical discography [59].

Pathophysiology of "Disc" Pain

Biomechanical Insult

Much of the pathophysiology of cervical disc pain has been inferred from lumbar studies [61]. The biomechanical theory of discogenic pain relates to the proprioceptive nerve endings in the outer annulus. As the lumbar spine ages, the natural history of the degenerating disc includes the loss of disc water content and, subsequently, nuclear hydrostatic pressure. As a result, most of the annulus then acts like a fibrous solid to resist compression directly. With continued stress, this leads to buckling of the annular lamellae. Increased focal segment mobility and increased shear stress lead to microscopic fractures of collagen fibrils and subsequently delamination and fissuring of the annular wall [62]. Generally, small mechanoreceptors are limited to the intact portions of the annulus, which have been shown to discharge with disc mobilization [63]. When neighboring annular fibers are destroyed, intact fibers are forced to compensate for the tensile loading not accommodated by the disrupted portion. The magnitude of stress transmitted to the intact fibers is proportional to the degree disruption [43]. With severe degeneration these mechanoreceptors become overloaded. An abnormal nociceptive afferent sensory message is relayed through the dorsal root ganglion. Repetitive stimulation of the dorsal root ganglion has been shown to create prolonged neural activity within the dorsal horn receptor fields [64–66]. As the disc is repetitively loaded, a cycle of painful signal transmission and heightened dorsal horn receptor field activity continues.

Delamination is, by definition, not part of the degradative process in the cervical spine [41]. However, fissuring of the annular wall, deep to the joints of Luschka, is thought to be an initial stage in the development of annular clefts [52]. Extension of horizontal clefts and the formation of vertical clefts may develop as a result of shearing stress to the disc by translational movement [52,67]. Some of the vertical clefts extend to the degenerated cartilaginous endplate which may tear off as part of a disc herniation [52]. Although no definite conclusion has been made as to relevance of these clefts to cervical discogenic neck pain, we do know this: The cervical disc is supplied with nerve fibers of various nerve sizes and small encapsulated fibers [60], similar to the lumbar spine [54]. The innervation is in greatest supply in the posterolateral regions of the annulus fibrosus [60]. In theory, it is subject to the same biomechanical overload hypothesized for the lumbar spine. Secondarily, the cervical disc is also supplied with nonencapsulated nerve fibers, which may have a separate nociceptive role [60].

Biochemical Insult

From lumbar data, we know a variety of inflammatory tissue reactions develop within degenerative discs [68,69]. Immunologic studies demonstrate the presence of phospholipase A2 [70], nitrous oxide (NO) [71,72], interleukin 1 [72], and metalloproteinase enzyme activity within the

degenerative disc [73]. Chemical nociception occurs when nerve endings in the annulus fibrosus become exposed to the cytokines, neuropeptides, and protein degeneration products of the disc [74–76]. In an in vitro study of the cervical spine, Kang et al. [77] demonstrated NO production in cultures of herniated discs that was significantly higher than in the control discs. In an in vivo study, Furusawa et al. [61] were able to accurately measure the amount of NO in cervical discs. Their results showed high levels of NO in both uncontained and contained cervical disc herniations. The exact role of NO in intervertebral disc metabolism is unknown, but it may play an important role in the degeneration of the intervertebral disc. Another inflammatory mediator, interleukin-1beta, is thought to sensitize annulus cells to mechanical load [78]. This increased responsiveness to mechanical load in the face of inflammatory cytokines may imply that the sensitivity of annulus cells to shear increases during inflammation and may affect initiation and progression of disc degeneration.

In association with this immunoreactivity, studies have shown nerve ingrowth into the inner third of the annulus and toward the nucleus of painful, disrupted lumbar discs [79,80]. This neoneurolization has been directly tied to the increased presence of certain neuropeptides and cytokines such as substance P (SP) [79]. In addition, nerve fibers may contain nociceptive neurotransmitters in addition to SP, including calcitonin-gene-related peptide, and vasoactive intestinal peptide [81–84]. Furthermore, cytokine release may be further triggered by mechanical disc stimulation [28,85].

The final piece of the biochemical model involves the concept of neovascularization accompanying neural ingrowth into the inner annular layers [75]. Peng et al. [75] demonstrated that the distinct histologic characteristic of the painful lumbar disc was the formation of a zone of vascularized granulation tissue from the nucleus pulposus to the outer part of the annulus fibrosus along the edges of the fissures. Growth of SP, neurofilament, and vasoactive intestinal peptide-immunoreactive nerve fibers deep into the annulus fibrosus and nucleus pulposus was observed mainly along the zone of granulation tissue in clinically painful discs. This suggests that the zone of granulation tissue, in association with extensive innervation along fissures in the posterior part of the painful disc, is responsible for causing discogenic low back pain.

Although studies in the cervical spine lag behind, extrapolation from animal and lumbar models lends a logical connection between nerve ingrowth and painful intervertebral discs. In an immunohistochemical examination of the cervical spine, Furusawa et al. [61] demonstrated a rich network of small blood vessels in 27 (75%) of 36 herniated cervical discs. Hypothesis suggests that inhibition of growth of nerve fibrils into the intervertebral disc or interference with nociception mediated by these nerves may benefit patients with chronic discogenic neck pain.

EPIDURAL CORTICOSTEROID INSTILLATION

Transforaminal cervical epidural steroid injections (TFCESI) have been the subject of much discussion and even controversy in recent literature [86–93]. Despite ongoing concerns regarding their safety, TFCESI are currently used, with apparent success, in the interventional management of cervical radicular pain syndromes [94–101]. To a lesser extent, TFCESI are used by interventionalists in the management of axial neck pain of discogenic origin [102].

Treatment options for chronic axial neck pain should address the pathophysiology of the injured structure. Evidence suggests an increased production of proinflammatory mediators and cytokines with disc degradation [61,68–77]. Injection of corticosteroids into the anterior epidural space has long been used to bathe the posterolateral periphery of the annulus with discogenic low back pain to help curtail the biochemical stimulation of the intervertebral disc [103]. The main goals of this approach are to improve pain and function and allow the patient to participate in a comprehensive physical therapy program addressing biomechanical deficiencies after this reduction of hyperalgesia. This anecdotal success has led some practitioners to use TFCESI to achieve the same clinical goals in patients with axial, discogenic neck pain.

The instillation of corticosteroid and anesthetic into the anterior epidural space introduces therapeutic agents with potent anti-inflammatory properties adjacent to suspected painful intervertebral discs. Local anesthetics help curtail inflammation by inhibiting phagocytosis, decreasing phagocytic oxygen consumption, reducing polymorphonuclear leukocyte lysosomal enzyme release, and diminishing superoxide anion production [104–107]. In addition, anesthetics improve neural blood flow and dysfunction [108,109]. Corticosteroids are well known for their anti-inflammatory properties [110], and also stabilize neural membranes, suppress ectopic neural discharges [111], and may have direct anesthetic effect on small unmyelinated nociceptive C-fibers [112,113]. Painful cervical intervertebral discs are innervated by unmyelinated C-fibers, and thinly myelinated A-delta fibers [59,60] that provide a potential substrate for corticosteroids and local anesthetics to act upon. Hence, corticosteroids and local anesthetics may exert a therapeutic benefit by bathing the posterolateral annular fibers, the initial location of fissuring and annular cleft development [52], in solutions with anti-inflammatory and neural stabilizing effects.

Indications

The primary indication for TFCESIs is cervical radicular pain or radiculopathy [114]. Despite minimal work having been completed investigating the efficacy of these interventions solely for axial cervical spine pain [115], such injections are often offered to patients presenting with nonradicular central neck pain. However, the role of these injections to treat axial neck pain of discogenic origin has not been well defined and is currently supported largely by conjecture and logic. Deciding which level to inject is influenced by imaging findings [116] (see Chapter 30) and pain referral zones [117,118] (see Chapter 29) and by initially targeting the levels most likely to be responsible for discogenic neck pain (C5–6) [117]. If the patient experiences only short-term or no improvement with a C6 TFCESI, awake provocation cervical discography (see Chapter 31) would

be warranted to determine if the suspected disc(s) is/are painful.

Procedure

The instillation of therapeutic doses of corticosteroid into the anterior epidural space to maximally reach the targeted intervertebral disc is best accomplished by a transforaminal approach (TFCESI) rather than with interlaminar epidural steroid instillation (ILCESI). The TFESI approach offers the potential advantage of delivering medication directly into the anterior epidural region, hence closer to the putatively painful structure [119]. The addition of fluoroscopy and contrast enhancement allowed the documentation of whether or not the medication reached the potential pain generator, maximizing the chance of therapeutic benefit. Interlaminar steroid injections have limitations because of pathway hindrance and medication dilution, as well as needle misplacement. With ILCESI, the injected agent is deposited into the posterior epidural space without a guarantee that it will flow anteriorly [120]. ILCESI achieve ventral epidural contrast spread in just 28% of attempts [120]. In contrast, TFCESI achieve ventral flow in 100% of injections [119]. With ILCESI, the existence of epidural fibrosis, spinal stenosis, or postsurgical scarring may prevent the steroid medication from diffusing ventrally. It will take the path of least resistance and may spread caudally. Even if the injectate reaches its intended target, the medication will have diffused to the posterior disc space, and will be diluted.

Technique

It is now standard that spine procedures be performed in a room equipped with oxygen, suction, and resuscitative equipment to manage airway patency, blood pressure, and cardiac rhythm. The operator must be advanced cardiac life support trained and have the ability to recognize and appropriately treat complications that may arise during the procedure.

The patient lies in a supine, oblique, or lateral decubitus position depending on operator preference and patient comfort. The priority is adequate visualization of the cervical intervertebral foramina in multiple planes. The skin over the anterior and lateral neck is cleansed and draped in the usual sterile fashion.

The fluoroscope is initially adjusted to visualize the cervical spine in the oblique projection, focusing attention on a clear view of the target intervertebral foramen. The anterior surface of the superior articular process (SAP) is the initial target. Select a puncture point that allows the most lateral view possible to access the cervical neuroforamina-contact posterior lateral projection of SAP [121]. The use of small procedure needles (25-gauge or 22-gauge, short beveled, 2 to 3.5 inches) makes subcutaneous anesthesia unnecessary in most instances (Figure 32.1).

Following skin puncture, the primary objective is to safely place the needle tip on to the bony surface of the SAP (Figure 32.2). This is best achieved by establishing an optimal initial puncture site, in which the target/bulls-eye

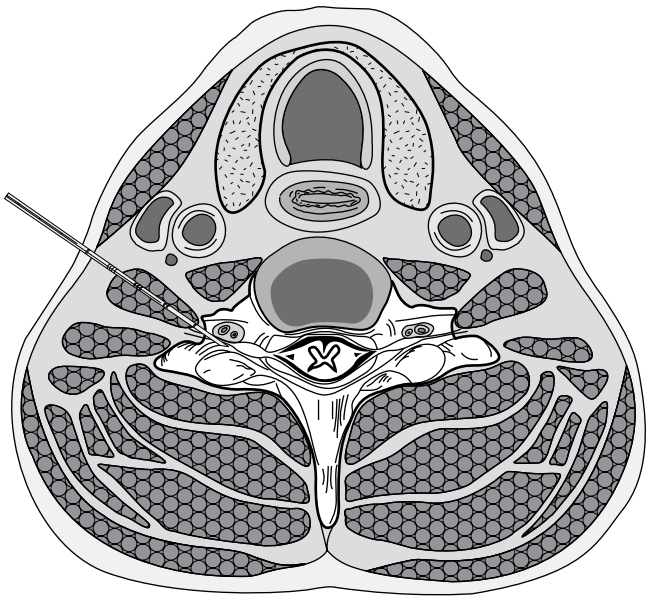


Figure 32.1 Cross-section of the cervical spine demonstrating optimal placement of a 25-gauge, bevel-tipped spinal needle in the posterior neural foramen.

view of the spinal needle is located directly over the SAP. The course of the needle should be monitored with repeat fluoroscopic screening. The needle is advanced in multiple small increments. Once the needle has reached the SAP, it may be retracted slightly and then advanced over the anterior surface of the SAP, into the intervertebral foramen tangential to its posterior wall, across its middle third. The needle insertion should not be advanced more than a 1 to 2 mm past the bony margin.

Next, an anteroposterior (AP) view of the cervical spine is necessary to check the medial depth of the needle tip. Ideally, the tip of the needle should lie opposite the sagittal midline of the articular pillars. A greater medial depth risks dural puncture, whereas a lateral position may result in inadequate epidural flow of medication. Once the final position has been achieved, the oblique and AP images should be verified once more and spot films obtained in both planes (Figure 32.2 and 32.3).

For the injection of agents, a short length of low volume (0.4 mL capacity) tubing is recommended. After attachment of the tubing to the needle hub, repeat images should be taken in the oblique and lateral views to rule out needle migration. Under real-time fluoroscopy, in the oblique view, a small volume of nonionic contrast material is instilled. A clear radiculo-epidurogram should be observed with spread centrally toward the epidural space (Figure 32.4, 32.5 and 32.6). To rule out undetected arterial injection, this author uses digital subtraction angiography for secondary confirmation of the safety of the final needle position. Even if the needle is correctly placed, the injection may be into a radicular artery. Intra-arterial injection is manifest by very rapid clearance of the injected contrast material. In the case of arterial puncture, the procedure should be aborted. Despite the low likelihood of any material subsequently injected inadvertently penetrating the

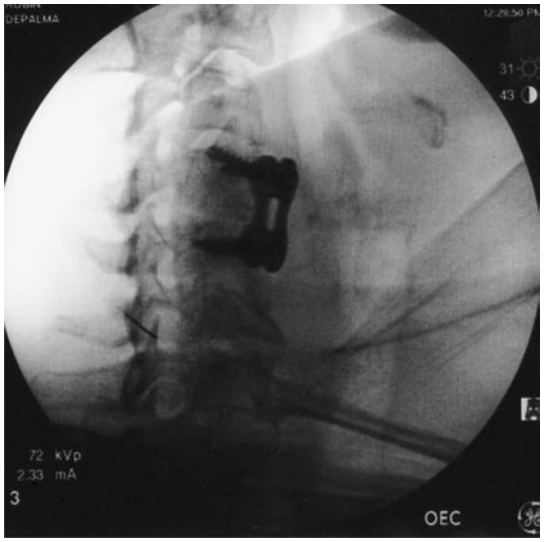


Figure 32.2 Oblique view of the cervical spine demonstrating proper needle position for a right C7 TFCESI. The tip of spinal needle rests at the anterior margin of the C7 superior articular process, in the posterior aspect of the C6–7 neural foramen.

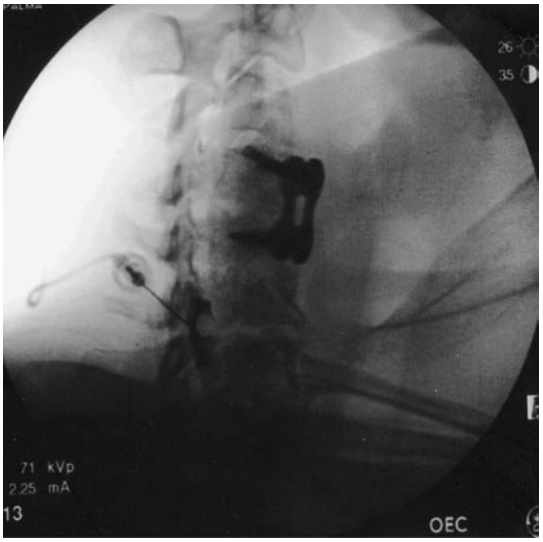


Figure 32.4 Oblique view of the cervical spine displaying flow of radiopaque contrast along the right C7 nerve root. Early posterior and anterior epidural flow of contrast is demonstrated.

punctured artery, it is prudent to avoid unnecessary risk and reschedule the TFCESI for a later date.

In the case of venous injection, the injection of contrast medium demonstrates slow clearance of the contrast material, characteristic of venous flow. If the interventionalist is confident of a venous pattern, the needle position adjusted slightly, by withdrawing by 2 mm and redirecting slightly cephalad or caudad to the original endpoint. All precautions taken in the original placement of the spinal needle must be carried out once more. Once the needle has been repositioned, the test injection of contrast medium should be repeated. If venous flow persists, the procedure should be aborted.

Rapid dilution of the contrast material implies subarachnoid spread. This may occur if the needle has been advanced too deeply and has punctured the thecal sac, or if there is a lateral dilatation of the dural root sleeve into the intervertebral foramen. In the event of suspected subarachnoid injection, the needle must be withdrawn and no injection of other solutions performed, until after a period of sufficient duration for the puncture to have healed [114].

Once the interventionalist is confident in the appropriate dispersal of the solution, spot films in the oblique and frontal planes are obtained. Next, injection of a small

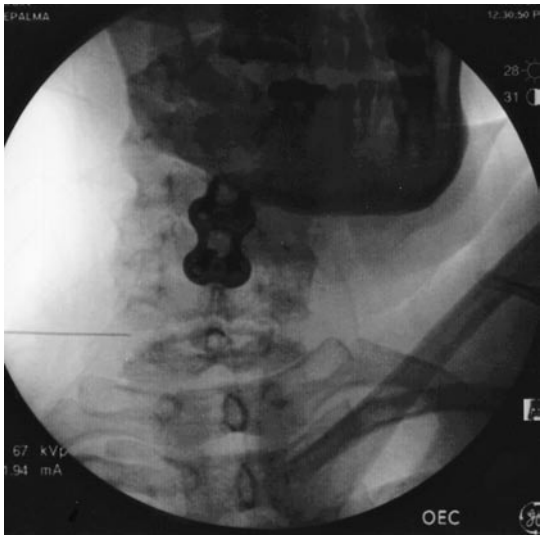


Figure 32.3 AP view of the same right C7 TFCESI procedure. The tip of the spinal needle rests slightly lateral to the sagittal midline of the articular pillars.

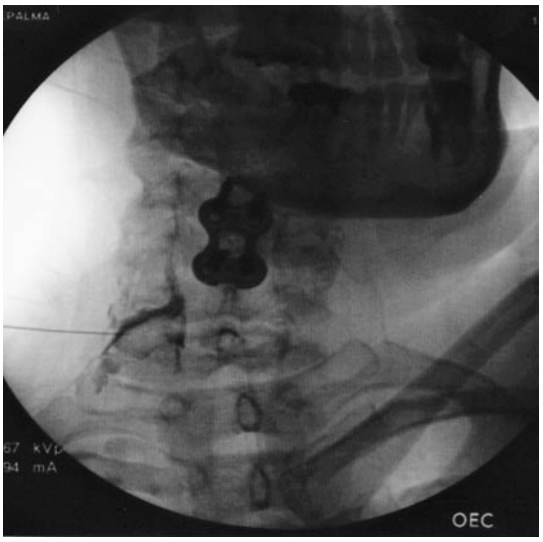


Figure 32.5 AP view of the same patient reveals a clear C7 radiculogram. Bilateral epidural flow of contrast is demonstrated from the C3–4 through C7-T1 intervertebral segments, confirming satisfactory medication dispersal.

volume of a short-acting local anesthetic is recommended. Typically, 0.5 mL to 1.5 mL of lidocaine, 1% or 2% without preservative, is used. The later dosage is used at a lower volume. This "test" dose represents yet another safety check point prior to instillation of corticosteroid medication. The patient may experience short-lived side effects as a result intravascular (perioral paresthesia, metallic taste in mouth, palpitations, altered sensorium) or intrathecal injection (transient quadraparesis), which had gone undetected at the time of contrast instillation.

After 2 minutes of monitoring, if the patient experiences no ill effects from the test dose of local anesthetic, one may proceed with corticosteroid instillation. Low particulate solutions are recommended [90]. This author favors the use of 20 to 30 mg of dexamethasone sodium phosphate (Decadron) because of its favorable low particulate matter profile [90,122]. Further monitoring should continue in the procedural suite for 2 minutes before transfer to recovery. Once transported to recovery, patients should be monitored for a minimum of 20 minutes before considering discharge.

Outcomes

The use of TFCESI in the management of axial neck pain has never been directly studied. Ferrante et al. performed a retrospective analysis on 100 patients who had received cervical epidural steroid injections for neck pain and cervical radiculopathy to identify the predictors of outcome after such treatment [115]. In their study, patients with radicular symptoms and signs achieved the best pain relief as opposed to those with solely axial neck pain. Minimal conclusions may be drawn from this study. Aside from the inherent limitations of the study design, the procedural technique was suboptimal. The author utilized a midline interlaminar approach with loss-of-resistance technique, without fluoroscopic guidance. It is unknown what percentage of injectate reached its target pathology, in each group. Ferrante et al. [115] addressed clinically important questions that remain to be clearly answered: is it possible to develop a clinical classification scheme to define the indications for TFCESI? Do patients with axial neck pain of discogenic origin represent a meaningful patient group, responsive to this intervention? Further research is necessary.

Complications

As with all injection procedures, transforaminal cervical epidural coricosteroid injections are subject to potential complications [123–125]. More recently, reports of catastrophic complications have received considerable attention [126–138]. These include cerebellar and cerebral infarct [133,135], spinal cord injury and infarction [132,138], massive cerebral edema [134], cortical blindness [128], and anterior spinal artery syndrome [126,130]. The potential for serious injury to the central nervous system has called into question the risk-benefit profile of this procedure, leading some interventionalists to update their technical approach or abandon the use of TFCESI altogether.

In a retrospective review of 1036 fluoroscopically guided transforaminal injections (of 844 patients), Ma et al. [124] reported an overall rate of complications of 1.64%. Following the author's methodology, placement of the 25G spinal needle was followed by injection of myelographic contrast material displaying a clear radiculogram/epidurogram. Digital subtraction angiography was not used. Once the needle was adequately positioned, Ma et al. instilled one of two local anesthetic/corticosteroid mixtures. They did not specify the number of patients exposed to a particulate solution (methylprednisolone acetate suspension—DepoMedrol) versus a corticosteroid solution containing particles of small size (betamethasone sodium phosphate and betamethasone acetate—Celestone Soluspan) [131]. They did document the final depth of the needle tip in both the AP and lateral fluoroscopic views.

No catastrophic complications such as death, paralysis, stroke, spinal cord injury, vertebral artery injury, or infection occurred [124]. Minor complications included headache (0.5%), transient neurologic (0.6%) deficits (pain or weakness), hypersensitivity reaction (0.1%), vasovagal reaction (0.1%), and transient global amnesia (0.1%). Chi-square tests were performed to determine whether there were any significant differences in the rate of complications associated with differences in needle placement. The rate of complications associated with anterior placement of the needle tip in the neural foramen was significantly higher than that associated with ideal or near-ideal placement ($P = 0.04$).

In a prospective nonrandomized controlled study of 151 patients undergoing either lumbar or cervical selective nerve root injections, Huston et al. reviewed the complications of fluoroscopically guided transforaminal injections [125]. Specifically, the authors looked at the complications of CSNRI of 89 injections in 37 patients with cervical radicular pain. At the time injection, cervical selective nerve root injections resulted in lightheadedness in 2.2% of patients, dural puncture in 1.1%, and nausea in 1.1%. In the immediate post-procedure interview, patients reported increased pain at injection site in 22.7% of cases; increased radicular pain in 18.2%; lightheadedness in 13.6%; increased spine pain in 9.1%; nonspecific headache in 4.5%; and nausea in 3.4% of cases. No serious complications were demonstrated acutely, at 1-week follow up, or 3-month follow up. The primary limitation of the study by Huston et al. is the lack of clarity as to what number of injections were "diagnostic" (0.5 mL of 1% lidocaine) and what number were "therapeutic" (1.0 mL of betamethasone mixed with 0.5 mL of 1% lidocaine). The leading theory as to the cause of serious neurologic sequelae (stroke, spinal cord injury) is the risk of corticosteroid particulate material being injected into a radicular artery [126], or vertebral artery [135], resulting in thrombosis or embolus [139].

Two surveys of physicians performing cervical epidural steroid injections demonstrate markedly different reporting of serious neurologic injury. Scanlon et al. recently conducted a survey of pain physicians inquiring about complications after TFCESI [137]. Of 287 respondents, 78 complications were reported, including 16 vertebrobasilar brain infarcts, 12 cervical spinal cord infarcts, and 2 combined brain and spinal cord infarcts.

The primary criticism of the study is the low response rate of 21.4%. However, when discussing absolute events, as opposed to incident rate, the data posted by Scanlon et al. [137] are significant and concerning. In a smaller survey by Derby et al. [140], 17 International Spinal Intervention Society instructors were asked to report their documented complications in the year 2003. The instructors described a total of 5978 cervical epidurals, interlaminar in 4389 patients, and transforaminal injections in 1579 patients. Of the interlaminar injections, there were 23 mild complications (0.5%), whereas there were five cases of minor complications in the transforaminal group (0.32%). No serious complications were reported. In the survey by Scanlon et al. [137], 11 serious complications were reported during the same time period (2003). Too many variables exist to allow final conclusions to be made from direct comparison of these two papers [137]. However, it serves as a necessary reminder that TFCESI should only be by performed by well-trained physicians, experienced in performing interventional spine procedures.

THERAPEUTIC INTRADISCAL PROCEDURES

Percutaneous Laser Disc Decompression

In 1986, Choy and Ascher first employed laser technology to decompress contained lumbar disc herniations in spine patients with radicular pain symptoms [141,142]. The US Food and Drug Administration approved PLDD in 1991. By 2002, some 35,000 PLDDs had been performed worldwide [143]. In 1990, Hellinger et al. [144] first used laser disc decompression in the cervical spine with the Neodymium:YAG Laser, for the reportedly successful management of monosegmental noncompressive radiculopathy. In 1991, Knight et al. began using KTP532 cervical laser disc decompression with broad-based soft disc protrusions in noncompressive radiculopathy with similar claims of success [145]. This same group then commenced Holmium:YAG cervical laser disc decompression in 1992 [145].

Percutaneous disc decompression procedures have traditionally been founded on the biomechanical principle that a small reduction of volume in a closed hydraulic space, like an intact disc, results in a disproportionately large decrease in pressure [141,146–148]. The thermal effects of laser also cause protein denaturation [149]. Because the laser energy is well absorbed in vascular tissues, it is hypothesized to result in greater absorption of energy in areas of annular neovascularization [145]. These areas are concurrently found to be the sites of neoneuralization [79], and the effects of lasing might effect concomitant denaturation of the annular neoneuralization with amelioration of pain [41]. In the cervical spine, the majority of nociceptive nerve fibers are found in the posterolateral region of the disc [59,60]. Depending on the penetration of the laser technology employed, these nerve fibers may lie directly in the radiation field of the laser fiber [145].

Early in the development of PLDD technology, various lasers were studied by Choy et al. [150,151]. Consensus is lacking on the type of laser used, the energy applied, or duration of application. The wavelength energy that is best absorbed by water should, theoretically, result in more discrete disc tissue ablation in areas of neovascularization, with limited tissue penetration and less tissue heating at equivalent distances from the probe [152]. Presently, there are three common lasers in use for cervical PLDD: (1) 1064-nm Nd:YAG, (2) KTP^{532}, and (3) the 2100-nm Holmium:YAG. Laser technologies vary in their emitted wavelength, with the greatest absorption occurring nearest to the absorption band of water (2000 nm) [153]. Low absorption may result in the vaporization of an insufficient amount of nucleus pulposus (NP), whereas high energy can increase risk of tissue burning. Potassium titanyl potassium:YAG (KTP^{532}) uses a side-firing fiber and is poorly absorbed by disc tissue and by vascular tissues. The 2100-nm Holmium:YAG wavelength energy is best absorbed by water and may result in more discrete disc tissue ablation [152–156]. However, the lack of scatter may lead to excess local heat accumulation, necessitating cooling by saline irrigation [143]. Comparable results between laser probes of different wavelengths with widely dissimilar tissue ablative and tissue penetrative effects suggest that efficacy may be not only related to the extent of tissue vaporization [145]. Instead, the clinical response may relate to factors such as tissue heating, collagen annealing [157], coagulation of neovascularization and inhibition of neoneuralization, and an effect upon the release of chemical irritants from within the disc. Previous investigations into the histologic effects of laser energy on collagen have demonstrated that the application of laser energy at non-ablative levels can alter collagen's structural and biochemical properties [158,159].

Procedure

Preoperatively, it is common practice for patients to receive intravenous antibiotics [145,160].The use of mild sedation is subject to the discretion of the operating physician. Care must be taken to maintain a level of alertness that easily allows patients to report unexpected discomfort. The patient is positioned in the typical position as per cervical provocative discography [114,161] (see Chapter 31). The patient is placed on the fluoroscopy table in the supine position, with the neck slightly extended. Rotating the head to the left will provide a greater working space while further moving the trachea and esophagus to the left. Strict aseptic technique must be maintained. The skin of the anterior and right lateral neck is sterilized and draped in the usual fashion. A right-sided approach is used for percutaneous cervical intradiscal procedures because the esophagus lies to the left in the lower neck. Some practitioners favor preoperative screening of the neck anatomy with ultrasound to rule out possible anatomic variants (thyroid, lymph nodes, superior and inferior thyroid and carotid arteries, recurrent laryngeal nerve) or pathologic conditions that could obscure the percutaneous surgical pathway and prevent a safe approach into the disc nucleus [162].

Once the level to be treated is identified in the AP view, the C-arm is tilted cephalad-caudad until the endplates at that level are parallel to the beam. Next, the C-arm is rotated cross-table, approximately 20º, to provide visualization of the anterolateral aspect of the disc space, while simultaneously rotating the trachea and esophagus

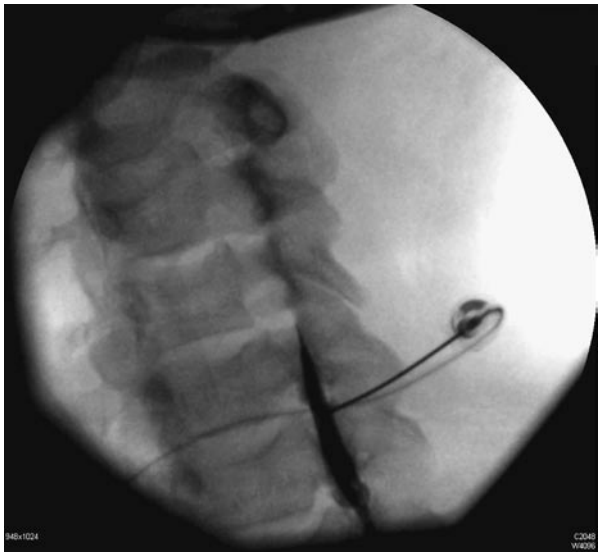


Figure 32.6 Oblique view of a left C6 transforaminal cervical epidural corticosteroid injection.

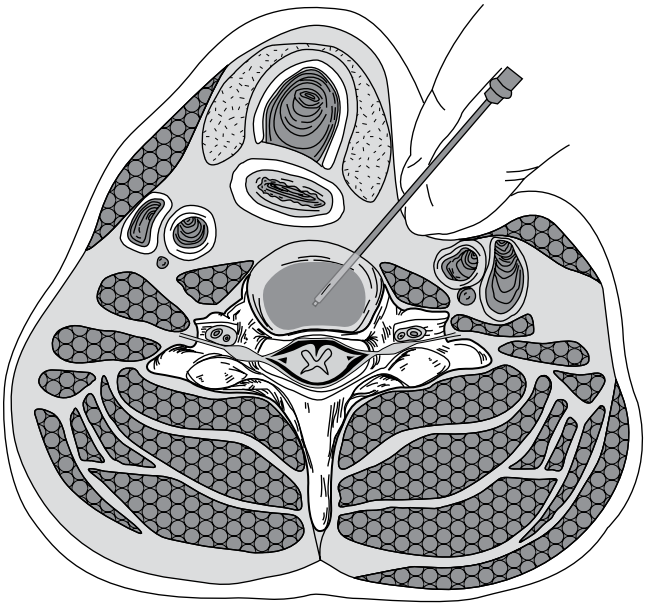
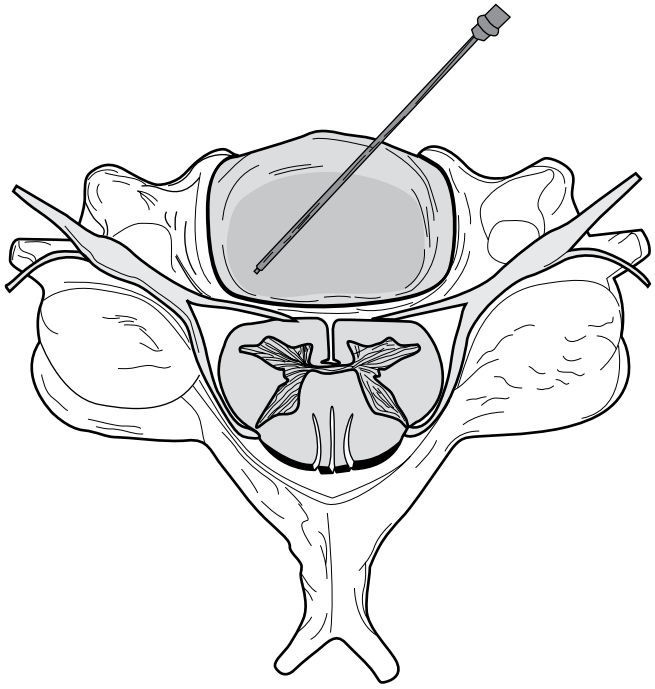


Figure 32.7 Cross-sectional view demonstrating proper hand position, prior to skin puncture. Note the displacement of the trachea and esophagus, medially, and the carotid artery and jugular vein, laterally.

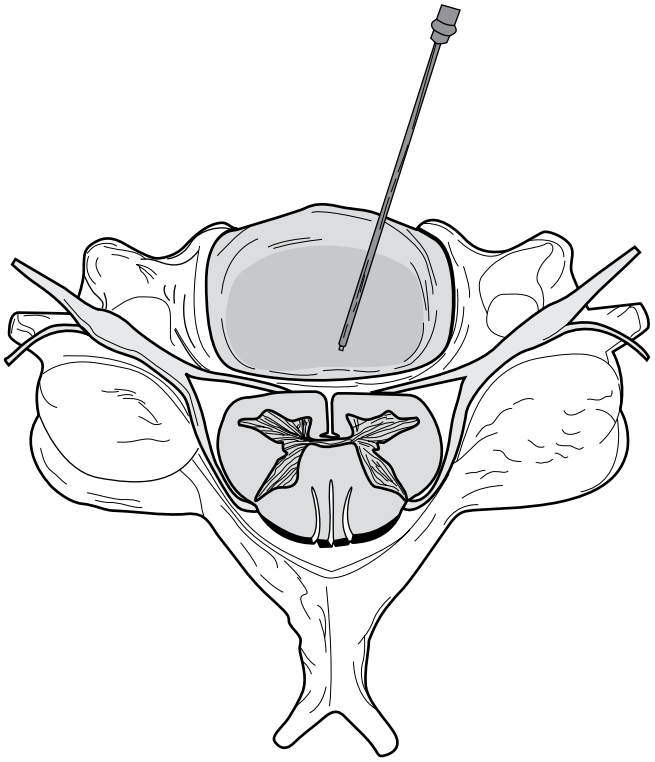


Figures 32.8 Introducer needle placement utilizing an oblique approach.

out of the center of the beam. With one hand, pressure is applied with the index and middle finger to the space between the trachea, medially, and the medial border of the sternocleidomastoid, laterally. This pressure will displace the trachea and esophagus medially, while moving the great vessels laterally. The right common carotid or internal carotid pulse should be clearly palpable laterally (Figure 32.7). The anterior surface of the disc and endplates may be palpated directly in thin patients.

The puncture point in the skin over the target is anesthetized with local anesthetic. The appropriate 18-gauge or 19-gauge introducer spinal needle is then advanced through the skin to its target, the anterolateral border of the superior endplate of the vertebral body below the disc. Close patient monitoring is necessary to rule out clinical suspicion of an insult to the trachea, esophagus, neural elements, or vascular structures. Once bony contact has been made, the palpating hand may be removed, while the needle is maintained in position by the driving hand. AP and lateral images are then viewed to ensure bony contact has been established on an acceptable trajectory. Next the introducer needle may be walked into the disc space in the lateral view. Repeated AP and lateral views are utilized to ensure the introducer is placed evenly between the endplates in the center of the cervical disc. Depending on the delivery system chosen, one may desire the needle tip reaching the middle or dorsal third of the disc diameter (lateral projection) when it is pointed toward the middle of the disc in the AP projection (Figure 32.8 and 32.9). A slightly off-center needle trajectory would be most useful for a sidelong delivery system (e.g., KTP^{532}) [145]. The laser fiber is then inserted through the needle into the center of the nucleus pulposus (Figure 32.10).

The proximal end of the fiber is then connected to the chosen laser previously calibrated to emit a preset power (typically 10–12 W). The pulse duration (0.2–1.0 seconds) with chosen interval (0.5–1.7 seconds) is calibrated to deliver the specified energy (10–12 J/s). Pulsed delivery of laser energy is used to allow dissipation of heat generated by a single pulse before administration of the next pulse, thereby avoiding excessive heating of surrounding tissues. When a patient experiences heat sensations during treatment, the use of longer pulse intervals or lower power settings are effective means for decreasing heat penetration. With a foot switch, under the interventionalist's



Figures 32.9 Illustration of introducer needle placement utilizing an anterior approach.

control, laser energy is delivered with pauses of 3 to 6 seconds (60- to 100-J intervals) for fluoroscopic confirmation of probe position, ruling out unexpected migration. This results in the controlled delivery of thermal energy until the expected total dosage (300–800 J) is delivered. Needle and fiber are then removed and a pressure dressing applied.

Patients are prescribed a soft cervical collar for 24 to 72 hours. No specific postoperative rehabilitation is standard. Gentle isometric exercises may be initiated after 2 weeks. A formal active physical therapy program may be initiated at 4 weeks and continued for another 6 weeks.

Clinical Outcomes

PLDD has been primarily utilized for the treatment of lumbar radicular pain secondary to a contained focal protrusion [143,163–171]. No randomized controlled trials have been performed. Most trials have been case series, with small sample sizes and a relatively low strength of evidence. Most studies report around a 75% success rate with PLDD [143,163–168], with more successful series reporting a rate as high as 87% [168]. The positive results from these outcome studies on PLDD have resulted in its widespread use as a minimally invasive treatment prior to considering open spinal surgery [143,163–168]. Recently, reports have surfaced indicating a possible role for PLDD in patients with discogenic low back pain diagnosed on discography [172,173]. However, these studies are deficient in strength to formulate any conclusions about the clinical

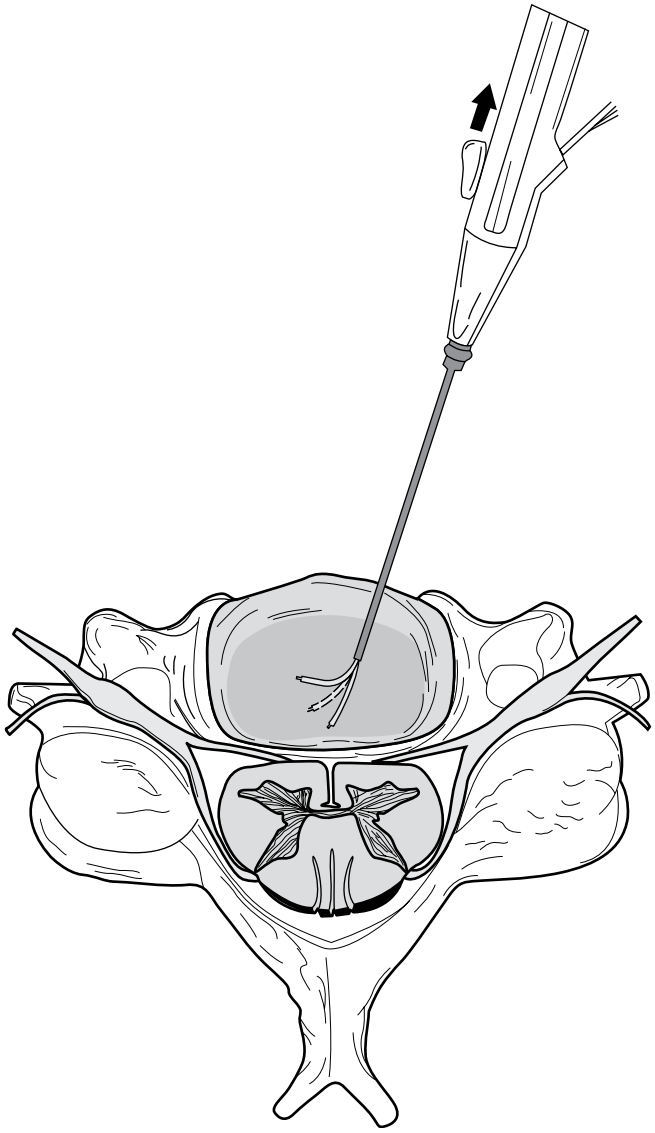


Figure 32.10 Cross-sectional view of a flexible laser fiber in the operative position, using an anterior approach.

efficacy of PLDD in lumbar internal disc disruption syndrome [172,173].

The literature on clinical outcomes in cervical PLDD is more limited. Most studies consist of case series or case reports in favor of cervical PLDD as a treatment option for cervical radicular pain secondary to a compressive focal protrusion [143,160,174]. At this time, clinical evidence to support the use of PLDD in patients suffering from cervical discogenic pain is lacking.

In one randomized clinical trial, published in 2001, Knight et al. [145] partially addressed the previously studied concept that radicular pain symptoms may occur in the absence of compressive pathology [175,176]. This is the first study to employ the reproduction of symptoms during provocation discography to determine the discal level to be treated. The primary objective was to compare the relative efficacy between Holmium:YAG and KTP[532] laser disc decompression in the treatment of cervical pain with

radicular symptoms with or without clear nerve root compromise on radiologic imaging. Patients with broad-based disc bulges, protrusions, or discal degeneration with neck pain and radicular symptoms of at least 6 months duration unresponsive to conservative treatment were included [145]. Reproduction of symptoms during provocation discography determined the discal level to be treated. Over a 7-year period PLDD was performed at 108 levels in 105 patients (54 Holmium:YAG, 51 KTP532). Regardless of which technology was utilized, the total energy delivered, per case, ranged between 600 and 800 J. After each procedure was completed, the disc was washed out with 10 mL of saline, and a total of 80 mg of Depo-medrone (methylprednisolone) was instilled into the disc space. The authors claimed corticosteroids offer the theoretical benefit of avoiding the postoperative flare of the symptoms secondary to the thermal effects of discal lasing [145]. The primary outcome studied was percentage change in the Vernon Mior Scores (The Vernon Mior Disability Index—VMI); 84 of the original 105 patients (80%) had results of complete clinical evaluation and review questionnaires returned at the time of data analysis. The average follow-up period was 43 months. Both groups showed clinical improvement, with no significant differences demonstrated between groups. As a whole, 51% had excellent to good outcome for neck pain, 38% for shoulder symptoms, and 53% for arm pain. This study [145] has multiple limitations, including an unclear randomization process, unknown follow-up rate within each laser group, the confounding variable of intradiscal steroids, and a lack of subanalysis of data comparing outcomes between patients displaying compressive lesion versus those with non-compressive degenerative changes. Despite these limitations, this is the first clinical study to begin to address the patient population with cervical internal disc disruption confirmed by provocation discography. This study introduces the feasibility of PLDD as a potential intervention in patients experiencing chronic axial neck pain secondary to cervical internal disc disruption syndrome.

Safety

Serious complications in cervical PLDD performed under fluoroscopic and computed tomography guidance are rare [145]. The overall complication rate for both minor and major adverse events is estimated at 1% of cases performed [177]. Common reported minor complications include transient postoperative swallowing discomfort [145], temporary perioperative Horner's sign [154], and intraoperative vasovagal reaction [177]. However, the quantity of published trials for cervical PLDD is insufficient to make strong conclusions on the complication rate of this procedure. Practitioners must be aware of the potential adverse outcomes learned from the cervical discography literature. Discitis, subdural empyema, spinal cord injury, vascular injury, and prevertebral abscess are major complications that have been reported [178–186].

Thus far, no significant injuries to the major vessels have been reported. Cases of trachea and esophagus lesions do exist [187]. The most common major complication that appears in the published literature is infection [143,177,188]. Choy et al. [143] reported one case of retropharyngeal abscess in 93 patients (0.6%) undergoing PLDD. Hellinger [188] experienced two infections in his first 261 cervical PLDD cases [177]. In 2004, Hellinger published an updated review of the complication rate from 3377 consecutive PLDD patients with cervical pain syndromes caused by bulging, protrusions, and contained and uncontained extrusions [177]. At the time of review, 95 more patients (total $N = 356$) had PLDD performed on their cervical spine. Injuries of major vessels, the trachea, or the esophagus did not occur. Three incidents (0.8%) of episternal hematoma were observed without consequences. No new infections were experienced. Hellinger calculated a rate of infection of 0.5%, comparable with that of Choy et al. [143] and previously published data on cervical discography [185].

When performing cervical PLDD, the interventionalist must be cognizant of the fact that the depth of vaporization created by laser cannot be controlled completely and the increased heat produced can cause damage to the surrounding tissues [189–194]. In an in vitro study, under standardized conditions, Schmolke et al. [148] concluded that a maximum energy of 600 J should be applied in cervical PLDD. Attempts to increase the applied energy would likely result in an insignificantly larger area of ablation and the heightened thermal risk to the neurovascular structures.

Percutaneous Disc Decompression with Coblation: Nucleoplasty

In 2001, the Food and Drug Administration approved Nucleoplasty, using Coblation technology, for treatment of contained herniated discs in the lumbar spine. Nucleoplasty is founded on similar scientific principles to PLDD to reduce intradiscal pressure [146,195]. Coblation is a non-heat–driven process that uses bipolar RF energy applied to a conductive medium (e.g., saline) to create a plasma field of highly ionized particles surrounding the electrode that have adequate energy to disintegrate molecular bonds within the nucleus material [196–198]. Essentially, the nucleus pulposus is vaporized. The products of the non-heat–driven process are elementary particles and low-molecular-weight gases that are removed quickly from the surgical site. When performed at the central portion of the disc, this causes a localized, low temperature molecular dissolution, resulting in volumetric nucleus tissue removal with minimal risk of collateral tissue necrosis [199].

Procedure

For cervical Nucleoplasty, a standard discography set-up and approach is also used [114,145,161] (see PLDD). The use of preoperative intravenous antibiotics is standard. Although mild sedation is commonly employed, the procedure is remarkably tolerable without sedative medication. The choice should be tailored to the individual.

Similar to PLDD, the angle of the 19G cannula is to 15° to 20° relative to midline so the tip of the needle will reach the center of the nucleus in both the coronal and sagittal planes (Figure 32.11). Tighten the lure lock on cervical

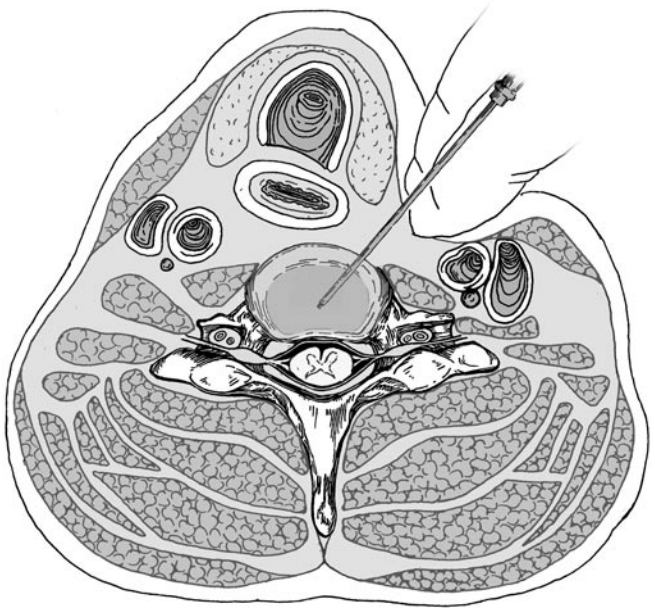


Figure 32.11 Cross-section of intradiscal placement of the introducer needle with a diagonal approach.

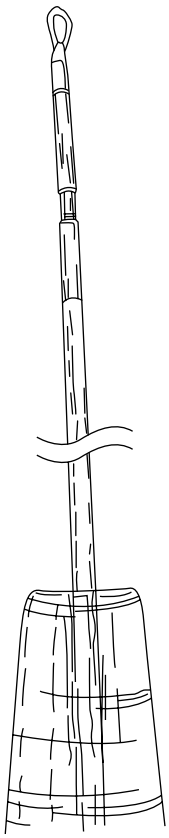


Figure 32.12 Perc-DC Wand.

needle and stylet before insertion to avoid stylet push-back. Once the needle tip is walked off the anterolateral border of the superior endplate, a lateral view is best as the introducer needle is advanced toward the dorsal third of cervical disc short of the posterior longitudinal ligament. Initial positioning is confirmed using AP and lateral views to gain a three-dimensional location of the needle tip. Next, the introducer needle is retracted slightly to the central disc. The catheter for the cervical disc (Perc-DC Wand; Figure 32.12) has a small looped tip. Creating this small channel distal to the introducer will ensure the looped tip has enough space avoiding excess bending. Withdraw stylet from needle and insert the Perc DC SpineWand under fluoroscopic guidance. SpineWand tip extends 5 mm beyond needle tip. Using AP and lateral views, needle tip position is confirmed slightly beyond (2 mm) center of disc.

The Perc-DC device is then fastened to the needle hub. The controller delivers RF energy to quickly ablate tissue at temperatures between 40°C and 60°C. The percutaneous decompression is done by the ablation mode. No coagulation mode is required, except for safety screening. Before initiating active ablation, depress COAG on the foot controller for one-half second. If stimulation (movement) is observed stop, and reposition the SpineWand tip. The Coblation is typically done in three cycles of 8 seconds rotating the tip of the Perc-DC wand for 180° each. The decompression should be started in the lateral view of the cervical spine with the loop of the Perc-DC Wand in the dorsal third of the nucleus pulposus of the treated motion segment. Depress ABLATION pedal on the foot controller for 8 seconds while rotating the flange 180° in a back-and-forth motion. Second, the medial third of the cervical disc would be treated and at least the ventral third of the disc for 8 seconds each. Special care must be taken (clear fluoroscopic imaging of the SpineWand tip) to avoid ablating too deeply into the tissue (Figure 32.13 A,B) or against vertebral body endplates.

Afterwards the catheter and the trocar are removed and the procedure is finished. During the operative procedure, the surgeon should hold the introducer needle when retrieving and then inserting the Nucleoplasty catheter (Perc-DC Wand). If the Perc-DC Wand begins to deflect or bend, withdraw the Perc-DC Wand into the needle and reconfirm introducer needle placement.

Patients are prescribed a soft cervical collar for 24 to 72 hours. No specific postoperative rehabilitation is standard. Gentle isometric exercises may be initiated after 2 weeks. A formal active physical therapy program may be initiated at 4 weeks and continued for another 6 weeks.

Clinical Outcomes

There are no published trials on the efficacy of Nucleoplasty for the treatment of axial neck pain confirmed by cervical discography. Singh [200] published a case report and scientific discussion on one patient with predominantly axial neck pain from the C6–7 disc. To date, the medical literature has been predominantly limited to case-control studies [162,201–203] of patients with radicular pain secondary to a cervical disc herniation.

The literature on the use of Nucleoplasty for the treatment of discogenic low back pain is also sparse [204,205].

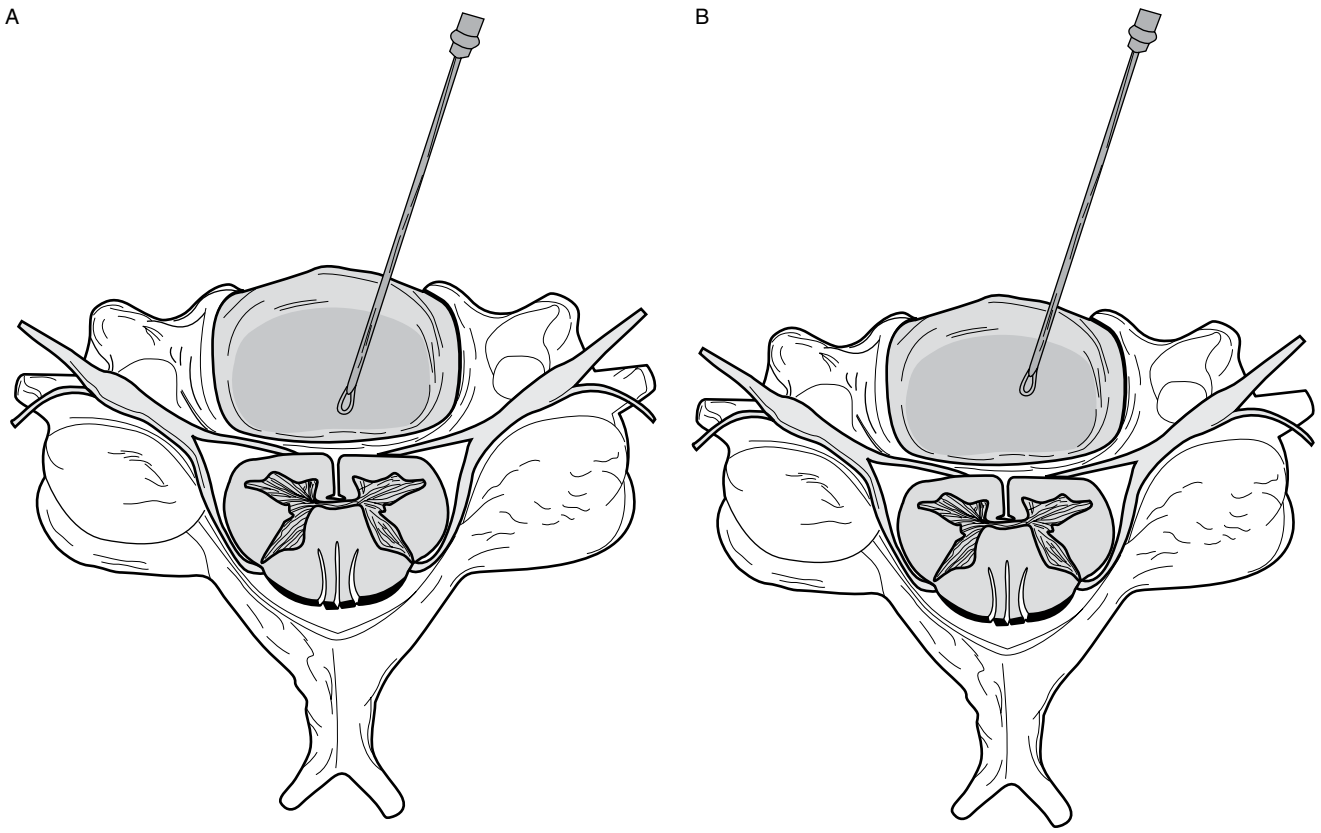


Figure 32.13 Cross-sectional view of the C5–6 intervertebral disc demonstrating the **(A)** distal and **(B)** proximal probe positions. Note the "anterior" approach used to create a central or ipsilateral ablation channel.

There are only two studies published demonstrating the clinical use of coblation for a cohort of patients with axial low back pain of discogenic origin [206,207]. Singh et al. assessed 47 patients with discogenic low back pain confirmed by positive provocative discography [206]. The proportion of patients obtaining 50% or more pain relief on numerical rating scale were 80%, 74%, 63%, and 53% at 1, 3, 6, and 12 months, respectively. Slipman et al. [207], in their pilot study on the use of Nucleoplasty in axial low back pain, noted a significant difference in patients displaying a central focal protrusion (CFP). Slipman et al. [207] had reported on 14 patients with CFP and 10 patients without a CFP, all of whom were selected by awake-provocative discography. At 3 months, the CFP group had a mean VAS score reduction of 5.3. The group without CFP displayed a mean VAS score reduction of 2.6. In the CFP group, there was a 64.3% success rate. In the group without CFP, there was a 30% success rate. Although neither of study [206,207] provides fundamental proof to support or disprove the clinical efficacy of Nucleoplasty for internal disc disruption syndrome, their existence may help lay foundation for studies that follow.

The results of Slipman et al. [207] suggest that the underlying pathology of a painful, degraded disc might dictate its response to intradiscal modulation. In animal studies, O'Neill et al. [208] demonstrated alterations in cytokine expression potentially associated with the mechanism of pain relief after plasma disc decompression. In the cervical spine, immunoreactivity and neovascularization are present in contained-type hernias [61]. This is probably related to detachment of the cartilaginous vertebral endplate associated with cervical herniations [52], where the detachment promotes neovascularization, and related immunologic phenomena [162]. The extent of the clinical biochemical effect of percutaneous plasma decompression may relate to the degree of modulation of the underlying neovascularization and immunoreactivity. The development of clinical trials with clear scientific method is required.

Safety

During plasma disc decompression, temperatures are kept between 40°C and 70°C to minimize thermal penetration and adjacent tissue damage. Laser temperatures are significantly higher in PLDD [209,210], which may result in significant heat propagation in excess of the desired therapeutic need [193]. In plasma disc decompression, histologic analyses [199] and temperature distribution studies [211] indicate very little damage or necrosis in surrounding disc tissue or end-plate cartilage with relatively low temperature readings within the disc during the procedure. Chen et al. demonstrated minimal increased temperature in adjacent neurovascular structures (spinal cord, nerve root, and vasculature) when coblation was performed at a distance greater than or equal to 5 mm away [211].

One lumbar study has directly assessed the side effects and complications following percutaneous decompression using coblation technology [212]. Lumbar Nucleoplasty seems to be associated with short-term increased pain at the needle insertion site and increased preprocedure back pain and tingling numbness but without other side effects.

No studies have been designed to directly address the safety of cervical Nucleoplasty. A review of the larger cervical Nucleoplasty studies provides a window to the potential complications of this procedure [162,202,203]. Bonaldi et al. [162], in an uncontrolled prospective case series study, found a 7% (4/55) incidence of local anesthetic-related side effects, which in all patients regressed within the first few hours. Included in this group were the transient experience of bradycardia (1/55), Horner syndrome (2/55), and bitonal voice (1/55). One case of infectious discitis was observed (1.8%). The patient was treated successfully by using a standard antibiotic regimen and rigid collar therapy. No bleeding complications were reported in this study. One patient experienced a device-related technical complication. During withdrawal of the device, the distal loop electrode detached from the tip of the device and remained in the disc space. They remained completely asymptomatic and had excellent clinical results at 29 months of follow-up.

In an uncontrolled prospective case series of 126 consecutive patients with 126 contained cervical disc herniations, Li et al. [202] reported one case of discitis for an overall incidence of 0.7%. As in study by Bonaldi et al. [162], there was one case where the Perc-D SpineWand had broken in the disc space during the procedure. Again, the partial Perc-D SpineWand that was broken in the disc space was not removed. At 1 year, the clinical outcome of this case was good, with no occurrence of any complication [202]. In an uncontrolled prospective case series of 50 consecutive patients, Nardi et al. [203] reported no complications. Transient side effects were not discussed.

As with cervical PLDD, the quantity of published trials is insufficient to make strong conclusions on the complication rate of cervical Nucleoplasty. Practitioners must remain cognizant of the potential adverse outcomes learned from the cervical discography literature. The risk of discitis, subdural empyema, spinal cord injury, vascular injury, and prevertebral abscess must be respected [178–186].

To limit potentially avoidable injury to the vital structures of the neck, a preoperative sonographic study may be performed to determine whether any normal or pathologic structures residing near the surgical pathway are at risk [162,213,214]. Although prudent, preoperative sonography may not necessarily affect complication rates. The most likely structures vulnerable to trocar injury would be the inferior thyroidal artery and the inferior laryngeal nerve when accessing the C6–7 disc space. It is unlikely the interventionalist will be able to mentally project the preoperative sonographic images onto the fluoroscopy screen with enough precision to alter the surgical pathway. Thankfully, inferior thyroidal artery and the inferior laryngeal nerve represent a small target when using a small-gauge (19-gauge) needle with a trocar tip.

Percutaneous Intradiscal RF

The prominent modes of thermal energy used in surgical applications are laser and RF [215–223]. The advantage of RF thermal energy is its ability to precisely target tissue, while simultaneously being accurately measured with temperature control technology. With RF, the targeted tissue temperatures stay localized within a 40°C to 90°C range, thus limiting heat dissipation and damage to adjacent tissue. Laser temperatures are significantly higher [224,225], which may result in significant heat propagation in excess of the desired therapeutic need [193]. RF has been employed for precise tissue ablation in conditions affecting vital organs such as the central nervous system where accurate ablation of the abnormal tissue is mandatory while excess ablation is undesirable and dangerous [226]. In cardiology, RF is used for ablation of aberrant pathways in Wolfe-Parkinson-White syndrome [227].

The performance of a RF procedure requires that an ablation probe be placed into the target tissue. The generator is turned on and target temperatures are input for the active tip of the RF probe. The RF probe does not directly generate heat. The electrode delivers an alternating RF current onto surrounding tissues. Component molecules are oscillated, causing ionic agitation and, therefore, friction in the tissue. This friction creates heat, and once sufficient temperatures have been reached, the heat destroys the target tissue within a few minutes. Thermocouples (miniaturized thermometers) incorporated into the tips of the electrodes allow continuous monitoring of tissue temperatures. This feedback enables automatically power adjustment ensuring that the target tissue temperatures remain constant.

Letcher et al. [228] demonstrated that RF current and heat preferentially block smaller C-fibers before the larger A-group, raising the possibility of using heat to modify nerves that transmit pain. It has been demonstrated that temperatures in and above the range of 42°C to 50°C are cytotoxic to nerve fibers [229–231]. This preference for small unmyelinated pain fibers, in combination with the accurate control over the location of the lesion, theoretically, makes RF extremely precise for treating various painful spinal ailments [232–237].

Previous thermal profile studies investigated the effects of lumbar spine intradiscal RF heating for treatment of symptomatic annular tears, by reducing or eliminating nociceptive transmission from posterior-lateral annular nerve endings [238–243]. Clinical evaluation of various intradiscal RF modalities including intradiscal RF, [234,236,244,245], distrode [237,246], intradiscal electrothermal therapy [247–252], [246,253–258], and biaculoplasty [259] has yielded modest results, at best.

It is only recently that the temperature distribution following the application of RF-generated thermal energy in the cervical spine has been investigated [260]. The clinical [236,245] and histologic [237,238] failures of lumbar intradiscal RF may not translate to the cervical spine, because of the known anatomical dissimilarities between the cervical and lumbar intervertebral discs [41,59,60,260]. In a recent benchtop study, Dreyfuss et al. [260] attempted to establish if single site, long duration intradiscal RF at

two different positions could generate adequate heating throughout the intervertebral disc to potentially ablate intradiscal nociceptors. Lesioning 85°C for up to 10 minutes in the middle of the disc with a 22-gauge, 4-mm active tip, Radionics (Burlington, MA) RF needle did not generate consistent temperatures that reached or exceeded 45°C (minimum threshold for denervation) in the posterior quarter of the disc, but lesioning in the posterior third of the disc did create temperatures at or above the minimum threshold in both the posterior and middle of the disc with mean peak temperatures of 54°C [229–231]. The authors were concerned, however, that lesioning in the posterior third of the disc failed to reach adequate temperatures in the anterior aspect of the disc. Dreyfuss et al. concluded that initial data suggest intradiscal RF fails to provide sufficient temperature increases throughout the entire disc to achieve adequate denervation. The results from this study [260] are consistent with previous lumbar data [239]. Further extrapolation from lumbar studies of intradiscal RF [242,243] suggests lesioning in the posterior third of the disc may reach sufficient temperatures in the posterolateral aspect of the disc, where the majority of nociceptors reside [59]. Different modes of intradiscal heating should be evaluated with appropriate bench studies to assess its effectiveness and safety [260].

DISCUSSION

Pain and disability from axial neck pain is a common complaint with a reported lifetime incidence of 66% [261]. Traditionally, treatment of presumptive cervical discogenic pain includes nonsurgical options, such as activity modification, medication management, physical and/or manual therapy, exercise, massage, and epidural corticosteroid injections [260]. When medical/interventional management fails, patients are forced to consider major surgery as the final step in their therapeutic algorithm. Given the prevalence of this problem and the limited treatment options, the development of fluoroscopically guided procedures, both epidural steroid injection and intradiscal, is a hopeful potential step in the treatment for chronic cervical discogenic pain. To date, no intradiscal procedure has been demonstrated as both a valid and efficacious therapeutic option in discography proven, axial, discogenic neck pain. Considering the benefits of minimally invasive care over surgery, further research is warranted.

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33 Diagnostic Imaging of Painful Cervical Facet Joints

Thomas R. Saullo and Frank Bender

INTRODUCTION

Neck pain is generally accepted to emanate primarily from either a painful cervical facet joint or from internal derangement of a cervical intervertebral disc [1]. The prevalence of cervical facet joint pain (CFJP) as a cause of neck pain is estimated [1,2] to be in the range of 25% to 63% (see Chapter 1). Production of concordant pain upon deep palpation over a symptomatic cervical facet joint can be predictive of which joint is symptomatic [3–5]. Physical examination findings of focal pain at a given joint region coupled with diagnostic injections resulted in increased diagnostic accuracy and gave rise to performing the accepted double block paradigm in use today (see Bogduk et al).

Raymond and Dumas [6] were the first to show that computed tomography (CT) scans did not reliably diagnose painful facet joints in the lumbar spine. Painful facet joints were diagnosed when patients experienced temporary relief of their symptoms after fluoroscopically guided injections of an anesthetic agent into the appropriate facet joint. Approximately 20 years later, a similar study, this time focusing on the cervical spine, was conducted with the same conclusion [7]. The end result has been the current gold standard of anesthetizing the facet joint with utilization of CT-guided [7–9] or fluoroscopic-guided [10–14] placement of a needle onto either the appropriate medial branches [11] or into the facet joint itself [3,7–10,12–14].

Dory [8] described using CT imagery for placing a needle into a suspected facet joint using a posterior approach. Murtagh [9] switched to CT from fluoroscopy anecdotally expressing that CT scan assisted him far better in placing the needle into the joint. However, most published articles and practicing clinicians utilize fluoroscopic guidance [10–14]. Recent literature supports using small amounts of anesthesia, volumes as low as 0.25 mL, to the medial branches [15] to increase diagnostic accuracy by redusing false-positives attributed to aberrant spread.

Advanced imaging techniques, such as magnetic resonance imaging (MRI), CT, and single photon emission computed tomography (SPECT) scans, constitute an essential component of diagnosis in many areas of medicine. It would seem logical that painful cervical facet joints, as identified by a focal source of pain on physical examination [4,16], would have corresponding findings identifiable with these imaging techniques [17–24]. However, physical examination [4,16] and imaging alone [6,7] have not been shown to be diagnostic of CFJP in clinical situations. In regards to CFJP, the utility of CT imaging and fluoroscopy lies in assisting in the actual performance of diagnostic injections, rather than as a diagnostic tool. There are certain clinical situations, although less common, where radiologic studies are essential in making the diagnosis of the painful facet joint. When infection [18,20,23], tumor [21,22], fracture [19], or dislocation are clinically suspected or incidentally found, imaging is invaluable. The purpose of this chapter is to review the use of advanced imaging, MRI, CT, bone scan, and SPECT in the diagnosis of the painful cervical facet joint.

COMPUTERIZED TOMOGRAPHY SCANS

CT scans have been found to be superior to MRI in determining morphologically normal facet joints, ankylosed facet joints (Figure 33.1), and has better inter-rater and intrarater reliability regarding facet joint characterization [25]. MRI is not reliable in adequately determining the presence or degree of facet joint arthrosis. CT remains the gold standard for typifying facet joint arthrosis [25,26]. Despite better facet joint imaging, no statistically significant relationship between the degree of cervical facet joint osteoarthritis seen on CT and pain relief reported from cervical facet joint injection has been reported [7].

MAGNETIC RESONANCE IMAGING

MRI can adequately assess cervical intervertebral discs and neural compression; however, MRI evaluation for facet arthropathy is limited [26–28]. Clinically, Cohen et al. [29] found that cervical facet pathology seen on MRI was not predictive of successful outcomes from RFA procedures. Recently, meniscoids have been shown on MRI T1 and T2 imaging of cervical facet joints [17]. Meniscoids have been hypothesized to be a pain source in the cervical facet joint. However, like CT scanning, MRI alone has failed to demonstrate whether a given joint is a pain generator in the absence of infection, dislocation, fracture, or cancer in a given joint. Meniscoid development may

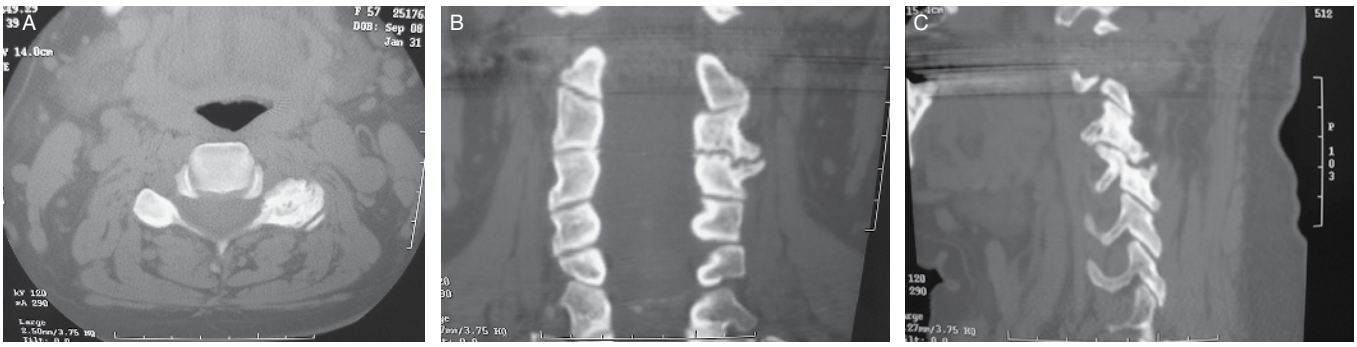

Figure 33.1 Facet arthrosis. Axial **(A)**, coronal **(B)**, and sagittal **(C)** CT imaging of facet arthrosis of the left C3-4 joint contrasted with a normal appearing joint on the right. Findings include joint space narrowing, asymmetrical joint angulations, joint irregularity, osteophytes, and subchondral sclerosis.

stem from degeneration due to rubbing of the synovial lining. Meniscoids are seen on microanatomy sections but at this point are not visible on standard MRI technology. Recently developed high-field 3T MRI has been shown to identify meniscoids on cadaver specimens as seen on frozen microanatomy sections [17]. However, at this juncture no studies have been performed to ascertain whether a finding of meniscoids on MRI correlates to the facet joint being painful or if meniscoids are equal to a synovial fold.

NUCLEAR IMAGING

Some studies have shown that SPECT scans can predict relief after intra-articular facet joint steroid injections [8–11]; however, this does not necessarily diagnose the facet joint as the pain generator. Other factors can result in pain reduction such as placebo response, systemic response to steroids, and leakage of injectate outside the joint effecting nearby neural elements [6]. A recent study by Makki et al. [24] found no correlation between SPECT and anesthetic blocks and therefore recommend not using positive facet joints on SPECT as the sole indication to anesthetize or denervate facet joints [24].

CERVICAL FACET PATHOLOGY

Joint Degeneration

Osteoarthritis of the facet joint is commonly seen along with degenerative disc changes and displays the same degenerative changes noted in other synovial joints. Severe degenerative changes in the facet joints include joint space narrowing, asymmetrical joint angulations, joint irregularity, synovial inflammation, osteophytes, subchondral sclerosis, and calcification at the insertion of the flaval ligaments [30]. Damage to the articular cartilage advances from superficial to deep starting as fibrillation and then progresses to fissures that ulcerate through the cartilage [31]. These degenerative changes become more prevalent with age. Twenty-five percent of individuals aged 50 and up to 75% of those above age 70 display degenerative cervical facet joint changes [32]. Degeneration may be associated with pain; however, many patients remain asymptomatic indicating that these changes are not in and of themselves painful [32,33].

Juxtafacet Joint Cysts

Synovial and ganglion cysts associated with a facet joint are termed juxtafacet cysts and may be related to progression of a disease process and rarely occur as a result of trauma [34–36]. Cysts associated with chronic spinal degeneration usually arise from herniation of the synovial lining or from chronic overloading [37]. Juxtafacet cysts are more commonly seen in the lumbar spine but they do occur in the cervical spine as well. Clinically they can present with symptoms similar to those seen with disc protrusions. Free fluid in the cyst is identifiable on MRI. If hemorrhage is present within a cyst, increased signal is seen on both T1- and T2-weighted MRI [38]. CT myelography may demonstrate extradural cysts originating from degenerated facet joints [39]. Both MRI and CT myelography can adequately diagnose epidural cysts.

Facet Joint Infection

Infection of cervical facet joints is rare and usually bacterial versus fungal or mycobacterial [18,40]. The origin of infection is most often from hematogenous contamination secondary to distant infections, commonly because of urinary tract infections, and rarely a result of direct inoculation [18]. Patients who develop facet joint infections often have predisposing factors such as an immunosuppressed state [41,42] or underlying joint disease [43]. Standard radiographs are usually normal but may reveal a widened facet joint space with hazy margins [18]. Hot spots are directly lateral to the spine and more pronounced on the posteroanterior compared to anteroposterior (AP) view with radionuclide bone scanning of infected facet joints (Figure 33.2) [18]. MRI may be positive within 2 days of symptoms onset [44] and can reveal joint destruction,

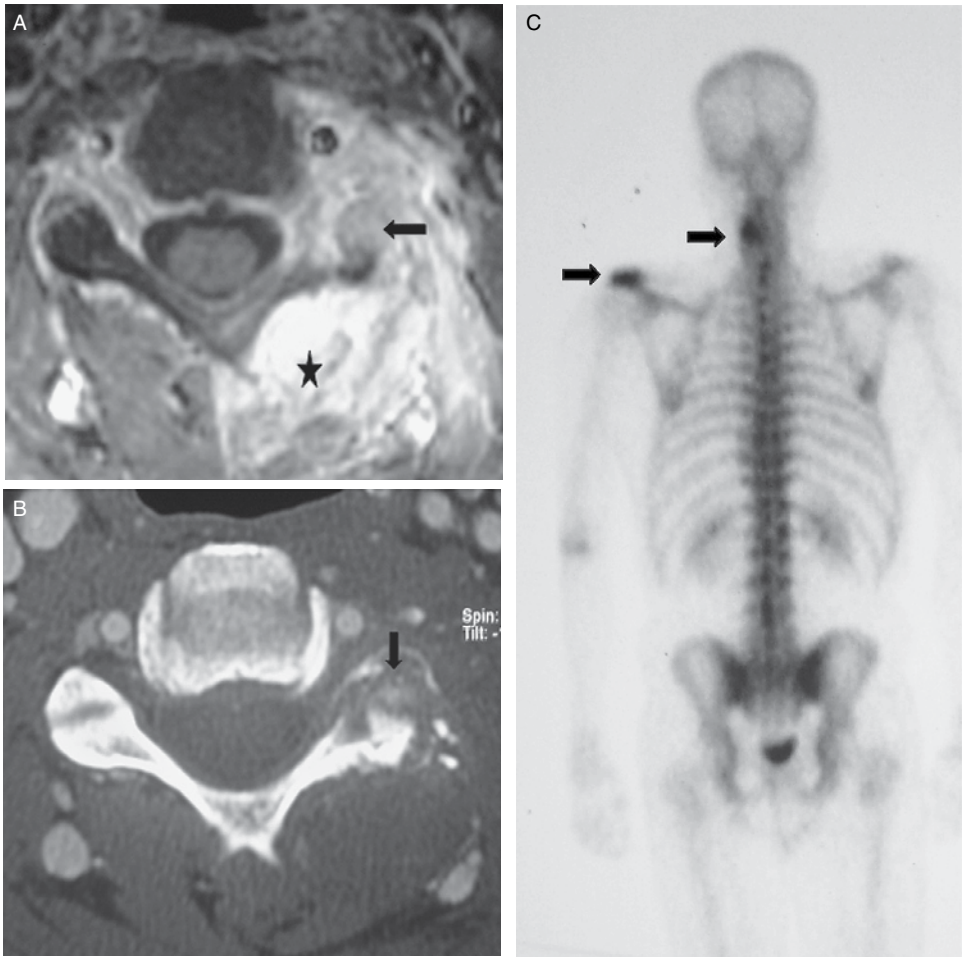

Figure 33.2 Facet infection. **(A)** Axial T1-weighted sequence MRI with fat saturation and gadolinium depicting destruction of the left C4-C5 facet joint space with erosions of the margins (black arrows). Spread of the infection is seen with enhancement of the paraspinal muscles (black star) and epidural space. **(B)** Axial CT with contrast revealing lysis of the left C4-C5 facet joint space with narrowing and erosions of the margins. Soft tissue enhancement anterior to the facet joint indicates an abscess (black arrow). **(C)** Tc 99m-labeled bisphosphonate scan reveals two hot spots, one at the left acromioclavicular joint and the one in the left cervical paraspinal area.

intra-articular effusion, synovitis, and abscess or spread to the epidural space or adjacent muscles that enhance with gadolinium [18]. MRI is the modality of choice for imaging suspected facet infections.

Cervical Facet Neoplasm

Neoplasms involving the cervical facet joints have been reported but are uncommon. Patients usually report axial pain but many also have radicular symptoms due to epidural extension of the lesion. Synovial-type giant cell tumors may arise from the synovial joint lining. Radiographic findings include erosion, scalloping, and destruction of bone (Figure 33.3). Aneurysmal bone cysts are well demarcated, eccentric, lytic lesions bordered by a thin rim of new bone that typically involve adjacent vertebrae including the cervical facet joints [45]. Osteoblastomas are benign neoplasms arising primarily from the posterior elements of the vertebra and tend to be associated with a palpable mass [22]. These are distinguished from synovial tumors, because they are well circumscribed and surrounded by a dense halo of reactive bone formation [22,45].

Cervical Facet Dislocations

Radiographs are usually sufficient to diagnose dislocations and fractures involving the cervical facets. If the findings are negative or subtle, a CT or MRI may be required to identify abnormalities (Figure 33.4). When the superior articular process (SAP) is located posterior to the corresponding inferior process, normally positioned anterior to the inferior articulating process (IAP), it is termed as "locked" or "jumped" (Figure 33.5). This is a fairly stable position and may occur unilaterally or bilaterally. A rotational vertebral displacement is seen with unilateral jumped facets. On lateral (oblique) radiographs, the foramen directly inferior to the jumped facet is obscured [46]. In comparison, bilateral jumped facets result in greater AP misalignment, due to ligamentous instability, and are associated with a higher incidence of spinal canal compromise and corresponding spinal cord injury. Facets can also be described as "perched" when the tip of the SAP is resting on the tip of the IAP (Figure 33.6). Perched facets may have only subtle radiographic findings. They are inherently unstable and potentially self-reducing or they may progress to a jumped position with subsequent compromise of the spinal canal [46].

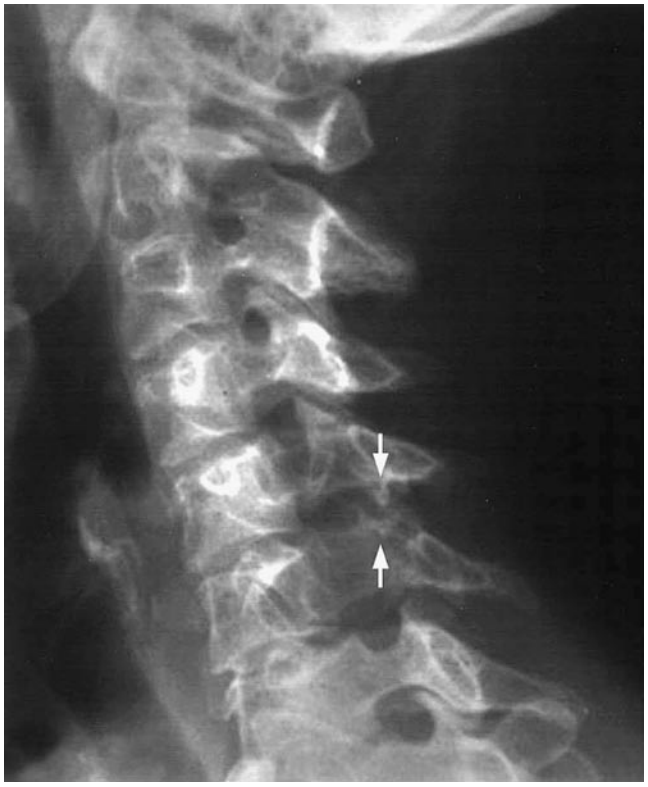

Figure 33.3 Facet tumor. Synovial-type giant cell. Oblique radiograph demonstrating extrinsic erosion of the C5-6 articular facets because of synovial-type giant cell tumor (arrows).

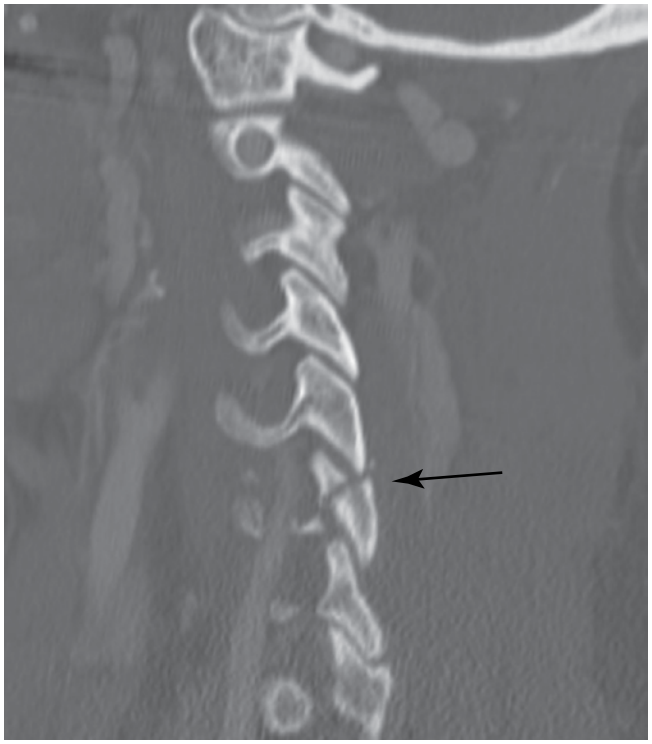

Figure 33.4 Facet fracture. Parasagittal thin-cut CT depicting fracture of the C6 SAP with involvement the C5-6 facet joint (arrow). The vertebral artery can be seen coursing through the C6 transverse process.

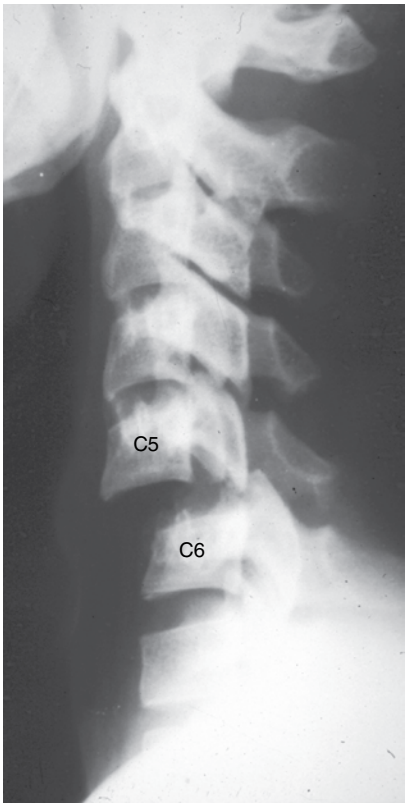

Figure 33.5 Locked facets. Radiograph depicting the C5 vertebra anteriorly displaced in relation to C6 as a result of bilateral "jumped" or "locked" facets. This injury may result in spinal cord compromise.

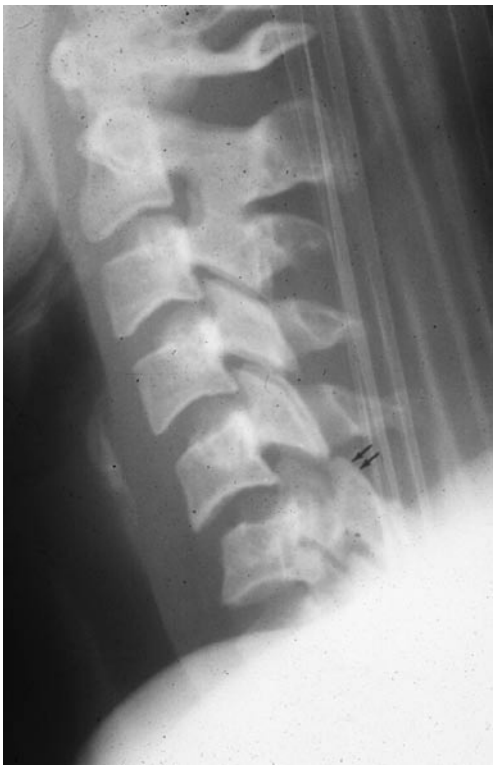

Figure 33.6 Perched facets. Radiograph of the IAPs of C5 "perched" on the SAPs of C6 in an unstable configuration. This tenuous condition may spontaneously reduce or progress into a fully "jumped" position.

CONCLUSION

Sings of degeneration, arthrosis, or pathology may be seen in cervical facet joints on advanced imaging; however, these findings do not correlate with the joint being a source of pain when infection, fracture, dislocation, and tumor are excluded. Diagnostic anesthetic block of the medial branches or the facet joint itself, performed with fluoroscopic or CT guidance, may accurately identify a painful facet joint. After two confirmatory positive diagnostic blocks, targeting either the medial branches innervating the facet joint or the joint itself, radiofrequency neurotomy of the appropriate medial branches is the preferred treatment.

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34 Diagnostic Blockade of Painful Cervical Facet Joints

Wade King

INTRODUCTION

Cervical facet joint pain is pain stemming from one or more of the posterior synovial joints of the cervical spine, commonly called the cervical facet joints. These joints are not uniform in shape, orientation, movement, or in their articular neurology. They vary significantly in those respects, especially at the upper cervical levels, and so are more accurately termed, from the top of the cervical spine downward, the atlanto-occipital joints (at the C0-1 level), the lateral atlantoaxial joints (at the C1-2 level) and the cervical zygapophysial joints (at lower levels from C2-3 down to C6-7). The lower joints, from C2-3 downward, are much more commonly involved in pain syndromes than those above them. As "zygapophysial joints" is the correct name for the joints from C2-3 to C6-7 in current anatomic nomenclature [1] and it is the term most commonly used in the relevant literature, in this chapter those lower joints will be designated cervical zygapophysial joints, cervical z. joints or CZJs.

CLINICAL DIAGNOSIS OF CERVICAL FACET JOINT PAIN

Cervical facet joint pain can be diagnosed definitively, using tests that are minimally invasive. Before proceeding to order (or perform) such tests, the clinician should decide whether specific investigation is warranted. The key issue in that decision is therapeutic utility: if the tests are done, will their results lead to more effective treatment? In relation to neck pain, that issue in turn hinges on the duration of the condition, that is, whether the pain is acute or chronic.

Acute neck pain (present <3 months) does not need investigation, other than by imaging if indicated to rule out fractures and other serious conditions. This is because most acute neck pain is due to sprains of fibromuscular tissues, which will resolve over a period of days or weeks by natural healing if simply left alone [2]. No treatment is required other than explanation of the favorable natural history, reassurance, and perhaps analgesic medication to make the patient comfortable; beyond that, all that is needed is follow-up until the pain settles.

Chronic neck pain (present >3 months) requires an entirely different approach. By convention, 3 months is considered a reasonable time to allow for natural healing; pain persisting for longer than that is considered chronic and unlikely to remit until an effective intervention is applied. The key to the management of chronic spinal pain is specific diagnosis: if the cause is determined, effective treatment can be applied and in many cases the pain will be abolished. Thus, specific investigation is not only justified but also essential for chronic neck pain.

Patients presenting with chronic neck pain may be suspected of having cervical facet joint pain, on epidemiologic grounds, as the synovial joints are the most common sources of neck pain that persists for more than 3 months [3]. The upper joints may also be sources of headache, of the type termed "cervicogenic headache" [4]. Although they are so commonly sources of pain, it is not possible to identify any specific joint or joints as the source(s) of a patient's problem by clinical assessment (eliciting the medical history and performing physical examination); there are no clinical methods that are valid for this purpose [5]. Some guidance is provided by established patterns of distribution of pain from individual joints, the so-called cervical "pain maps" [6–8]. The more common patterns are illustrated in Figure 34.1.

It can be seen from this figure that pain maps are insufficient to identify a specific joint as a pain source because the maps overlap so much. Each part of a patient's neck lies in at least two and perhaps as many as four of the areas to which pain is referred from particular joints.

Medical imaging does not provide accurate identification of cervical joint pain sources either: none of the radiologic changes seen on plain radiography, computed tomography scanning, magnetic resonance imaging, or any other imaging modality have been proven to be correlated with cervical facet joint pain [9–11].

The only way of determining that a cervical joint is (or is not) a source of pain is to test it by anesthetic blockade of that joint alone, to see if the pain is abolished by the blockade: such tests are called diagnostic cervical facet joint blocks.

Readers should note that diagnostic block procedures are described in this chapter in sufficient detail to promote understanding of them but no more than that. The practice of interventional spine care requires particular skills that can only be gained by proper practical training under the supervision of someone experienced in the relevant

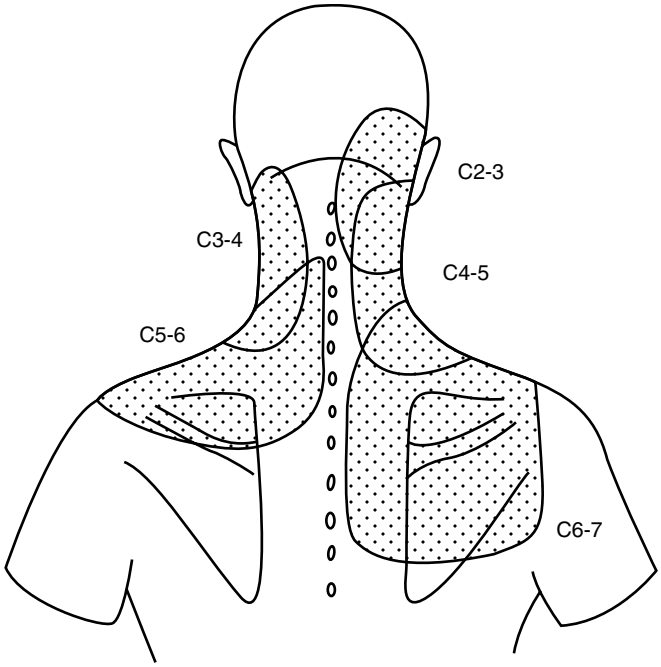

Figure 34.1 Pain maps showing patterns of distribution of cervical zygapophysial joint pain stemming from the C2-3 to the C6-7 levels. From ref. [6].

techniques. A person who has not had such training should not attempt to perform any of these procedures simply by following the descriptions and figures in this chapter.

Diagnostic Blockade of Cervical Facet Joints

Generically, the term *diagnostic cervical facet joints blocks* includes blocks of the joints themselves and blocks of their sensory nerves. The distinctions between them are significant. Historically, the first method used to test a cervical joint as a possible pain source was injection of a local anesthetic agent into the joint cavity to block sensory signals from the joint's internal structures; such tests are called *intra-articular blocks* (IABs). Later, less invasive methods were developed to achieve blockade of a specific joint by blocking the nerve(s) outside a joint, which carry pain signals from that joint; such tests are designated by the particular nerves involved as *medial branch blocks* (MBBs) and *third occipital nerve blocks* (TONBs). As articular nerve blocks are less invasive and they are performed more often to investigate the common sources of chronic neck pain (the CZJs from C2-3 down to C6-7), they will be described first.

Facilities and Equipment Required

Safety and specificity are of critical importance for any diagnostic block of a cervical joint. A block test involves inserting one or more spinal needles into the patient's neck and placing the needle tip(s) at specific target point(s) on the cervical vertebrae for injection of local anesthetic. It is vital for the operator to know where the needle is at all times, both

Figure 34.2 An X-ray procedure suite in which a patient is having a cervical facet joint block. Note the C-arm fluoroscope with image intensifier and high-resolution monitors. Courtesy of Pendlebury Clinic, Newcastle, Australia.

for the safety of the patient and for the accuracy of the test procedure. Accordingly, all such procedures must be done with clear fluoroscopic guidance. A high-resolution image intensifier is an absolute requirement, preferably one with a C-arm, which allows the X-ray beam to be directed readily at different angles to provide anteroposterior (AP), oblique, and lateral views without moving the patient. Other equipment required includes a radiolucent X-ray table, preferably one with a mobile plinth that can be moved to modify the fluoroscopic views, as in Figure 34.2.

To undertake a block procedure, the operator will need facilities for antiseptic hand washing (a scrub sink or similar), a sterile towel, sterile gloves, a skin preparation set with an appropriate antiseptic to prepare the skin of the target area, one or more sterile drapes for the patient, one or more 25- or 26-gauge spinal needles of at least 3.5 inches (88 mm) in length, a 2 mL syringe, an extension tube of minimal volume, a drawing-up needle, and a local anesthetic agent for injection (Figure 34.3).

Patient Preparation

The patient must be properly prepared for a joint block procedure in the same way as for any other intervention. The clinician should inform the patient about the purpose of the test, the technique to be used, what they can expect while undergoing the procedure, and all associated risks. Having received this information, preferably in printed form as well as verbally, the patient must give informed consent in writing before each test procedure is undertaken.

Articular Nerve Blocks

The nerves that carry afferent fibers for modalities of sensation including pain from the CZJs from C3-4 down to C6-7 are the medial branches of the cervical dorsal rami;

Figure 34.3 Sterile preparation tray and equipment required for a cervical facet joint block. Courtesy of Pendlebury Clinic, Newcastle, Australia.

hence, the procedures used to test those joints as sources of pain are called MBBs.

The sensory nerves that supply the C2-3 z. joints are the third occipital nerves; the procedures used to test those joints as sources of pain are called TONBs.

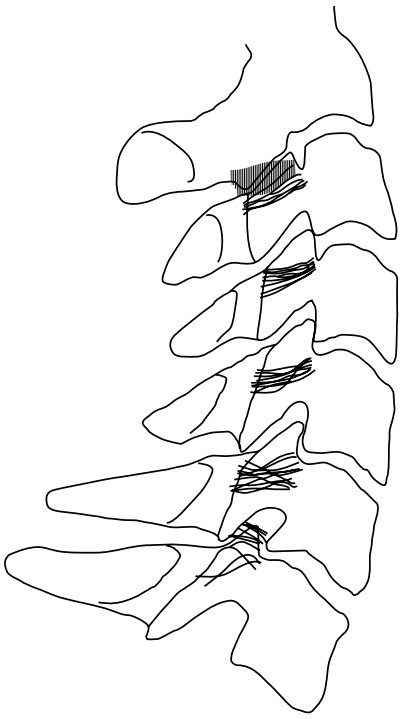

Figure 34.4 Plots of the courses of cervical medial branches across the articular pillars of the vertebrae. From ref. [12].

Medial Branch Blocks

The intention of an MBB test is to determine whether a particular CZJ is a source of pain. The test is designed to be specific, so only one joint should be tested at a time, at least initially. If the results of specific tests suggest that two or more joints are contributing to the patient's neck pain, on a subsequent occasion they may be tested together to see if the combination is causing all the pain in that region.

Each of the CZJs from C3-4 down to C6-7 is supplied by two medial branch nerves, the medial branch from the cervical dorsal ramus above, and that from the cervical dorsal ramus below. Thus, the C5-6 z. joint is supplied by the C5 medial branch and the C6 medial branch. The procedure to test that joint as a source of pain involves blocking each of those two innervating nerves. The courses of the medial branches have been plotted by cadaveric dissection studies and shown to lie more-or-less horizontally across the middle section of the articular pillars that join the superior and inferior articular processes of each vertebra [12]. These medial branch courses are illustrated in Figure 34.4.

These nerves are the targets at which MBBs are aimed. The nerves themselves are not seen on fluoroscopy during a procedure but by appreciating the ranges of their courses it can be understood how a particular medial branch can be anesthetized. Such blockade is achieved by placing the tip of a spinal needle at the midpoint of the relevant articular pillar and injecting a small volume of local anesthetic, just sufficient to reach the highest and lowest of the known courses of the nerve in question. The volume of the injectate must be large enough to achieve that coverage reliably but not so large as to spread to and block other nerves, as that would compromise the specificity of the test. Local anesthetic preparations suitable for this purpose are lidocaine 2% or bupivacaine 0.5% and the volume of injectate sufficient to achieve the desired spread has been found to be 0.5 mL [13].

The first step in an MBB procedure is to select the z. joint to be tested. When the patient is admitted to the test facility, he or she should be seen by a nurse trained in pain assessment. The nurse should ask the patient to identify the pain for which the test is to be done; this is designated the "index pain" for that day's test. For example, if a patient has lower neck pain and headache, the lower neck pain, if more severe, may be identified as the index pain that day. The nurse should then ask the patient about the intensity and the distribution of the index pain. The intensity should be recorded as a score out of 100, using a printed Visual Analogue Scale (VAS), a pain ruler, or some similar device. The distribution should be recorded by the patient, marking it on a body chart. The selection of the joint to be tested can then be done by comparing the patient's pain pattern to the pain maps shown in Figure 34.1. Some further help in selection may be gained by considering the known epidemiology of CZJ pain [3,4]. The prevalence of C5-6 z. joint pain is greater than that of other z. joints in the lower neck and the prevalence of C2-3 z. joint pain is greater than that of other z. joints in the upper neck, so if other features are equivocal those joints may be selected as the first to be tested for lower and upper neck pain, respectively.

The MBB procedure recommended by the International Spine Intervention Society (ISIS) and described fully in

the relevant ISIS practice guideline [14] involves preparing the patients (as outlined above) and placing them on the table of the fluoroscopic suite, lying comfortably on their side with the painful side uppermost (as in Figure 34.2). After skin preparation and draping, the operator and radiographer together should determine the position of the first target nerve. For example, if a C5-6 MBB is to be performed, the target point for the C5 medial branch will be the midpoint of the C5 articular pillar. The radiographer should then obtain a clear lateral view of the target region and cone the image to it. Coning is important to minimize the exposure of the patient (and the operator and radiographer) to radiation. The operator should select a suitable needle (usually a 25- or 26-gauge spinal needle at least 3.5 inches or 88 mm in length) and place its tip on the skin of the patient's neck directly over the target point, as in Figure 34.5.

The skin point over the target will be the needle insertion site. The operator should keep the tip there and stand the needle up so it is aligned with the fluoroscopic beam, then tell the patient they will feel a slight prick and introduce the needle through the skin as gently and smoothly as possible. After insertion, the position of the needle should be checked in intermittent fluoroscopic views as it is directed through the muscles of the patient's neck. The needle is steered (by moving its hub, shaft, and/or bevel utilizing techniques acquired in ways that are learned through rigorous practical training) until the needle tip reaches bone at the target point. Its position there should be checked and recorded on a fluoroscopic image, as in Figure 34.6.

When the needle tip has been confirmed as at the target point, a syringe loaded with local anesthetic solution (lidocaine 2% or bupivacaine 0.5%) should be connected to it by an extension tube, and after aspirating to check for blood in the usual way, 0.5 mL of the local anesthetic preparation should be injected.

The position of the second target should then be determined and a second needle inserted in a similar way, until its tip is resting on bone at the second target point (the midpoint of the articular pillar below the joint). The position of the second needle at its target should be checked and recorded on a fluoroscopic image, as in Figure 34.7.

The second injection of 0.5 mL should be made over the second medial branch and the needles withdrawn to complete that part of the MBB procedure.

When the needles are out, the patient should be told the procedure is over. The drape should be removed and the antiseptic sponged gently from the skin. The patient should be asked if they feel lightheaded or dizzy. If they do they can be reassured that such symptoms are quite normal after a cervical nerve block and they should be allowed to lie quietly until the dizziness settles (which may take a minute or two). When the patient is not dizzy, they should be allowed to sit up, being supported by the operator and/or nurse in case the change of posture makes them lightheaded. When they feel ready, the patient should be taken from the fluoroscopy suite to a room in the facility where he or she may rest and be observed by a nurse trained in pain assessment. The nurse should ask the patient to assess the intensity of the index pain and record the score on a pain chart at half-hourly intervals beginning immediately after return to the observation area and continuing until

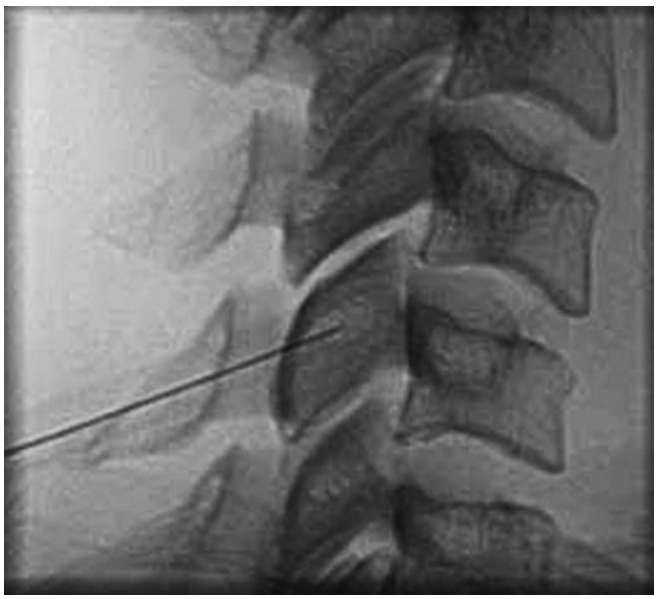

Figure 34.5 Lateral fluoroscopic view of the right C5-6 z. joint with the needle tip on skin over the midpoint of the C5 articular pillar, the target point for the C5 medial branch.

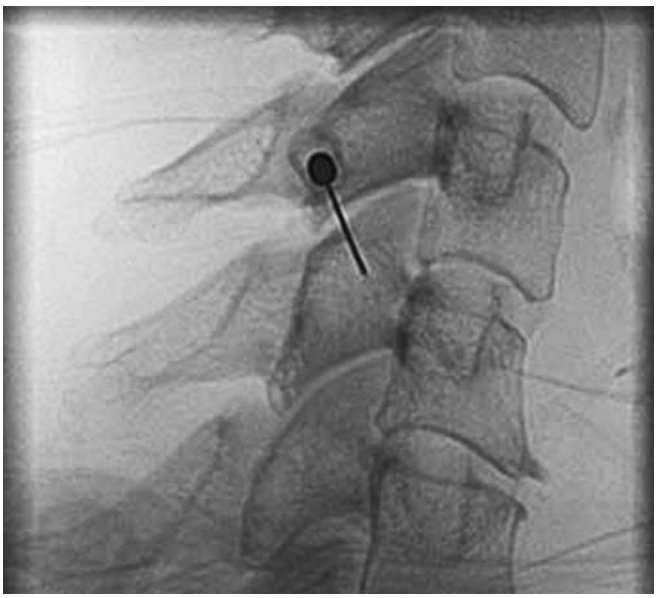

Figure 34.6 Lateral fluoroscopic view of the right C5-6 z. joint with the needle tip on bone at the target point for the C5 medial branch.

90 minutes after the injections. At the end of the observation period, the pain chart should be reviewed to determine the result of the test (Figure 34.8).

At the C7 level, the medial branch follows a course somewhat different from those above it and its range of possible positions is more diverse from person to person (as seen in Figure 34.4). Thus its blockade (as part of a C6-7 MBB test) requires a slightly different technique. The C7 medial branch passes backward over the superior articular process of its vertebra, somewhere between its peak and the root of the transverse process; in some patients it is slightly superficial to the bone rather than immediately adjacent to it, because the nerve is separated from the bone by a slip of the semispinalis capitis muscle. The main target for a C7 MBB is on the lateral aspect of the curved surface of the articular process and up near its peak, with a secondary target about 3 mm superficial to that. Injections of 0.3 mL of local anesthetic at each of these sites can be expected to produce C7 medial branch blockade. The injection technique is similar to that for other levels in that when the needle tip reaches bone at the main target site its position should be checked (first) in a lateral view (Figure 34.9).

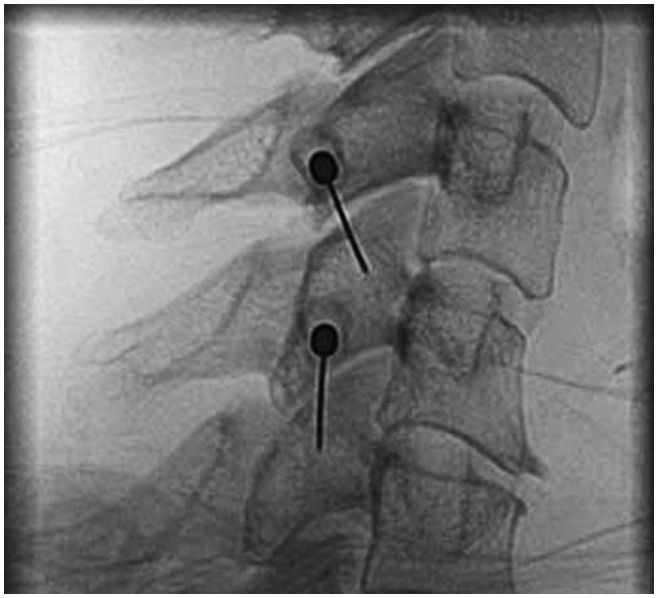

Figure 34.7 Lateral fluoroscopic view of the right C5-6 z. joint with the two needle tips on bone at the target points for the C5 and C6 medial branches.

The C-arm should then be rotated for an AP view, as in Figure 34.10, in which the needle tip position can be checked again to ensure it is above the transverse process and on the lateral aspect of the bone, not on the anterior or posterior parts of its curved surface.

After the first injection is done, the needle should be withdrawn about 3 mm and the tip position recorded again in an AP view before the second injection is made.

Third Occipital Nerve Blocks

The C2-3 zygapophysial joint is not supplied by two medial branches, as are those below it, but by a single nerve, the third occipital nerve on that side. Hence, test blocks of the C2-3 joints are known as TONBs. The third occipital nerve runs backward across the C2-3 joint from somewhere between the top and the bottom of the intervertebral foramen, as

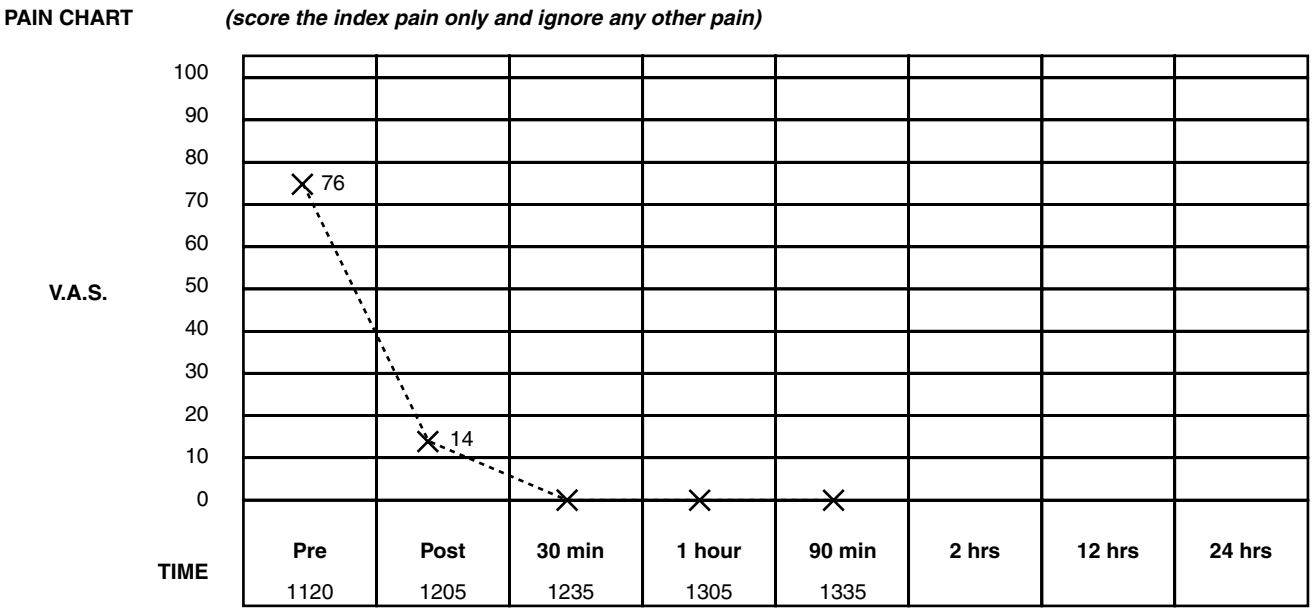

Figure 34.8 Pain chart showing the initial intensity of the index pain and the Visual Analogue Scale scores recorded after a medial branch block with a positive result. Courtesy of Pendlebury Clinic, Newcastle, Australia.

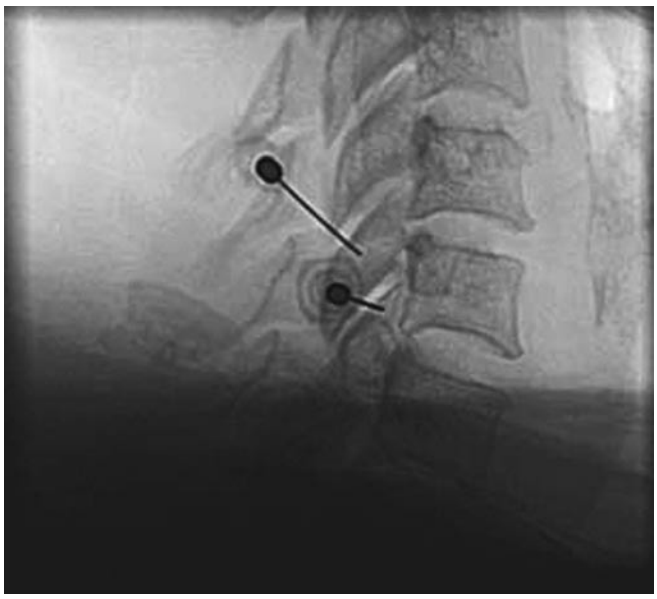

Figure 34.9 Lateral fluoroscopic view of the right C6-7 z. joint with the needle tips on bone over the midpoint of the C6 articular pillar and the peak of the C7 articular pillar, the target points for the C6 and C7 medial branches, respectively.

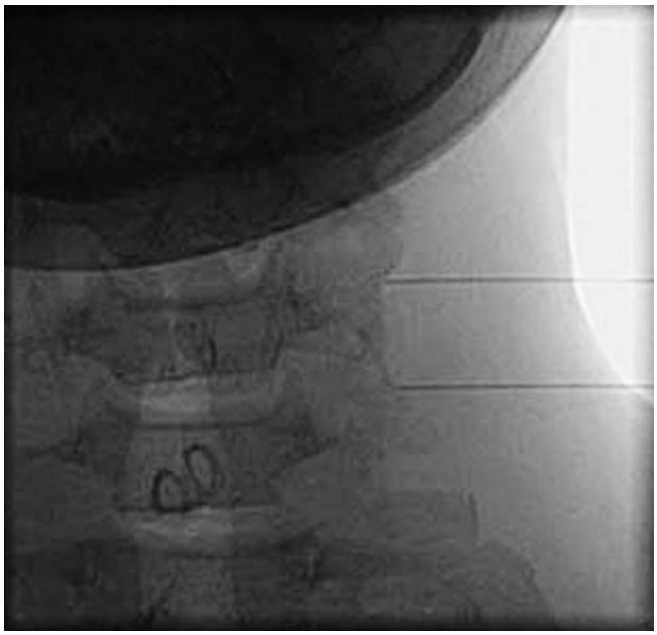

Figure 34.10 AP fluoroscopic view of the right C6-7 z. joint with the needle tips on bone at the target point for the C6 and C7 medial branches.

shown in Figure 34.4. It is blocked effectively by a series of three injections of local anesthetic at three target points: a high position just above the C2-3 joint space, a middle one on the capsule over the joint space, and a low one just below the joint space. As three doses of local anesthetic are used, a volume of 0.3 mL is injected at each of the three points.

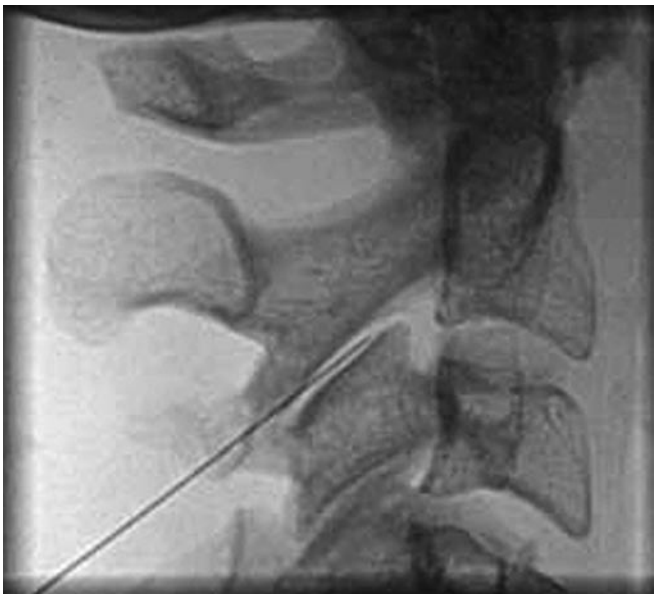

Figure 34.11 Lateral fluoroscopic view of the right C2-3 z. joint with the needle tip on skin over the middle target point for the third occipital nerve.

The technique is similar to that for lower cervical joints. The patient is placed on the table on their side with the painful side up. After skin preparation and draping, the operator and radiographer together should confirm the position of the target nerve and the radiographer should obtain a clear lateral view of the target region and cone to it. The operator should take a suitable needle and place its tip on the skin of the patient's neck directly over the target point, as in Figure 34.11.

The operator should then insert the needle and direct it through the muscles under fluoroscopic guidance until its tip is on bone or the joint capsule at one of the three target points. Its position there should be checked and recorded on a fluoroscopic image, as in Figure 34.12.

The first injection of 0.3 mL of local anesthetic should be made at that point. Then the needle tip should be moved to the other two target points in turn, checked, and recorded fluoroscopically there, as in Figures 34.13 and 34.14, and further injections of 0.3 mL made at each to complete the procedure.

The patient should then be taken from the fluoroscopic suite and any effects of the test recorded by real-time assessment, as described earlier. In the case of a TONB, there is an extra observation to be made. The third occipital nerve usually supplies a small area of skin behind the ear on that side. In the vast majority of cases, if the third occipital nerve has been blocked effectively, the patient will have a small area of numbness there. The nurse observer should ask about it and record whether or not such numbness occurs. The presence of numbness reinforces the likelihood of effective TONB; a lack of numbness suggests the block may not be effective but does not absolutely prove that, as in the normal range of anatomic variability there are people whose third occipital nerves do not innervate skin.

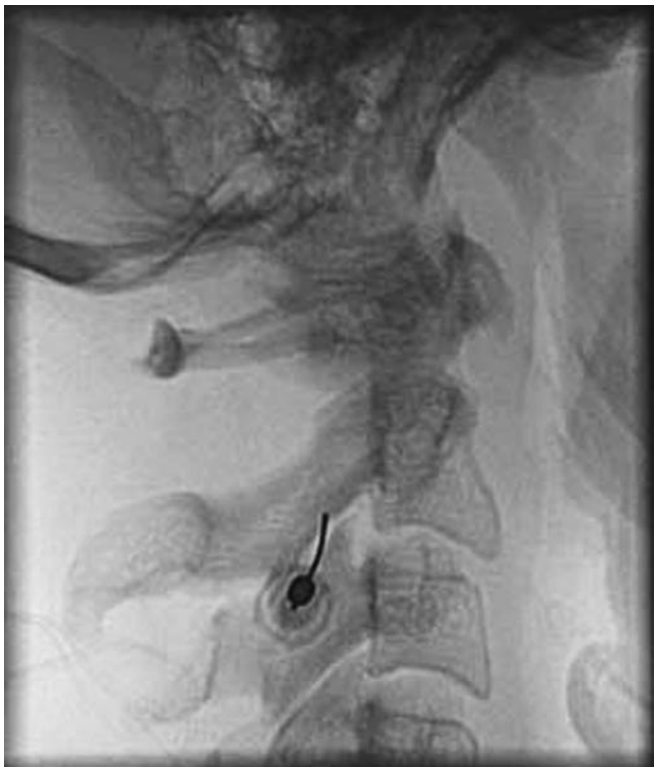

Figure 34.12 Lateral fluoroscopic view of the right C2-3 z. joint with the needle tip on bone at the lower high target point for the third occipital nerve.

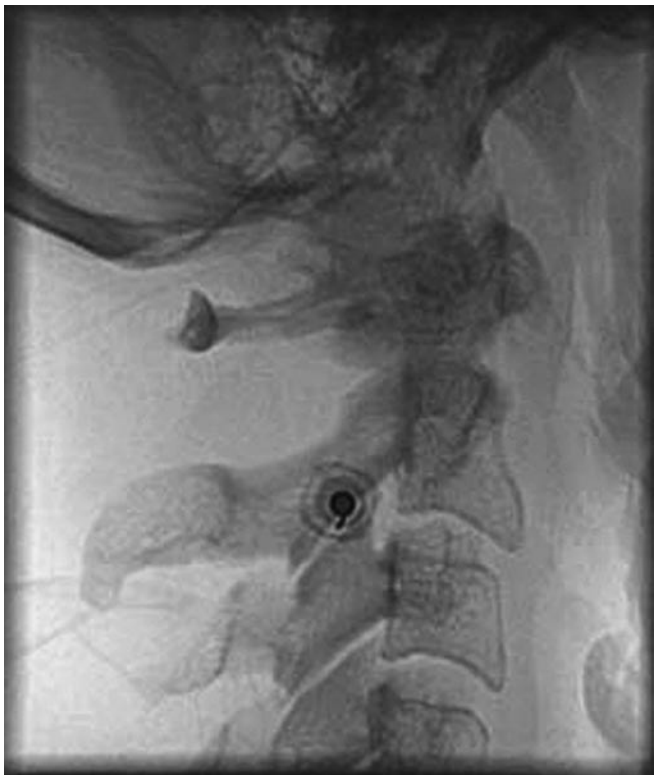

Figure 34.13 Lateral fluoroscopic view of the right C2-3 z. joint with the needle tip at the middle target point for the third occipital nerve.

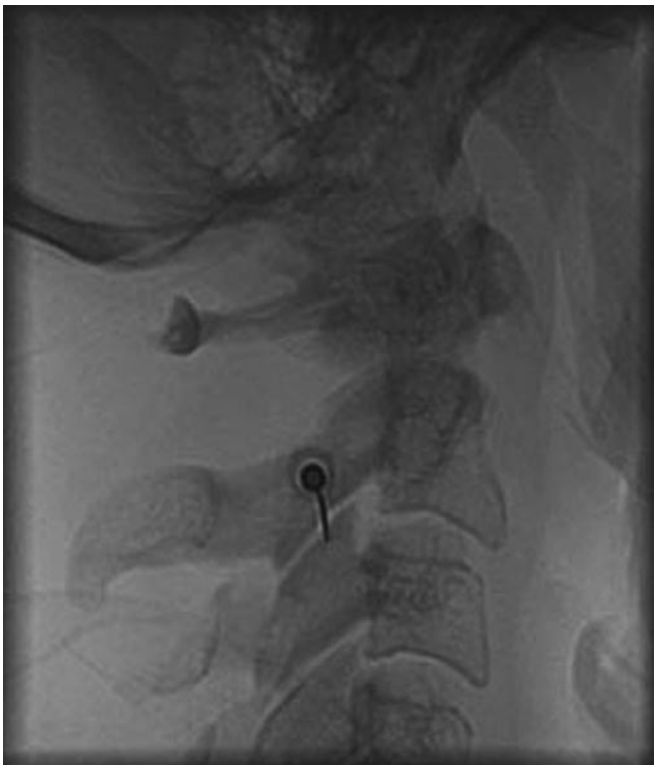

Figure 34.14 Lateral fluoroscopic view of the right C2-3 z. joint with the needle tip on bone at the low target point for the third occipital nerve.

Recording and Interpretation of Block Results

If block tests are to yield accurate diagnostic information, they must be done precisely and their results must be recorded in ways that address the potential liabilities of bias. Then the results must be interpreted carefully in the light of the data that are available on the diagnostic implications of particular outcomes.

Potential biases in the recording of outcomes include reporting bias (if the patient knows in advance what will comprise a positive response) and observer bias (if the recording nurse knows when the pain might be relieved and when it might begin to return). If the test is performed single-blind, meaning the patient is unaware of the anesthetic agent injected, the procedure is controlled for reporting bias. If a randomized, double-blind protocol is followed, with both patient and recording nurse unaware of the (randomly allocated) agent used, the method is controlled for observer bias too. Another potential bias is recall bias (when the patient has to report the outcome some time later, from memory, and has an inaccurate recollection of the outcome). Recall bias is obviated by what is called "real-time assessment," the procedure's effect (if any) on the index pain being recorded at the actual times after the blocks by a suitably trained, objective observer. Any other method of recording results, like letting the patient leave soon after the procedure and contacting them by telephone subsequently to record what they remember of the effects, will be biased by inaccurate memory, by the pain they will

have again after any effect of the block has worn off, and possibly by the patient's expectations and/or uncertainty about the investigative process.

Accurate interpretation of results depends on understanding the factors that contribute to particular outcomes and applying the standards set in the literature. If those factors are not understood or those standards are not applied, erroneous interpretation of responses may lead to inappropriate treatment of the wrong target, which not only will inevitably fail but will deny the patient the chance to obtain relief from what is often a debilitating condition.

An individual MBB or TONB test result is considered positive (only) if the index pain is abolished (i.e., the VAS score goes down to zero) in the postinjection period, as shown on the pain chart in Figure 34.8 [14]. Any other result, with postinjection pain scores of more than zero must be considered negative or inconclusive. Some may be tempted to call a test result positive if the pain is reduced substantially, such as (say) from 70/100 to 15/100, but the MBB procedure is essentially a test of a specific joint and that joint cannot be said to be the source of the index pain unless all the pain is abolished by an effective block of that joint's nerve supply. A reduction of the pain score may suggest the joint tested is partially responsible for the index pain or that the pain is actually from an adjacent joint, which has been partially blocked because it shares one of the medial branches targeted in the test, or that the result is due to some other factor.

Single MBB and TONB results do not provide specific diagnosis even if the procedure is done precisely and according to the double-blind protocol, because the tests are subject to both false-positive and false-negative results. In a study, the rate of false-positive responses to single MBBs was measured as 27% and the rate of false-negative responses as 5% [15]. These figures mean that even if the index pain is abolished after a single-block test, that positive response is only 73% likely to be dependable in identifying that joint as a pain source, and if the pain is unaltered after a single-block test, that negative response is only 95% likely to be trustworthy. The same study assessed the positive predictive value of analgesic responses to single blocks and found it varied with the prevalence of the condition in the cohort, but even when the prevalence was 70% a positive response to a single-block test would identify the problem in only 89% of cases. This is clearly not enough for a single-block test result to be considered sufficient for definitive diagnosis of CZJ pain.

A more discriminating test process, designated according to the elements it involves as "randomized, double-blind controlled, comparative MBBs (or TONBs)," overcomes the shortcomings of single blocks. It involves the administration of a second block, on another occasion, after a single-block test has had a positive result, to test the joint further and to check the first result. The second block should be done in a similar manner but using a different local anesthetic preparation with different pharmacodynamics. Again both the patient and the nurse observer should be blinded to the agent injected. The use of two different anesthetics on separate occasions produces a more complex range of responses than the binary options after a single block. If the result of the second test is also positive, the durations of relief resulting from each test can be compared with the expected durations of action of the anesthetic agents used, to add another objective dimension to the test process. If the index pain is found to have been abolished by each of two comparative blocks, and the duration of pain abolition was longer with the longer-acting anesthetic (bupivacaine) than with the shorter acting (lidocaine), that joint can be said to be identified positively as the source of the patient's neck pain [14].

The interpretation of double-block test results is straightforward when the results are both positive and the index pain is abolished for longer with the longer-acting anesthetic (bupivacaine) than with the shorter (lidocaine). The interpretation of negative results is straightforward too. A study of MBBs and TONBs showed that when two blocks are done, the responses can be classified into five categories according to the durations of effect and only two of those categories meet the criteria for positive diagnosis [16]. The groups are:

- *Concordant*: index pain abolished after each block and for longer after bupivacaine than after lidocaine; lidocaine effect lasting <7 hours and bupivacaine lasting <24 hours.
- *Concordant prolonged*: index pain abolished after each block and for longer after bupivacaine than after lidocaine; lidocaine effect lasting >7 hours and/or bupivacaine effect lasting >24 hours.
- *Discordant*: index pain abolished after each block but for longer after lidocaine than after bupivacaine; lidocaine effect lasting <7 hours and bupivacaine lasting <24 hours.
- *Discordant prolonged*: index pain abolished after each block but for longer after lidocaine than after bupivacaine; lidocaine effect lasting >7 hours and/or bupivacaine effect lasting >24 hours.
- *Discrepant*: index pain abolished after first block but not after second block.

The explanation of the last three categories is that some block results are false-positive, as outlined earlier. Accordingly, only double-block responses of the concordant and concordant prolonged types meet the criteria set by the ISIS for positive identification of a painful joint [14].

Again it is stressed that a response is considered positive only if all the requirements for a positive result are satisfied. Failure to fulfill all the criteria makes the test negative or inconclusive.

Evidence of Validity of Cervical MBBs and TONBs

The validity of diagnostic blocks (or of any other diagnostic test) means the extent to which the test determines what it is supposed to determine. More specifically, the validity of cervical MBBs (or TONBs) means the extent to which the block results determine whether the specific joint tested is, or is not, a source of pain. The preceding section on the interpretation of block results makes it clear that the results of a single-block test cannot be relied upon for accurate diagnosis. In other words, single MBB and TONB results are not valid for specific diagnosis even if the procedure is done precisely and according to a double-blind protocol.

Double-blind controlled, comparative MBBs and TONBs, performed according to the ISIS guidelines, are the criterion standard tests for diagnosis of CZJ pain and third occipital nerve pain, respectively [14]. They are valid for definitive diagnosis and their validity is well supported by sound scientific evidence. Validity can be assessed in three domains, termed face validity, construct validity, and predictive validity (which is related to therapeutic utility). All three domains contribute to the overall validity of any diagnostic test or measuring device.

Face validity refers to the appearance of validity; a test is said to have face validity if it appears to determine what it is meant to determine. Tests such as MBBs and TONBs, which depend on blocking conduction in particular nerves, have face validity if the test procedure results in blockade of the specific nerve(s) targeted and no other. In other words, the face validity of MBBs and TONBs depends on their target specificity. The issue was investigated in a study [13], which showed that when 0.5 mL of local anesthetic is injected over a medial branch in the manner described earlier, it will bathe that medial branch but will not spread to any other nerve (or other structure) of diagnostic significance. MBBs and TONBs are target specific. They have face validity.

A study of injectate placement in MBBs showed that intravascular injection occurred in 3.9% of cases in that series [17]. That would possibly account for some of the 5% of false-negative responses to blocks. The authors of the study suggested using contrast medium to check injectate spread. To do so would increase confidence of correct placement, and may be useful in a research setting. Contrast use is not considered necessary in everyday practice because it would add to the risks of the procedure (because of possible allergy to contrast) and would dilute the local anesthetic injectate (and so may actually increase the rate of false-negative responses, the very thing it is designed to reduce). Also, contrast use would not rule out all false-negative responses, which occur for reasons including, but not limited to, incorrect placement. Moreover, the study showed more than 96% of block injections in the series were target specific. MBBs and TONBs have face validity whether contrast is used or not.

Construct validity refers to the degree to which the interpretation of a test result reflects the theoretical construct of the phenomenon being tested. In relation to tests such as MBBs and TONBs, the construct is that theoretically a z. joint which is generating pain can be identified by blocking the nerve(s) along which pain is transmitted from the joint. The construct validity of MBBs and TONBs depends on the extent to which positive or negative block test results reflect that the joint tested is, or is not, actually a pain source. The issue hinges on whether the results of double-block tests done according to the double-blind, comparative protocol allow the clinician to discriminate between true responses and false responses. This was the issue addressed in the study of outcomes [16] quoted in the preceding section on interpretation of results, the study that described five categories of responses to positive blocks. That study showed false-positive responses can be identified or at the least excluded by the double-blind controlled, comparative block protocol. MBBs and TONBs done in that way do have construct validity.

A more stringent protocol, using injections of saline as placebo controls in addition to the two anesthetic agents, has been shown [18] to be even more discriminating. Saline controls are useful in exploring the significance of analgesic responses of durations not concordant with the agents used and in the clarification of false-negative responses. However, the advantages of the saline control method over comparative blocks are not great and the injection of placebos for such purposes raises ethical questions. The administration of a third block also adds to the logistic and financial burdens of the investigative procedure. Placebo-controlled blocks have some uses in research and for elucidating complex medico-legal issues. In most patient management situations, randomized, double-blind controlled, comparative MBBs and TONBs provide definitive diagnosis of CZJ pain [14].

Predictive validity refers to the value of a test in predicting responses to treatment. It is the aspect of validity of greatest relevance to the clinical application of the test and is directly related to therapeutic utility. The predictive validity of MBBs and TONBs depends on whether they lead to effective treatment and, if so, whether they accurately predict response to that treatment. An effective treatment for zygapophysial joint pain is radiofrequency medial branch neurotomy; several studies of that procedure, done on the indication of positive responses to double-blind, comparative MBBs and TONBs, have shown that positive double-block results do lead to effective treatment and are predictive of satisfactory outcomes of the treatment [12,19,20]. MBBs and TONBs when performed in accordance with the ISIS guidelines do have predictive validity.

Validity cannot be claimed for blocks that are not performed in accordance with these guidelines. The foundation literature on which the ISIS guidelines are based refers to blocks performed in particular ways, the ways described earlier in this chapter. The scientific evidence on such blocks cannot be generalized to blocks done in other ways. Whether blocks that are not performed in accordance with the ISIS guidelines are valid or not is simply unknown.

Intra-Articular Blocks

Injection of a suitable dose of a local anesthetic agent into the cavity of a joint will block any pain from the joint structures. An advantage of the method is that it is specific to the joint in question, so long as the injection is confined to the inside of the joint capsule and that capsule is intact. A disadvantage of the method is that the intrusion of the needle into the joint may injure the articular cartilage, a meniscoid, or other soft tissue structures of the joint.

Because of its relatively invasive nature, IAB has largely been superseded by less invasive articular nerve blocks and they are generally preferred for testing the joints from C2-3 down. However, IAB is the method of choice for testing the lateral atlantoaxial joints, the synovial joints at the C1-2 level, as their articular nerves are not amenable to specific neural blockade.

Lateral Atlantoaxial (C1-2) Joint Blocks

Lateral atlantoaxial joint blocks are useful for the investigation of upper neck pain and headache, which are suspected on clinical grounds as being of joint origin. The distribution of C1-2 joint pain has been described in the literature [8] as occurring in patches over the occipital region and other parts of the head up to the vertex, the most common site being at the base of the occiput, as illustrated in Figure 34.15.

Patients may be selected for lateral atlantoaxial joint blocks if they have chronic pain in a distribution like that shown in Figure 34.15 and a history suggesting injury of the C1-2 spinal segment. As pain from C2-3 and C3-4 z. joints is more common [3,4] and the distributions of pain referral from those upper z. joints overlap that from C1-2, it makes sense to test the C2-3 and C3-4 joints first, but if those tests are negative or if there is residual pain in the head after C2-3 and/or C3-4 joint pain has been treated effectively, C1-2 blocks are indicated.

Anyone considering injection of a lateral atlantoaxial (C1-2) joint should be aware of two significant structures that reside in close proximity to the target joint. The vertebral arteries lie immediately lateral to the C1-2 joints in most people and in some cases (in the normal range of anatomic variation) lie posterior to the lateral aspects of those joints. The dorsal ramus of the C2 spinal nerve passes inferolaterally across the lower part of the back of the lateral C1-2 joint and usually lies just behind the superior articular process of the C2 vertebra. When passing a needle toward a lateral C1-2 joint from behind, care must be taken to keep its tip over the middle third of the coronal aspect of the joint, and above the upper margin of the superior articular process of C2, so as to avoid these two structures.

IABs of the lateral atlantoaxial (C1-2) joints are done using a posterior approach. The patient is positioned prone on the table and with their neck flexed and their chin supported comfortably. After antiseptic skin preparation and draping of the procedural area, the radiographer should obtain an AP view of the patient's upper neck and cone to it. In that view the lateral atlantoaxial joints will be seen as ellipsoid structures on either side of the base of the odontoid process. The operator and the radiographer should then identify the image of the arch of the axis, which tends to lie over the C1-2 joints in the AP view, and angle the fluoroscopic beam so the arch of the atlas moves up away from the central part of the target joint. That will enable a clear view of the upper edge of the C1-2 joint in its middle third, which is the initial target area. The operator should then take a suitable spinal needle and place its tip on the skin of the patient's neck over that target point, as shown in Figure 34.16.

Keeping the needle tip at the insertion site, the operator should stand the needle up in line with the fluoroscopic beam, warn the patient of a slight prick, and pass the needle through the skin as smoothly as possible. After insertion, the needle's position should be monitored as it is guided through the patient's neck muscles toward the target; intermittent AP fluoroscopic views should be used to keep the needle tip over the target zone and lateral views used to check the depth of penetration (as in Figures 34.17–34.19).

When the needle tip reaches bone at the initial target point, on the middle third of the inferior articular process of C1 near the upper margin of the C1-2 joint, its position there should be checked and recorded on both AP and lateral fluoroscopic images, as in Figures 34.18 and 34.19.

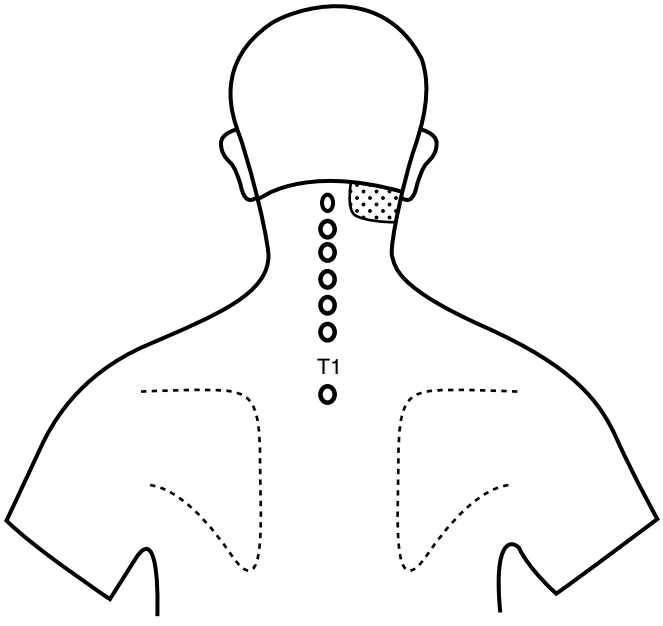

Figure 34.15 Pain map showing pattern of distribution of lateral atlantoaxial (C1-2) joint pain. From ref. [8].

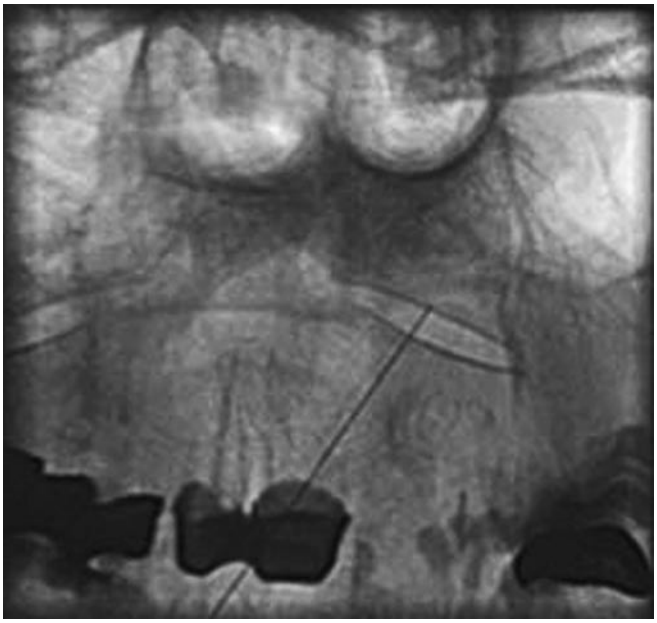

Figure 34.16 The anteroposterior fluoroscopic view required for an intra-articular block of the right lateral atlantoaxial (C1-2) joint. Note the arch of the atlas, above and clear of the superior process of the joint in its middle third (the initial target area) and the needle tip on skin over the initial target point.

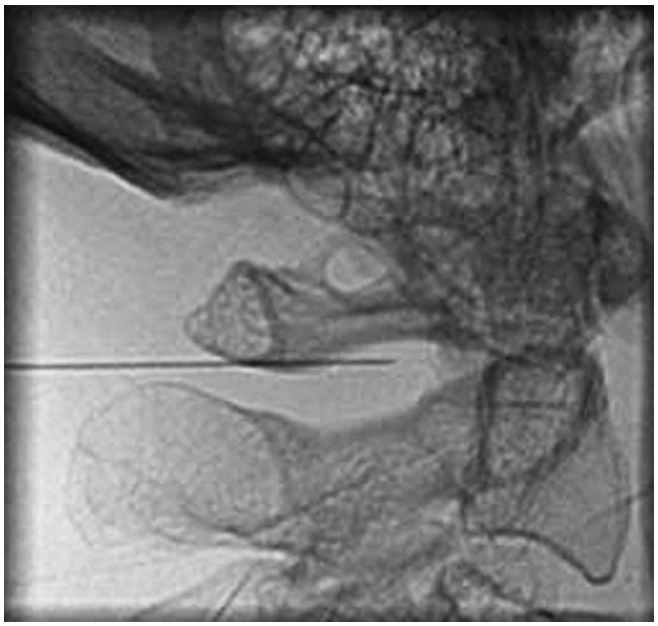

Figure 34.17 Lateral fluoroscopic view of the right lateral atlantoaxial (C1-2) joint showing the needle tip passing under the arch of the atlas and on track toward the initial target point for an intra-articular block of the joint.

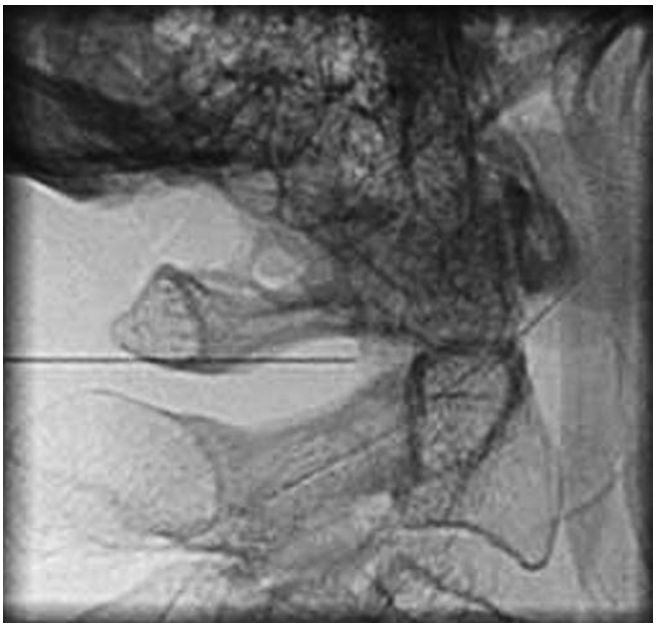

Figure 34.19 Lateral fluoroscopic view of the right lateral atlantoaxial (C1-2) joint with the needle tip on bone at the initial target point for an intra-articular block of the joint.

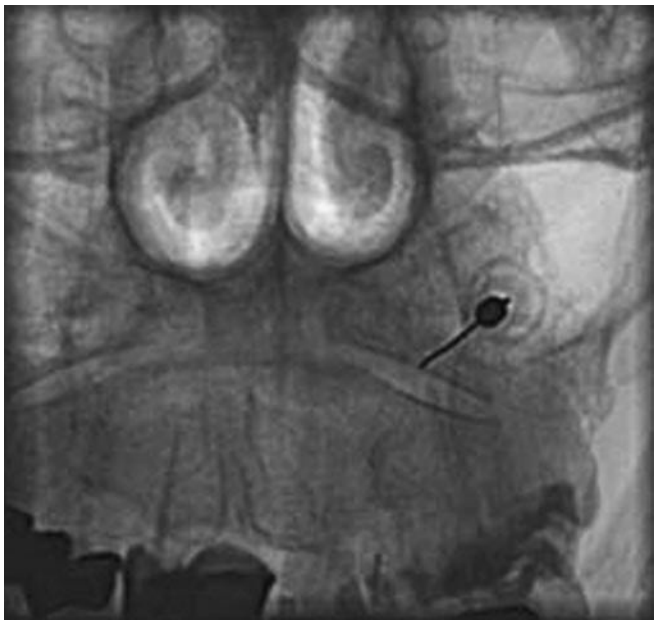

Figure 34.18 Anteroposterior fluoroscopic view of the right lateral atlantoaxial (C1-2) joint with the needle tip on bone at the initial target point for an intra-articular block of the joint.

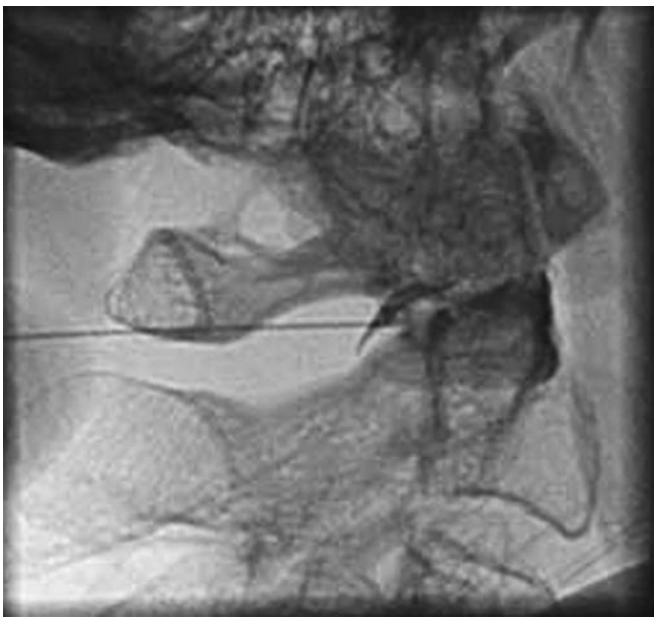

Figure 34.20 Lateral fluoroscopic view of the right lateral atlantoaxial (C1-2) joint after injection of contrast medium, showing the arthrogram produced to confirm intra-articular placement of the needle tip.

The needle should then be withdrawn very slightly and directed downward into the joint cavity.

The operator will feel a distinct sensation (appreciated by experience) as the needle passes through the capsule into the joint cavity. Intra-articular placement should be checked on a lateral fluoroscopic view and then confirmed by the injection of a small volume (about 0.2 mL) of contrast medium to produce an arthrogram (Figure 34.20).

The fluoroscope should be rotated again and the arthrogram recorded on an AP view (as in Figure 34.21).

Then the operator should withdraw the plunger of the contrast medium syringe to suck as much of the contrast

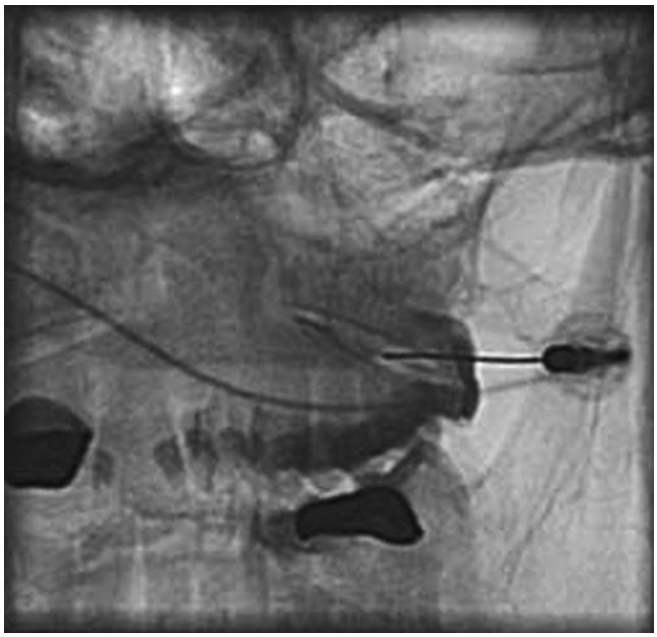

Figure 34.21 Anteroposterior fluoroscopic view of the right lateral atlantoaxial (C1-2) joint after injection of contrast medium, showing the arthrogram produced to confirm intra-articular placement of the needle tip.

medium as possible out of the joint because if left there it will reduce the volume of local anesthetic that can be injected. A short run of continuous fluoroscopic screening can be used to visualize the extraction of the contrast. After that about 0.5 mL of 2% lidocaine is injected slowly; the actual volume will be determined by the feeling of pressure the operator can sense as the joint cavity is infiltrated (this sense is something else that is learned by experience). It is important not to inject too large a volume as to do so might cause rupture of the joint capsule. Longer-acting local anesthetics such as bupivacaine should not be used at the C1-2 level because if such a drug should leak from the capsule it could cause prolonged high spinal block.

When the injection is complete the needle should be withdrawn smoothly and the patient should be advised the procedure is over. The drape should be removed and the antiseptic solution sponged from the skin and hair at the injection site. The patient should be asked about dizziness (which is more common after C1–2 blocks than after blocks at lower levels) and if they do feel lightheaded they should be reassured and left to lie quietly until the feeling passes. When they are not dizzy, they should be helped to sit up, being supported by the operator and/or nurse in case the change of posture makes them feel lightheaded. Then when they feel ready, the patient should be taken from the fluoroscopy suite to another room in the facility for postprocedural observation.

Atlanto-Occipital (C0-1) Joint Blocks

IABs of the atlanto-occipital joints are theoretically possible but are seldom done in practice. One reason for this is that the atlanto-occipital joints are rarely involved in pain generation because of their structure and biomechanics. Another reason is that although C0-1 blocks may have diagnostic value, they have little therapeutic utility. The technique of injecting a C0-1 joint is a highly specialized one carrying risks of damage to vital structures, including the vertebral artery, the internal jugular vein, and the vagus nerve, all of which lie adjacent to the joint. When a C0-1 injection is deemed necessary, and the risks worth taking, a lateral oblique approach is used with depth monitoring on an open-mouth AP view. A posterior approach must not be used because the vertebral arteries cross the atlanto-occipital joints posteriorly; any attempt to inject a C0-1 joint from behind carries a serious risk of injuring the adjacent vertebral artery, with potentially disastrous consequences. IABs of the atlanto-occipital joints should not be attempted by anyone without supervised rigorous training in the procedure under the supervision of an interventionist experienced in performing it. The principles are similar to those of other IABs described, but the details of the technique are beyond the scope of this work.

Interpretation and Validity of IAB Results

IABs were developed before articular nerve blocks (MBBs and TONBs) and their use in both experimental studies and clinical practice was largely curtailed by the introduction of the newer techniques. Hence, there are very few data that can be relied on for the interpretation of IAB responses. There is also not much hard scientific evidence of the validity of IABs, and what little there is must be construed in the light of what has emerged about false-positive and false-negative responses to MBBs and TONBs, as several of the factors that affect articular nerve blocks are likely to be relevant to IABs too.

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35 Therapeutic Intra-Articular Cervical Facet Joint Injections

Sarjoo M. Bhagia

INTRODUCTION

Chronic neck pain is common in the adult general population, with a lifetime prevalence of 26% to 71% [1,2]. Specific spinal structures such as nerves, nerve root ganglia, uncovertebral joints, intervertebral discs, facet joints, ligaments, and even muscle may give rise to neck pain symptoms [3,4] (see Chapter 1). Formulating a probability analysis of the structures involved is the most important step in the diagnostic and therapeutic algorithm for neck pain (see Chapter 29). The successful treatment of neck pain requires a multifactorial approach using pharmacologic, nonpharmacologic, manipulative, anesthetic blockade, therapeutic injections, and occasionally surgical interventions. In the current health care environment with focus on evidence-based medicine and health care costs, it is difficult to recommend any intervention in the absence of consistent, strong evidence with respect to benefits, risks, and costs. Although this view is understandable from a purely scientific and third-party payer perspective, it cannot easily be reconciled with the daily practice of health care in which clinician and patients attempt to make informed decisions using the best available evidence with the common goal of symptom relief and improvement of quality of life. This chapter will review the role for intra-articular therapeutic facet joint injections, evidence and knowledge-based analysis of the utility of therapeutic intra-articular injections including corticosteroids, and viscosupplementation.

NEUROANATOMY AND PATHOPHYSIOLOGY

The zygapophyseal joints are implicated as a major source for chronic neck pain. The prevalence of zygapophyseal joint pain after whiplash has been reported as high as 50% in the C2-3 joint and 49% in the lower cervical joints [5,6]. Yin and Bogduk [7] demonstrated the prevalence of zygapophyseal joint pain in 55%, and lateral atlantoaxial joint pain in 9%, of 143 patients with chronic neck pain in a private practice pain clinic in the United States.

The facet joint is a diarthrodial-synovial joint comprising articular cartilage and rheological synovial fluid, encased by an inner synovial membrane and outer joint capsule. Histological studies have confirmed the presence of nociceptive C-type fibers on both the synovial membrane and joint capsule, as well as group III high-threshold, slow-conducting mechanosensitive, somatosensory units, within both the articular and subchondral cartilage [8]. Thus, the synovial membrane, joint capsule, and articular and subchondral cartilage have capabilities of transmitting pain. The medial branch of the dorsal ramus above and below its location innervates the facet joints, except C2-3. The C2-3 joint is innervated by the superficial medial branch of the C3 dorsal ramus, also known as the third occipital nerve [9–12].

Pain patterns from stimulation of the cervical zygapophyseal joints have been studied in normal volunteers [13] and from clinical evaluation [14,15]. These studies suggest that the cervical zygapophyseal joints produce characteristic pain patterns according to the segmental distribution [13,14]. Dwyer et al. distended the C2-3 to C6-7 zygapophyseal joints in asymptomatic patients to report that only the C2-3 zygapophyseal joint referred symptoms to the head [13]. Aprill et al. investigated the C2-3 to C7-T1 zygapophyseal joints and reported that the C2-3, C3-4, and C4-5 zygapophyseal joints could refer to the head [14]. Dreyfuss et al. stimulated the atlanto-occipital and atlantoaxial joints by injection of radiopaque contrast with subsequent distention of the joint capsule. Both joints tended to refer pain into the ipsilateral occipital or suboccipital area, with possible referral into the face [15]. Bogduk et al. performed medial branch and zygapophyseal joints blocks in 24 symptomatic patients from C1-2 to C5-6 levels and reported that the C2-3 zygapophyseal joints refer to the head and face, whereas the C3-4 zygapophyseal joints only refer to the head [16]. Slipman et al., reviewing data from 100 patients, demonstrated that C4-5 and C5-6 zygapophyseal joints can refer pain to the head and the C1-2, C2-3, and C3-4 zygapophyseal joints can also refer pain to the face [17].

DIAGNOSTIC TESTING

Cervical spine radiographs are not a sensitive method for diagnosing facet-mediated pain, because no specific radiologic abnormalities are usually found [18]. In patients with a history of whiplash injury or traumatic insult, cervical flexion and extension radiographs should be obtained. Although MRI has a high sensitivity to detect facet joint arthropathy, there are concerns about its clinical specificity [19]. If the patient is claustrophobic, then

a high-quality open MRI is obtained. If this is unavailable, then a multiplanar computed tomography (CT) scan is requested.

Diagnostic anesthetic blockade is required to confidently render a diagnosis of facet-mediated pain [20] (see Chapter 34). Fluoroscopic guidance with contrast is necessary to assure accurate and specific localization of the pain source [21–23]. Diagnostic blocks use small amounts of local anesthetics that are infused into or around the suspected structure with the goal of temporarily interrupting pain. Because diffusion of the anesthetic to adjacent structures would muddle the results, it is essential to use the least amount of local anesthetic feasible to limit the anesthetic affect to the target site. Although this approach is quite appealing, it does not offer 100% accuracy [20,24]. False-negative and false-positive responses to diagnostic blocks do occur as a result of technical failure, placebo response, administration of sedative agents before or during the diagnostic block, concurrent pharmacologic treatment, and secondary psychosocial factors. The Bone and Joint Decade 2000–2010 Task Force on Neck Pain and its Associated Disorders concluded that there is no evidence to support the diagnostic validity or utility of anesthetic facet or medial branch blocks [25]. However, systemic review of literature reveals evidence supporting the use of these diagnostic techniques, as presented later in the chapter.

TREATMENT

The Bone and Joint Decade 2000–2010 Task Force on Neck Pain and its Associated Disorders, recently concluded that spinal manipulation therapy, mobilization, and exercise therapy could all be considered for the treatment of facet joint–mediated pain[26]. Interventions that focused on regaining function and returning to work as soon as possible are relatively more effective than interventions that did not have such a focus [27]. The Task Force compared quality-of-life years (QALYs) associated with standard nonsteroidal anti-inflammatory drugs (NSAIDs), Cox-2 NSAIDs, exercise, manipulation, and mobilization. None of the treatments was found to be clearly superior to any other in the short or long term, when estimates of the course of neck pain, adverse event risks, treatment effectiveness and risk, and patient-preferences for health outcomes were considered [28]. Noninterventional treatments alone are often ineffective or provide only modest benefit. Positive diagnostic blocks, as outlined in diagnostic testing, may direct treatment toward more invasive interventional or neuroablative therapy.

Confirmation of joint involvement in facet-mediated pain is through unequivocal relief of pain after the local anesthetic block of the joint, by intra-articular injection or medial branch blocks. There are reports of a high false-positive results of 27% to 63% [29,30] with a single diagnostic facet block. Thus, to maintain the accuracy of diagnosis, facet joint blocks must be performed under controlled conditions, either with placebo or with controlled comparative local anesthetic blocks using two local anesthetics of different durations of action. Controlled comparative local anesthetic blocks are easier to implement in conventional practice compared to the placebo-controlled blocks, especially in the United States [31]. Further, when compared with placebo-controlled blocks, comparative local anesthetic blocks have been shown to have less false-positive rates [32]. The outcome measurement needs to be appropriate providing significant pain relief (more than 80%) and an outcome of the ability to perform previously painful movements with sustained pain relief. If the double block is negative, the next suspected structure in the diagnostic algorithm should be assessed. When upper cervical facet joint syndrome (CFJS) is suspected, diagnostic blocks are performed sequentially at C2-3, C3-4, and C1-2 levels, until the offending site is identified. This sequence is based on clinical experience and is supported by epidemiological studies [33].

There is a strong evidence for diagnostic accuracy of cervical facet joint blocks, per the systematic review by Sehgal et al. [30]. In addition, Rubinstein and van Tulder [34] in a best-evidence review of diagnostic procedures for neck and low back pain concluded that there was strong evidence for the diagnostic accuracy of facet joint blocks in the diagnosis of neck pain. However, a significant controversy surrounds various treatments used in the management of chronic neck pain arising from cervical facet joints [1,35–42] even though diagnosis has been well established. Thus far, the evidence for long-term therapeutic benefits of intra-articular injection of facet joints is limited [34,36,42], for medial branch nerve blocks is promising [35–37,43,44], and evidence for radiofrequency (RF) neurotomy is moderate to strong [36–38,45–48] (see Chapter 36). Despite this lack of evidence-based support, cervical facet joint interventions for managing chronic neck pain are one of the most commonly performed interventions in the United States [49,50].

Therapeutic Injections

If a diagnostic facet joint double block is positive, fluoroscopically guided therapeutic intra-articular steroid injections may be offered. However, evidence is lacking to support routine use of intra-articular steroid injections [27,43]. Barnsley et al. investigated the effectiveness of intra-articular corticosteroid injections for chronic pain in the cervical zygapophyseal joints [43]. Less than half the patients reported relief of more than 1 week and less than one in five patients reported relief for more than a month. They concluded that intra-articular injections with betamethasone were an ineffective treatment for pain emanating from the cervical zygapophyseal joints. However, this study used one outcome measure, that is, verbal pain score, and only evaluated the efficacy of one intra-articular steroid injection per joint without restricting physical activities or physical therapy.

Conversely, Slipman et al. [51] demonstrated good to excellent results in 61% of patients treated with intra-articular steroids who experienced daily unremitting headaches stemming from the C2–3 facet joint subsequent to a whiplash injury. In that study, the average duration of symptoms was 3 years and no patient obtained relief with any analgesics prior to the injections. Most patients received one to three injections per joint. Although the change in average pain score (5.5 at follow-up compared

with 8.2 at the time of initial presentation) does not seem to be a significant clinical difference, the frequency of patient's headaches and their responsiveness to analgesic use were clearly improved. Patients with previous employment restrictions were observed to return to full-time work status. During treatment, patients in this study [51] were advised to avoid forceful, rapid, or sustained cervical extension or rotation whenever possible. The basis for such a strict protocol is the observation that CFJS, especially when associated with whiplash injury, may be associated with subchondral fractures [52,53], joint capsule ruptures [54,55], and intra-articular hemorrhages [54,56]. These structural insults may be responsible for triggering zygapophyseal joint headaches when stressed by overactivity or exercise. When the symptoms are reduced, the patient gradually returned to engaging in normal physical tasks rather than letting the patient participate in unregulated physical activities. If a patient experiences greater than 80% relief of symptoms after a therapeutic intra-articular facet injection that lasts until the date of a planned subsequent injection, then the second intervention is cancelled. Such relief typically heralds the onset of continued symptom relief provided the patient adheres to specific activity prohibitions and patiently returns to a normal activity level. As previously alluded, this regimen is conducted under direct physician supervision and must be individualized.

Folman et al. [57] compared the pain relief with intra-articular steroids in patients with zygapophyseal joint osteoarthritis versus pain relief in patients similarly treated following whiplash neck injury. The mean time for relapse of 50% of the preinjection level of pain was 12.47 ± 1.89 weeks in patients with zygapophyseal joint osteoarthritis, compared to just 3 days in patients with whiplash neck injury. They concluded that selective blockade of zygapophyseal joints may be offered as an adjunct for diagnostic and therapeutic purposes for patients with chronic neck pain due to facet osteoarthrosis. However, the study had numerous limitations, including the small number of patients (30 patients), relying on a single diagnostic block to establish the diagnosis of facet joint–mediated pain, and the short follow-up of less than 6 months.

Kim et al. [58] assessed the effects of steroid injections into the cervical facet joints in patients diagnosed with myofascial pain syndrome, cervical herniated nucleus pulposus, and whiplash-associated disorders. They concluded that the analgesic effect of steroid injection into the cervical facets lasted longer in patients with associated cervical herniations, than in patients with myofascial pain syndrome or whiplash-associated disorders. The authors in this study relied on imaging studies for diagnosis of facet joint–mediated pain rather than use of controlled diagnostic blocks, which raises questions regarding the reliability of their diagnosis.

Although the use of steroid injection for facet-mediated pain is controversial, patients who have responded to diagnostic/therapeutic blocks of the zygapophyseal joints with unequivocal but unsustained relief of head and neck pain may be good candidates for RF neurotomy of the medial branches of the dorsal rami supplying the involved facet joint (see Chapter 36).

Atlanto-occipital and Atlantoaxial Joints

The atlanto-occipital and atlantoaxial joints are involved, respectively, in the flexion-extension and horizontal rotation of the head. These two joints are innervated by the C1, C2, and C3 spinal nerve roots and can be a source of neck pain [9,14,16]. Racz et al. [59] reported that the atlanto-occipital and atlantoaxial joint headaches are rarely seen and frequently misdiagnosed. Aprill et al. [60] tested the hypothesis that C1-2 headaches are a rare entity. Thirty-four patients with suspected C1-2 pain underwent diagnostic blocks of the joint with a local anesthetic and steroid. Twenty-one patients obtained complete relief of headache. Pain relief lasted for the duration of the injected local anesthetic. The overall incidence of carefully selected patients who had pain in the occipital or suboccipital region resulting from atlantoaxial joint pain was reported as 16%.

The technique for injecting these joints has been described by Racz et al. [59] and Dreyfuss et al. [61]. Potential complications include: (1) injury to the brain stem, vertebral artery, or spinal cord; (2) intravascular injection of anesthetic or steroids that results in central nervous system toxicity or stroke; and (3) inadvertent epidural and intrathecal injections. Because of the close proximity of these joints to major neural and vascular structures, these procedures should only be performed by physicians who have great experience in the use of fluoroscopic-guided injection techniques.

Viscosupplementation

Although intra-articular steroid injection into the facet joint is directed toward altering the inflammatory pain pathway and/or stabilizing neural membranes, another strategy to counter facet joint osteoarthritis (FJA) related pain may be to counter or reverse the pathophysiology of FJA with reparative therapy using viscosupplementation. Intra-articular viscosupplementation represents a potentially useful biologic treatment option aimed at repairing tissue injury and dysfunction, thereby countering the effects of the arthritic condition itself. Animal and clinical studies of exogenous hyaluronic acid (HA) have demonstrated various physiological effects that implicate its ameliorative capabilities to counter osteoarthritis. Viscosupplementation is the injection of an elastoviscous HA solution into a synovial joint. Intra-articular administration of HA has been used for degenerative osteoarthritis for more than 35 years. Injection of HA has demonstrated safe and effective results in the hip [62], knee [63], ankle [64], sacroiliac [65], patellofemoral joint [66], and, more recently, lumbar facet joints [67–69]. However, other than in the arthritic knee, its use in these joints is off-label.

An extensive review of peer-reviewed published medical literature did not reveal any study investigating the efficacy of intra-articular HA injection into the cervical facet joints. It is likely that the effectivity and complications associated with HA injection in the cervical facet joints would be similar to the lumbar facet injections. A randomized, controlled, blind-observer clinical study demonstrated equivalence between CT-guided intra-articular sodium hyaluronate compared with intra-articular glucocorticoids

for low back pain diagnosed as FJA-mediated solely by imaging findings [67]. By not utilizing controlled diagnostic lumbar facet joint blocks, these investigators likely enrolled numerous patients who were not experiencing lumbar facet–mediated pain. In a more recent study, Cleary et al. evaluated the utility of intra-articular HA, concluding no benefit of viscosupplementation in the management of symptomatic lumbar FJA [68]. However, Cleary et al. performed only one HA injection, whereas three to six weekly injections have been most widely studied [70], and did not use diagnostic blocks to confirm facet joint symptoms. Consequently, patients without painful lumbar FJA may have been enrolled, and, if FJA patients were enrolled, each may have undergone a subtherapeutic schedule of viscosupplementation. DePalma et al. [69] performed a prospective uncontrolled pilot study, with 15 patients enrolled, to investigate the clinical safety, efficacy, and utility of intra-articular lumbar facet injection of HA. They concluded that viscosupplementation for lumbar FJA is associated with modest efficacy that lasts up to 6 months. The noted improvements in Visual analog scale (VAS), analgesic use, Oswestry Disability Index (ODI), SF-36, finger to floor distance (FTF), and sitting tolerance were not sustained at 12 months. There was no evidence of changes over time in standing or walking tolerance, which could be a reflection of persistent loading of the arthritic facetal cartilage in these weight-bearing positions. The limitations of this study include a small sample size and lack both a control and blinding. The authors also noted one adverse event of transient S1 radiculopathy. This transient nerve root dysfunction may have occurred because of incidental extravasation of HA through a ventral fissure in the capsule. Intra-articular zygapophyseal joint contents can access the anterior epidural space, conceivably triggering an inflammatory radiculopathy [71]. This has profound significance while considering injection at the cervical facet joints because of the proximity of cervical cord and the limited space available for the cord under normal conditions.

TECHNIQUE

Cervical facet injections should always be performed under x-ray fluoroscopy guidance. There are two main approaches that can be used to perform cervical facet injections—a direct lateral approach (author preferred) and a posterior lateral approach.

For the direct lateral approach, the patient is placed in a lateral decubitus position with the painful side up and a folded towel or sheets under the head. It is preferred to have the patient's head slightly flexed laterally away from the suspected facet joint so as to open up the joint space and make intra-articular needle placement easier. The patient's cervical skin is sterilely prepped and draped. The appropriate facet joint is identified under fluoroscopy guidance, counting down from C2, which is easily recognized. The overlying skin and subcutaneous tissue are anesthetized with approximately 1 to 2 cc of 1% Xylocaine. A spinal needle (preferably a 25-gauge 2.5-inch spinal needle) is advanced toward the facet joint via a direct lateral approach. Needle insertion is performed with gentle, small incremental advances while observing under fluoroscope intermittently. Needle depth is assessed by contacting the inferior articulating border of the facet joint. Once the depth of the needle is assessed by contacting the bony edge, the needle can then be drawn back by a millimeter or two and redirected cephalad into the targeted facet joint. The needle position should be evaluated in anteroposterior and lateral planes to ensure proper needle position and ascertain that the needle has not passed through the joint capsule and into the spinal canal (Figure 35.1). A minimal amount of iodinated contrast (0.1–0.5 cc) can be used to confirm needle disposition into the joint. The intra-articular spread of the contrast dye without vascular uptake is documented by taking spot films under fluoroscopy (Figure 35.1). The injectant solution is then slowly infiltrated into the joint under real-time fluoroscopy. The needle is withdrawn and the punctured site is dressed.

The second approach, the posterior lateral approach, is technically more difficult for most interventionalists. The patient is placed prone on the table looking to the opposite side. The appropriate facet joint is identified under fluoroscopy guidance. The needle puncture site is located along the posterior lateral skin surface two or three levels below the intended facet joint. A 22-gauge 3.5-inch spinal needle is advanced in the caudocephalad direction under fluoroscopy guidance to enter the facet joint parallel to its oblique craniocaudal plane (Figure 35.2). The depth of the needle insertion is assessed by contacting the inferior articulating border of the facet joint. Once the depth of the needle is assessed by contacting the bony edge, the needle can then be drawn back by a millimeter or two and redirected into the facet joint. Confirmation of the needle position is obtained by injecting a minimal amount of iodinated contrast (0.1–0.5 cc) under real-time fluoroscopy, prior to injecting the injectant solution (Figure 35.2).

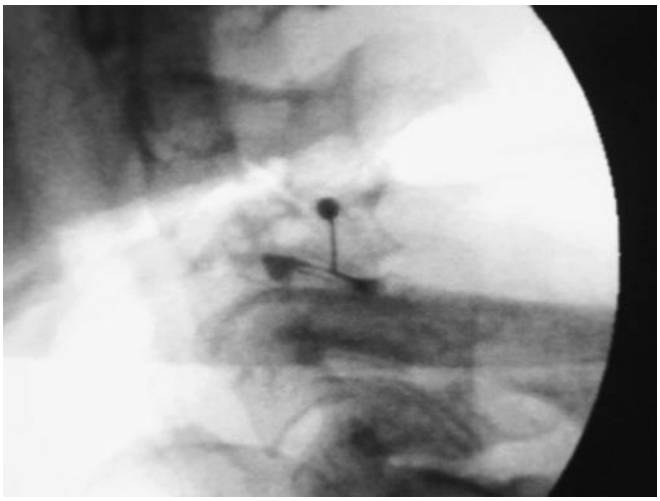

Figure 35.1 Cervical facet joint injection, lateral radiograph after insertion of the needle into the left C3-4 facet joint using a direct lateral approach.

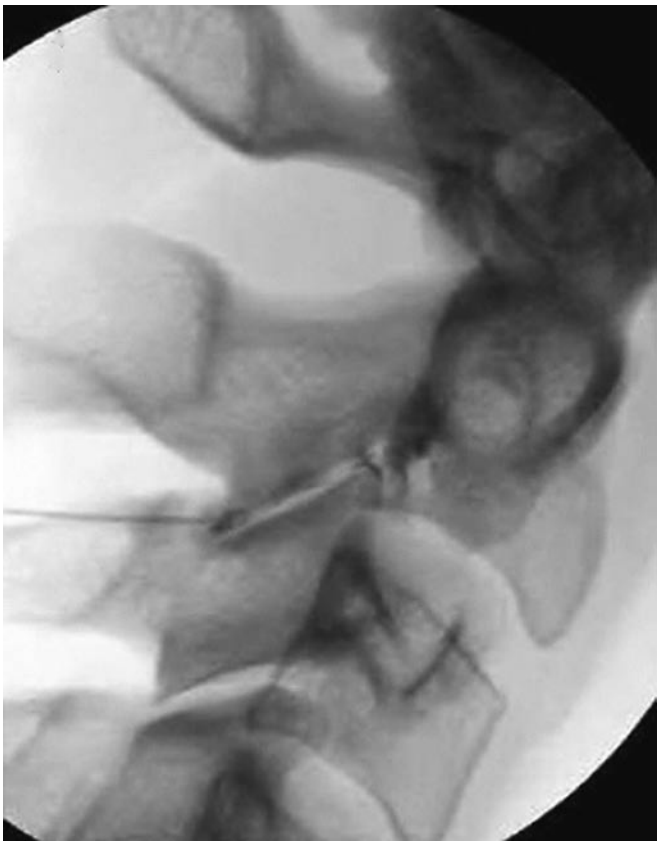

Figure 35.2 Cervical facet joint injection, lateral radiograph after insertion of the needle into the right C2-3 facet joint using a posterolateral approach.

CONCLUSIONS

Despite a growing body of literature on facet-mediated pain, there remains considerable controversy and confusion concerning diagnostic and therapeutic utility of facet joint injections. Anesthetic blockade plays an essential role in the diagnosis and treatment of facet-mediated pain. A positive or negative response to a diagnostic block must be considered in conjunction with the complexity of the patient with chronic neck pain, the placebo effect, and concurrent medical therapy before proceeding with more invasive interventional or neuroablative treatment. It is essential to have a profound knowledge of the anatomy of the cervical spine, an understanding of the pathophysiologic mechanisms of facet-mediated pain, and a high degree of technical skill before performing these procedures. Conservative therapeutic options should be attempted first. Acknowledging that conducting randomized, double-blind, controlled surgical trials are difficult, it is important that further studies are done if therapeutic injections are to become standard and accepted therapies for facet-mediated pain. Further investigation of a larger number of subjects in a controlled, randomized study will establish whether FJ injection of HA is safe. As the literature on this topic grows in volume and quality, the debate will intensify and, hopefully, will result in the clarification of the diagnostic and treatment algorithm for facet-mediated pain.

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36 Radiofrequency Denervation for Cervical Facet Joint Pain

Les Barnsley

INTRODUCTION

Neck pain is the cause of significant morbidity within the community. A large population-based study from Canada attests to a prevalence of 4% of disabling neck pain[1]. Notwithstanding the significant burden the quantity and quality of literature dealing with the management of chronic neck pain is, for the most part, unimpressive. Studies of conservative treatments have been summarized, and although there are some positive signals of benefit from various interventions, particularly exercise-based interventions[1], the effect sizes are small and do not match the patient's hopes for profound relief of their symptoms.

Against this background, there has been interest in establishing targeted interventions attempting to identify and treat pain from specific cervical structures. Foremost among these has been consideration of the role of the cervical zygapophyseal joints, otherwise known as cervical facet joints, and the etiology of chronic neck pain. This has been dealt with in detail in the preceding chapters but where validated diagnostic techniques, specifically controlled medial branch blocks, have been applied the prevalence of cervical zygapophyseal joint pain in groups of referred patients with chronic neck pain is substantial [2–4].

Once the diagnosis of cervical zygapophyseal joint pain has been established, the therapeutic options are unfortunately limited. The underlying pathology is not known. There have been no trials dealing with specific conservative interventions for a population of patients with proven cervical zygapophyseal joint pain. Corticosteroid injections into the zygapophyseal joints has been shown not to be any more effective than local anesthetic injections in achieving durable pain relief [5]. There are intriguing observations that medial branch blocks alone may produce some long-lasting pain. These are based on both anecdotes and published series and would accord with the author's experience in a small number of patients[3]. The mechanisms by which this occurs are unclear but could relate to improved patterns of movement following loss of pain or from the uncoupling of autonomous pain circuits within the spinal cord.

The leading contender for efficacious treatment for chronic zygapophyseal joint pain is cervical radiofrequency denervation targeting the medial braches of the cervical dorsal rami. An alternative name is radiofrequency neurotomy or RFN. The principle behind cervical RFN is that the nerves which convey nociceptive signals from the zygapophyseal joint are interrupted through the application of heat resulting in a long-term but reversible loss of function. Essentially, the nerves are "cooked" in situ denaturing the protein components of the neural tissue and hence anesthetizing the painful joint until such time as they recover.

DIAGNOSIS OF CERVICAL ZYGAPOPHYSEAL JOINT PAIN

It is axiomatic that targeted therapy can only be useful if the target is the correct one. In considering cervical RFN, this is particularly true. Fortunately, the diagnostic approach to cervical zygapophyseal pain gives a "dry run" of the radiofrequency denervation procedure. The targets of the diagnostic work-up are the medial branches of the cervical dorsal rami. Anesthetic blocks directed at these structures (Chapter 34) not only enable the source of pain to be inferred but simulate the benefit that may be procured from subsequent RFN. It is vital that these blocks are performed accurately and with appropriate technique to eliminate the significant false-positive rate that may ensue from placebo or nonspecific reactions to such blocks[6].

EVIDENCE FOR CERVICAL RADIOFREQUENCY DENERVATION

Radiofrequency denervation of the cervical dorsal rami has been formally tested in a randomized study which was published in the mid-1990s[7]. There have been no subsequent randomized controlled trials of this technique which have used appropriate diagnostic criteria. Where the quality of the study has been considered by third parties, the design has been considered strong and appropriate[8]. One of the difficulties that has arisen from there only being a single study is the way that this work has been reported in the literature which seeks to formally aggregate the results of clinical trials. Specifically, systematic reviews and meta-analyses tend to concentrate on the volume of concordant material in support of a given intervention to draw their conclusions. Consequently, despite

the fact that this trial was unequivocally positive and well designed, it is described as providing only "limited evidence" of the efficacy of cervical RFN[9]. It is vital that this phrase be deconstructed so that the true state of the evidence can be revealed. The term limited evidence does not refer in any way to the quality of the study. It does not refer to the strength of the finding, and it does not refer to the number of people who are likely to benefit from the procedure. Rather, it is a formal epidemiologic description detailing the number of concordant studies. It is, therefore, inappropriate to criticize the existing study for being the only one. It could be equally stated that all available clinical trials show a clear-cut positive benefit in favor of cervical zygapophyseal joint denervation over placebo. Another consequence of the applaudable desire to aggregate data to facilitate clinical practice has been that different procedures or procedures on different parts of the spine have been combined together[9]. This has meant that the procedure of cervical radiofrequency denervation has been lumped with other procedures for which there are inferior trial data or where there are technical problems relating to the performance of the procedures. This has the effect of diluting the message concerning the efficacy of cervical RFN. Presentation of the data in virtually any area of medicine is typically accompanied by the statement that more research needs to be done. This specific issue with regards to interventional spinal procedures was addressed in a recent editorial[10]. It concluded that the expense of such studies was often prohibitive, and there appears to be no natural funding body but that carefully conducted observational studies may be a solution.

The trial of Lord et al. studied the time to recurrence of significant symptoms (assessed by the patient to be greater than 50% of preprocedural pain). Once the patients' pain had returned, they were offered the active procedure. The control group received a sham operation, identical in all respects to the active intervention except that the electrode tip was not heated. Outcomes were assessed by a blinded assessor. The median time to the return of at least 50% of the preoperative level of pain was 263 days in the active-treatment group and 8 days in the placebo group, which was a statistically significant difference. Patients engaged in litigation were no more likely to report relief from pain than nonlitigants in either group.

Subsequent studies to the randomized trial of Lord et al. have provided additional data. Sapir et al. demonstrated the significant efficacy of cervical RFN in both litigants and nonlitigants[11]. Longer follow-up by Lord et al. of procedures performed outside their clinical trial demonstrated clinically satisfying, persistent, and reproducible relief following cervical RFN[12]. Seventy-one percent of patients achieved relief following RFN, and in those patients the median duration of relief was more than 400 days. A published audit of the author's own practice demonstrated an 80% success rate in achieving substantial relief of pain, lasting for a mean of 9 months in patients with the established diagnosis of cervical zygapophyseal joint pain[13]. There would, therefore, appear to be generalizability of the procedure beyond the patient's originally considered in the trial of Lord et al. One controlled study of cervical RFN for cervicogenic headache has been reported, with a negative conclusion[14]. However, this study is fatally compromised by patient selection. The participants had explicitly not obtained profound relief from diagnostic blocks, even when all levels from C2 to C6 were targeted, and the diagnosis was based on clinical grounds alone. It is hardly surprising that RFN failed to help.

The trial of Lord et al. has recently been criticized under the aegis of the bone and joint decade review[15]. The criticisms of the study were surprisingly vehement and were not matched by similar critiques of other literature offered in the same chapter, particularly those dealing with surgical techniques for the management of radiculopathies. A comprehensive refutation of these criticisms has been published elsewhere [16]

The desire to perform procedures that are safer and less damaging than the thermal lesions created by conventional radiofrequency denervation has spawned interest in pulsed or cooled RFN. These approaches involve the application of radiofrequency energy in a manner insufficient to cause thermal injury to tissues but which putatively affect nerve transmission. To date, there are no randomized controlled trials of this technique, and the extant reports are limited and constitute a very low level of evidence. As such there is insufficient evidence to establish any efficacy or otherwise of this intervention and is not further considered in this chapter.

In summary, the evidence for cervical medial branch radiofrequency denervation is internally consistent. It has been portrayed as contentious by its detractors, but no evidence of inefficacy has ever been presented.

ANATOMY

Paramount to the safe and efficacious performance of a radiofrequency denervation is understanding the relevant anatomy. The zygapophyseal joints are innervated by the medial branches of the cervical dorsal rami. The dorsal rami of C4 to C8 originate from the spinal nerves after they have exited the intervertebral foramina. They then pass posteriorly over the roots of the transverse processes before coursing around the waste of the articular pillars. They are covered laterally by the tendinous origin of the semispinalis capitis. Branches to the articular zygapophyseal joints arise as they approach the posterior aspect of the pillar. There is a superior and an inferior branch supplying the joint above and joint below, respectively. Each zygapophyseal joint therefore receives half of its nerve supply from above and half from below. At the C3 level, the dorsal ramus has a deep medial branch which passes around the waste of the C3 articular pillar and innervates the C3-4 zygapophyseal joint. The more superficial medial branch is large and is known as the third occipital nerve. It crosses the lateral margin of the C2-3 zygapophyseal joint providing articular branches to that joint. Importantly, the third occipital nerve has reliable sensory innervation to the skin in an area inferolateral to the occipital protuberance approximately 3 × 3 cm in area. Anatomical studies have demonstrated that the C3 medial branch also reliably supplies superficial skin but that there is variable cutaneous innervation from C4 and C5 while the medial

branches of C6 to C8 very rarely furnish any cutaneous sensation.

RADIOFREQUENCY LESION CREATION

The key issue in considering radiofrequency lesion generation is the shape of the lesion created by the electrode. Radiofrequency lesions are created by passing radiofrequency energy through tissue, resulting in rapid movement of charged molecules (principally proteins) leading to friction and heat. If the density of radiofrequency energy and time of application are sufficient, an irreversible heat injury is created—the tissue is effectively denatured. Radiofrequency lesioning requires a circuit to allow the electrical energy to pass through the patient. By restricting the area through which radiofrequency energy passes, through having a small exposed area of conducting material, radiofrequency energy can be focused and directed. This is the principle underlying an electrode with an exposed tip. This is usually around 4 to 6 mm (See picture of electrode [Figure 36.1]). The circuit is completed by a broad grounding plate which disperses the energy over a large area and therefore causes no significant heating.

The lesion created around the electrode tip is a small oblate spheroid. It does not extend beyond the tip of the needle and typically extends beyond the surface of the electrode for no more than 1.5 times the diameter of the electrode used [17]. Therefore, the thermal lesion created by the radiofrequency energy is immediately adjacent to the tip of the needle and has its maximal dimensions away from the needle in the middle of the exposed tip (Figure 36.2). This is counterintuitive to the usual use of needle and injection techniques where the needle is typically placed perpendicular to the target structure such as occurs in medial branch blocks or other nerve blocks. To incorporate an appropriate length of nerve, the electrode needs to be inserted with its tip in contact and parallel with the target nerve. These considerations dictate the approach that must be taken to produce an effective radiofrequency denervation. The needle needs to be inserted parallel to and in contact with the target nerve. In the neck this means the needle is inserted from a slightly caudad angle and needs to make contact with the nerve as it passes around the curved articular pillar. At best a straight needle can be tangential to the curved structure. Therefore, to incorporate a longer length of nerve it is often desirable to have two approaches or to utilize a curved active need tip. In the study of Lord et al., the technique used was a parasagittal insertion so that the electrode was in contact with the most lateral portion of the medial branches as it passed around the articular pillar and then the 30° oblique approach so that the needle was in contact with a more anterior portion of the nerve (Figure 36.3).

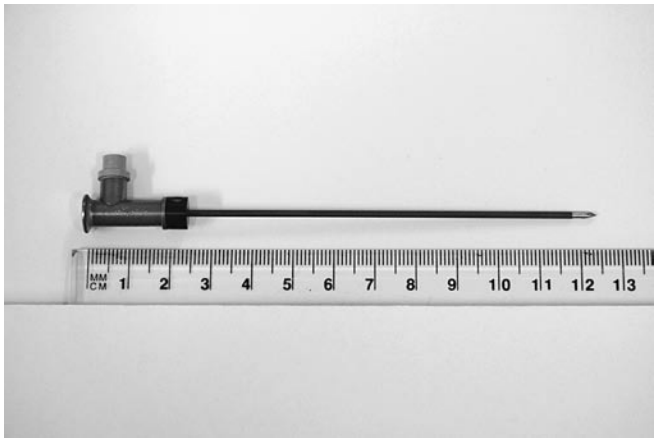

Figure 36.1 A Cosman 10-cm radiofrequency thermistor electrode. Note the insulated shaft and exposed tip.

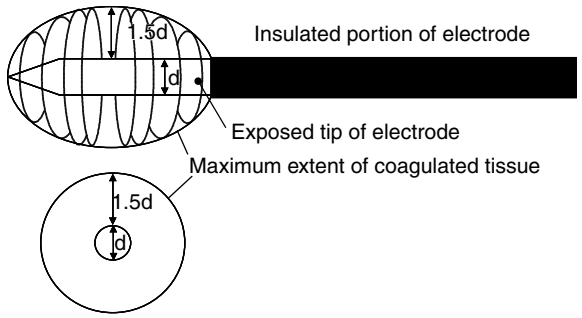

Figure 36.2 The area of heating around a radiofrequency electrode is an oblate spheroid, which extends approximately 1.5 diameters in its maximum dimension away from the surface of the exposed tip.

These approaches can be achieved safely as the key neurovascular structures in the cervical spine are anterior to the anterior border of the articular pillar. Consequently, provided that the electrodes are kept well behind the anterior border of the articular pillar on a lateral view, then they are well away from any of the structures such as the vertebral arteries, cervical nerve roots, or carotid arteries.

TECHNIQUES

The technique described in this section is that utilized in the study of Lord et al. [7].

Equipment

The procedure must be performed under real-time radiologic guidance using a C-arm image intensifier or ceiling mounted image intensifier such as is used in angiographic studies. The orientation of the C-arm needs to be frequently changed during the procedure, which can either be done manually by an assistant or, with a powered device, by the operator via sterile controls of the image intensifier. It is important that the site of lesion creation can be recorded in hard or electronic copy for future reference.

The electrodes used can vary in shape and size. However, they need to both be able to generate heat and as well as monitor the temperature at their tip. A 12-gauge electrode with a 5-mm exposed tip represents a reasonable compromise between the size of the lesion and the trauma

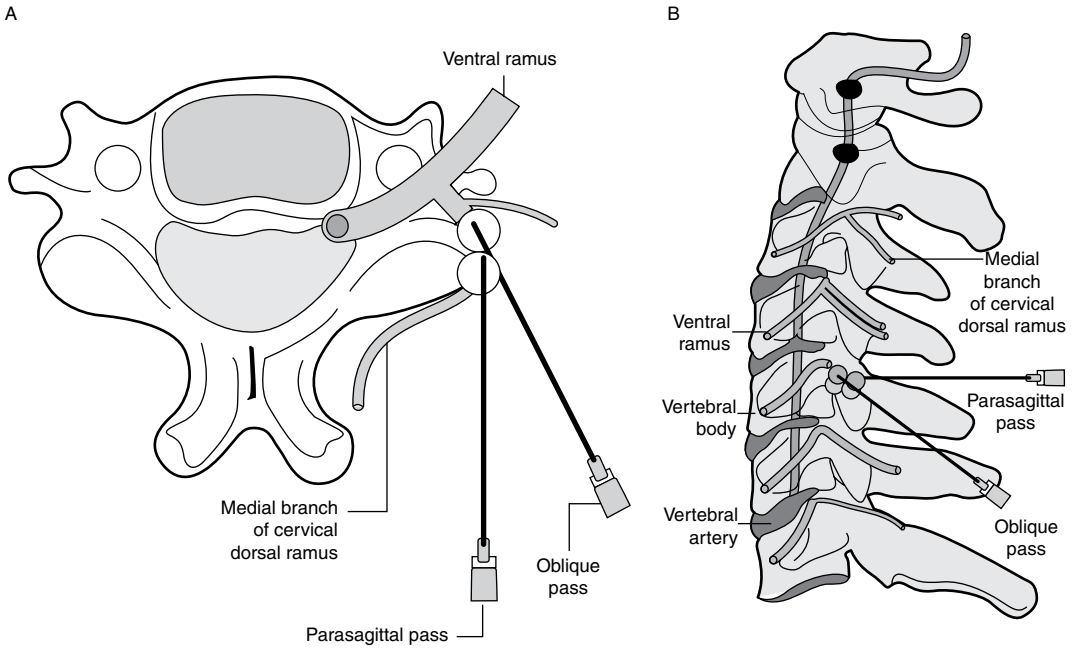

Figure 36.3 Axial **(A)** and lateral **(B)** views of an idealized cervical spine depicting the position of electrodes for 30 degree and parasagittal passes to target a C5 medial branch. From Lord et al [7]. Used with permission.

induced by electrode insertion. A length of 10 cm provides access to the medial branches in most individuals, but longer electrodes may be required for lower cervical joints in larger patients (Figure 36.1).

The radiofrequency generator should allow the temperature of the tip to be monitored, and on contemporary machines this can be controlled thermostatically. The time that the radiofrequency energy is applied can be controlled manually or through a timing circuit (Figure 36.4).

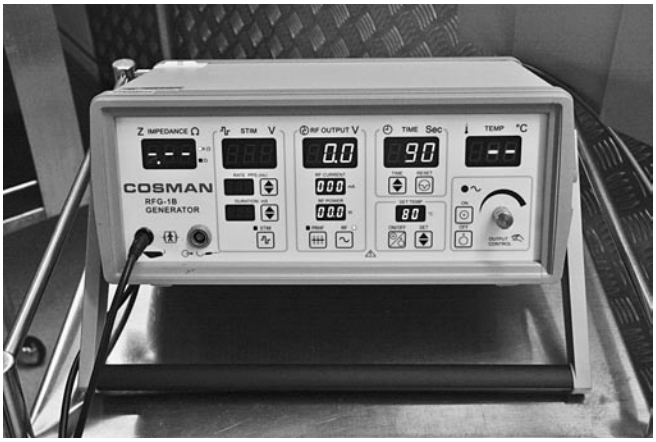

Figure 36.4 Radiofrequency generator.

Patient Preparation

It is vital that patients have a clear understanding of the nature and expectations of the procedure and the potential adverse events. Informed consent should be obtained from all patients before undergoing this procedure. The procedure is typically performed on an outpatient basis. It is the author's experience that premedications are rarely required unless patients are particularly anxious. Alert patients can cooperate fully and report any unusual or unexpected sensations during the procedure. Patient cooperation is vital for issues such as correct positioning, retraction of the shoulders to access lower levels, and to enable them to comply with instructions such as opening their mouth to facilitate visualization of the upper articular pillars on certain views.

The patients are then positioned on their side with the target side uppermost. A grounding plate is placed on the thigh and the target area is prepped with antiseptic solution before draping, leaving the neck and face exposed. Often the use of pillows on the x-ray table to enable the patient to stay comfortable through the reasonably prolonged period of immobility is helpful. In particular, giving them a pillow to hug can help patients keep their arms comfortable and away from the field of view.

Imaging

The procedure is performed under full imaging guidance. Prior to any needles being introduced into the skin, it is vital to obtain a clear, true lateral view of the target articular pillars (Figure 36.5). The C-arm is then rotated so that a 30° oblique approach and parasagittal view of the target area are readily obtained (Figure 36.6, 36.7). Some

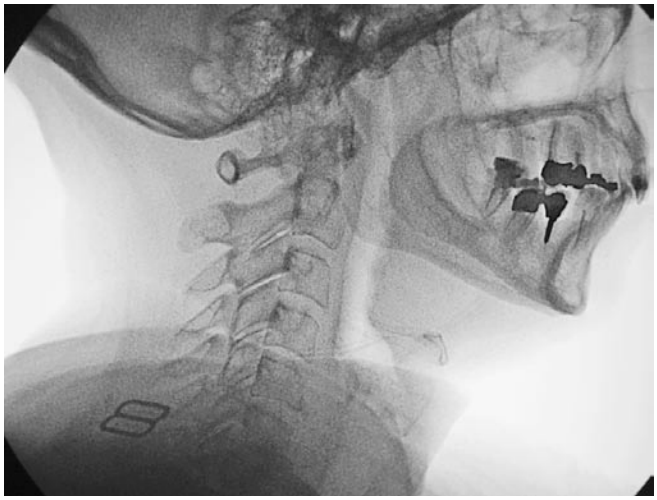

Figure 36.5 A true lateral view of the cervical spine centered on C4. Note that the articular pillars are superimposed and the disc spaces are clear.

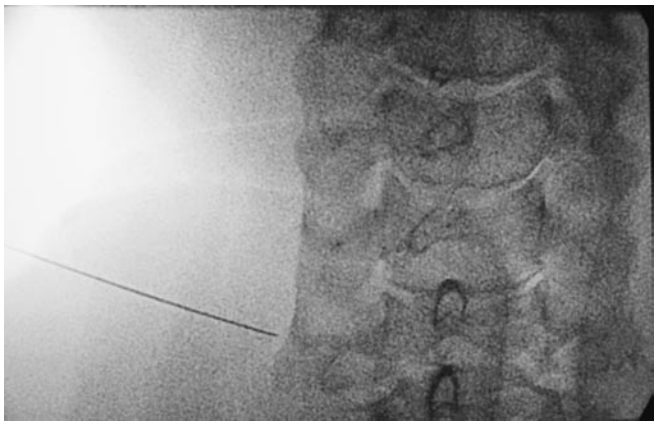

Figure 36.6 Anteroposterior view of the cervical spine demonstrating that the spinous processes are centered and the waists of the articular pillars can be appreciated at the lateral bony margins of the spine.

servo-driven equipment can lock these coordinates into memory so that the correct position can be readily reproduced without the need to repeat screening. Radiation exposure can be minimized in a number of ways. First, relatively low resolution is required for the procedure. No diagnostic information is being obtained from the images. The issue is to be able to clearly delineate the articular pillars on the lateral view and lateral bony margins of the articular pillars on the 30° oblique and parasagittal views. Using the lower resolution image intensification decreases the amount of radiation. Once the target level has been correctly identified, coning in on the area minimizes radiation exposure to surrounding tissues and decreases the total amount of absorbed radiation. Finally, all images should be obtained with the minimum screening time required to generate an appropriate image. In the author's experience, total screening times for two level radiofrequency denervations are in the order of 2 to 4 minutes.

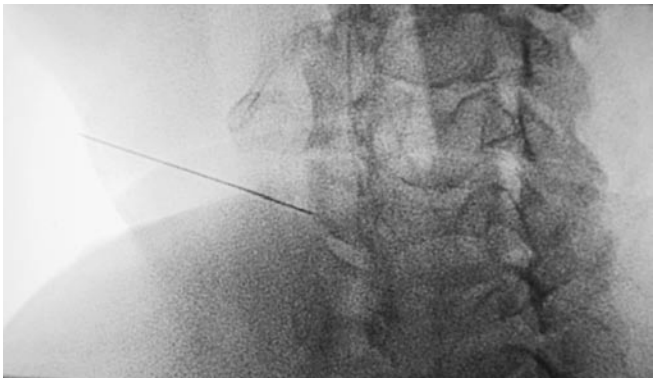

Figure 36.7 A 30° oblique view of the cervical spine. The spinal needle is correctly positioned for a medial branch block at C5. This is used to introduce local anesthetic to render the procedure pain-free, and it provides a target point to guide insertion of the radiofrequency electrode.

Local Anesthesia and Target Identification

The target medial branch is anesthetized through injection of 2 to 3 mL of long-acting anesthetic (bupivacaine 0.5%). This is performed in a manner identical that of a typical medial branch block utilizing 25-gauge, 10-cm spinal needles. On a true lateral view, the needle tip is placed on the skin so as to lie over the centroid of the articular pillar. The needle is then advanced using bevel steering until it contacts the periosteum of the articular pillar. Local anesthetic is then injected slowly. The use of a minimum volume extension tube between the needle and the syringe minimizes inadvertent movement of the needle during injection. On larger articular pillars additional injection points above and below the centroid may be important to ensure that the area is densely anesthetized. During the injection the needle can be withdrawn marginally from the articular pillar so that the tissues more superficial to the articular pillar that may be affected by the radiofrequency lesions are also anesthetized. At the third occipital nerve level, the injection of 1 to 1.5 mL at each of the usual target points for a medial branch block, that is, at the joint line and just above and just below the joint line in the midline of the articular pillars, is appropriate. The needle should then be left in contact with the central point of the target level to facilitate electrode placement.

Once the local anesthetic has had adequate time to have its effect, usually 2 to 4 minutes, then the electrode can be introduced. Through leaving the needle over the target site, even on unconventional views such as a 30° oblique view the correct site for placement of the electrode can be readily appreciated (Figure 36.7). A syringe charged with local anesthetic and a 23-gauge needle is used to anesthetize a tract through which the electrode passes. From a 30° oblique view, the tip of the local

anesthetic needle is inserted at the point overlying the tip of the spinal needle identifying the target. Local anesthetic is then introduced in a bleb into the skin and then along a tract down toward the target point. Once this has had a chance to have its effect a small nick is made in the anesthetized skin by stabbing with a number 11 scalpel to create a small aperture through which the larger electrode can be inserted.

The electrode is then slowly inserted and steered toward the target using frequent brief screening from both the true lateral and oblique views to ensure that the electrode tip does not pass beyond the target point. It is useful to direct the electrode tip slightly medially to the target point defined by the tip of the spinal needle. This means that the electrode contacts the back of the articular pillar and can then be repositioned laterally until it glides past the bone. As soon as there is any sense of the electrode progressing beyond the back of the articular pillar or whether there is any uncertainty as to the position of the electrode, the imaging should be reconfigured to provide a lateral view. The position of the electrode should then be assessed both on lateral and the parasagittal or 30° oblique views. The target electrode positions on a typical articular pillar are overlying the centroid of the pillar (Figure 36.8). The tip of the electrode should be positioned at the junction of the posterior three-fourth and anterior one-fourth of the articular pillar on the true lateral view. Once the lesion has been created, the electrode is then repositioned to a more cephalad (high) position, a further lesion created, and then to a more caudad (low) position for the final lesion to create a matrix of lesions likely to incorporate the course of the medial branch. Leaving a gap of no more than 1 electrode diameter between these lesions minimizes the likelihood of missing the nerve.

The procedure is then repeated using a parasagittal track. This is performed in identical fashion to that of a 30° oblique track with the exception that the tip of the needle should be advanced to a line joining the anterior third and posterior two-third of the articular pillar. This is to create a lesion tangential to the lateral aspect of the articular pillar.

The lesioning parameters used are a temperature of 80°C sustained for 90 seconds. If a patient experiences pain at any point during this procedure, the radiofrequency machine is quickly turned off, the position of the electrode is checked, and if it is in the correct site further local anesthetic is infused using the spinal needle that has remained in situ. Care should be taken to make sure that the electrode does not come into contact with the spinal needle during radiofrequency lesion creation. In this circumstance, the spinal needle conducts heat away from the tip of the electrode resulting in difficulty achieving the target temperature and causing heating of tissue along the course of the spinal needle. The patient will often experience pain during this that is rapidly relieved by slightly withdrawing the spinal needle.

The number of lesions required on a given articular pillar will depend upon the size of the articular pillar and whether the expected course of the nerve has been incorporated in the lesions. Narrower articular pillars may only require two lesions.

C7 Medial Branch Denervation

The C7 medial branch has a different course than the branches between C3 and C6. Specifically, the lateral transverse process is more prominent and the medial branch passes over the root of the transverse process. The superior articular pillar above the transverse process is smaller and a parasagittal approach alone is usually adequate to incorporate this nerve. However, it is important that at least two sites along the root of the transverse process should be targeted so that the variable course of the medial branch over the root of the pillar, which maybe more lateral along the superior border of the transverse process, can be incorporated (Figure 36.9) [18].

Third Occipital Nerve

The third occipital nerve passes over the midpoint of the C2-3 zygapophyseal joint seen on a lateral projection. The target points recreating lesions are therefore over the joint itself and above and below as for other levels. The same depths of needle insertion described earlier are appropriate for the third occipital nerve.

Imaging Pitfalls

It is vital that images be obtained and recorded immediately before any lesion is created so that the position of the electrode can be known. Sometimes connecting the electrode or unrecognized patient movement can move the electrode before the radiofrequency energy is applied.

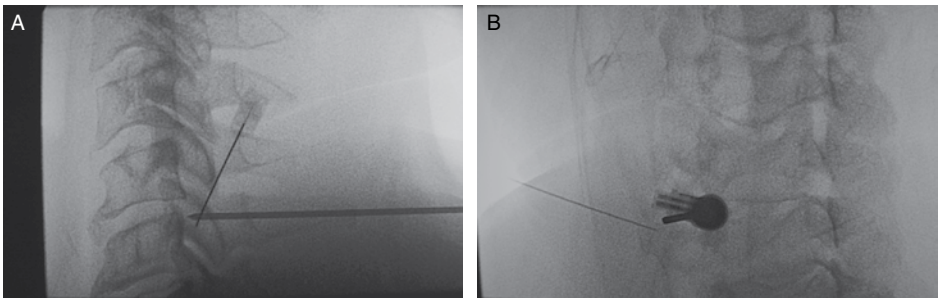

Figure 36.8 Lateral (**A**) and 30° oblique (**B**) views of an electrode correctly placed for a midposition lesion of the C6 medial branch.

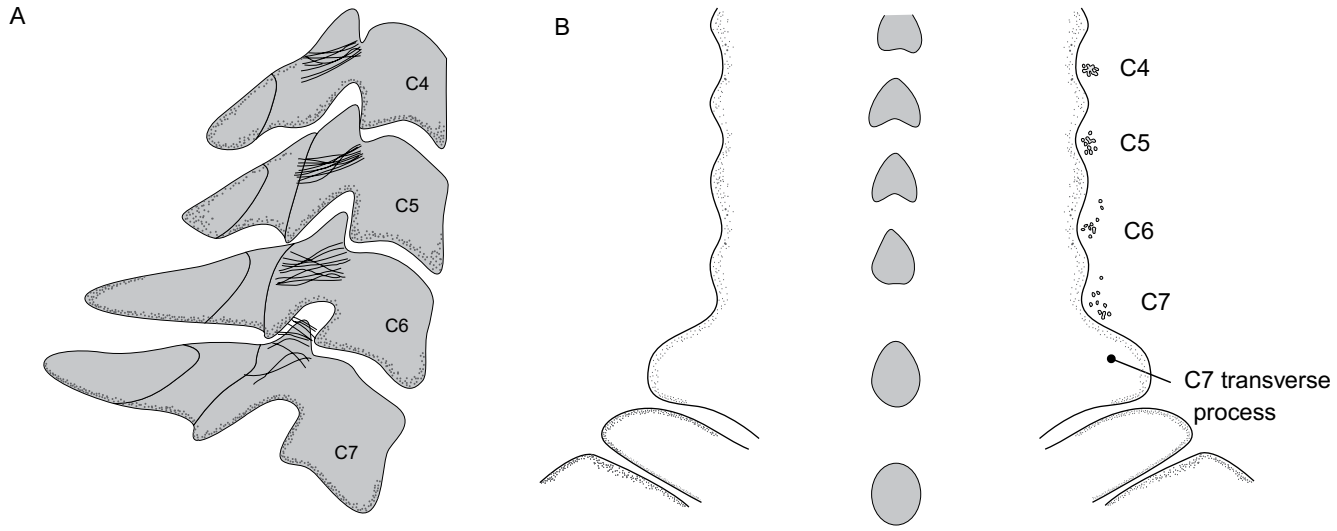

Figure 36.9 Line drawings of the course of the lower cervical medial branches derived from a series of cadaveric dissections. Each nerve is represented by one line or dot. The nerves are drawn in relationship to an idealized cervical spine relative to radiographic, bony landmarks. On the lateral view (**A**) the C7 medial branch can be seen to course over the top of the transverse process. Variations to C7 include a more lateral position along the root of the transverse process as seen on the anteroposterior view (**B**). Used with permission.

It is possible to be misled by the incorrect interpretation of imaging obtained during this procedure. In particular, it is possible to produce images on the screen that may appear to be lateral images but are in fact not true laterals. These can arise where undue cephalad or caudad orientation of the imaging device causes the articular pillar of one level to be overlaid on another. This will result in loss or blurring of the intervertebral disc spaces (Figure 36.10).

Similar problems can emerge where undue axial rotation has occurred that projects the articular pillar of one level over the vertebral body itself. It may appear that there is a true lateral image with all of the articular pillars perfectly superimposed when in fact the articular pillars of only one side are being seen (Figure 36.11).

These problems can be avoided by checking the orientation of the x-ray beam relative to the patient's neck. It should be more or less perpendicular to the neck when the patient is lying on their side. It is also possible, when coning down, to miscount the veritable bodies and potentially create lesions at the wrong level. This should always be checked before any lesions are created.

CARE FOLLOWING THE PROCEDURE

The patients should be warned that because of the anesthetic injection they may feel unsteady on sitting up. This relates to local anesthetic effect on cervical musculature and medial branches which, particularly at the high levels in the cervical spine, have an important contribution to maintaining balance.

The wounds should be cleaned and dressed. The patient should be advised to keep them clean and dry for 48 hours until they fully seal. Appropriate analgesia should be prescribed with the expectation that the local anesthetic will wear off several hours after the procedure and it is best to preemptively treat with oral analgesia. Patients in open series have typically experienced neck pain related to the procedure for 1 to 2 weeks. They should be encouraged to report any discharge, temperature, or worsening pain as these may indicate infection.

ADVERSE EFFECTS

There has been no comprehensive evaluation of the adverse effects related to radiofrequency denervation. In the available studies, adverse effects were relatively minor. Local bleeding particularly in patients on antiplatelet agents such as aspirin may be a problem but can typically be managed by local pressure. Full anticoagulation would be a contraindication to the procedure. Provided appropriate measures are taken, the radiation exposures during these procedures are minimal. The use of older imaging equipment, computed tomography guidance, or prolonged exposure times will significantly increase the amount of radiation exposure. It is the author's practice to include mention of radiation exposure and a possible increase in future cancer risk to all patients who are undergoing radiofrequency denervation. It should be emphasized however that there are no extant reports of radiation exposure related to this type of procedure leading to any specific malignancy such as thyroid cancer. Using the parameters noted earlier, x-ray exposure during a procedure is less than that experienced during a chest x-ray and considerably less than would occur from a computed tomography scan of the cervical spine.

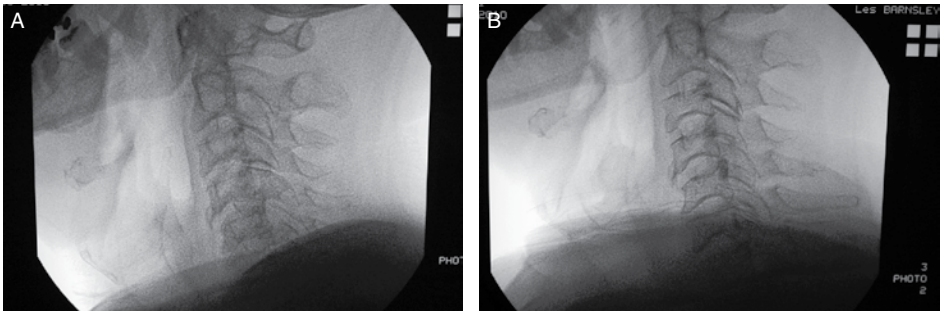

Figure 36.10 Radiograph obtained during screening for a medial branch block. The articular pillars appear superimposed, but in reality there is excessive caudal tilt, so that the apparent superimposition is between articular pillars at different levels (**A**). Note that the disc spaces are not seen, and the anatomy at the upper levels looks abnormal. Contrast this with a true lateral obtained by the same procedure (**B**).

The main adverse effect related to the procedure is really a lack of efficacy. Failure to correctly incorporate the nerves or the procedure being directed at nerves that are not the conduit for the patient's pain will result in failure of the procedure to achieve pain relief. In an open series of consecutive patients, approximately 20% of patients failed to experience significant pain relief following a first procedure.

Cutaneous numbness is an unavoidable effect of radiofrequency lesions of the upper cervical medial branches, particularly C3, C4, and the third occipital nerve. The area of the altered sensation is located over the posterior aspect of the neck and rarely presents a problem for patients. Occasionally, patients report an unpleasant dysesthetic sensation in association with the numbness. The mechanism for this is unknown, but it may relate to partial coagulation of the nerve or wind-up of nerves supplying adjacent areas of skin. The literature descriptions concerning this are sparse, but in the author's experience these symptoms typically settle over weeks to months following the procedure. If intrusive, symptoms can often be controlled with antineuropathic pain medications such as gabapentin or pregabalin.

Infection is an important potential risk following this procedure. Its frequency would appear to be low. In the author's experience, the infection rate is less than 0.5%. Detecting deep-seated infection can be difficult. However, the presence of temperature, any wound discharge, increased pain, neck stiffness, and elevated inflammatory markers would all be important clues to the presence of underlying infection. Imaging studies can be used to delineate infection, but studies of magnetic resonance imaging scanning following radiofrequency denervation have revealed that the procedure itself causes signal changes in the neck that can mimic those of infection [19]. Other studies such as labeled white cell studies or other nuclear medicine techniques may be appropriate.

Some patients experience significant unsteadiness following radiofrequency denervation. They do not report true vertigo but rather a feeling of being unsteady on their feet akin to getting off a moving boat. Typically, patients adapt to this over weeks but the problem is more marked when upper cervical medial branches are being targeted. Patients most often complain of difficulties in situations where there is a mismatch between visual cues and their body position such as can occur on spiral staircases. They should be warned to avoid climbing ladders or to place themselves in situation where balance is important to prevent serious injury. Patients who will experience marked unsteadiness following radiofrequency denervation will often have had similar symptoms develop after their medial branch blocks during their diagnostic work-up and this should be openly discussed prior to the procedure.

Vasculature and Central Nervous System Injury

When performed properly, with a thorough understanding of the radiologic anatomy, the likelihood of cervical RFN causing injury to vital structures such as the spinal cord or vertebral arteries is extremely small and is currently only a theoretical possibility. The author has been

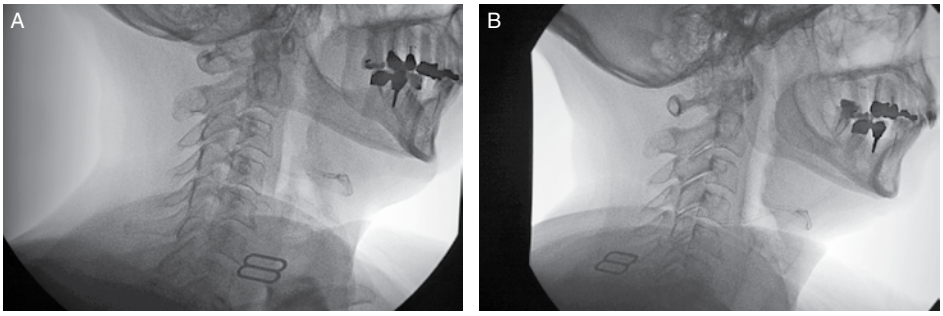

Figure 36.11 Radiograph obtained during screening for a medial branch block. The articular pillars appear superimposed, but there is excessive axial rotation and one of the articular pillars is projected over the vertebral body (**A**). Contrast this with a true lateral obtained during the same procedure (**B**).

unable to find any case reports of properly performed cervical RFN causing vascular injury or cerebrovascular accident. Keeping the tips of any needles posterior to the anterior border of the articular pillars and always lateral to the lateral border of the articular pillar will prevent any of the anterior vascular structures from being affected.

Long-term Effects of Denervation

The long-term effects of cervical RFN of the medial branches of the cervical dorsal rami have not been studied. A concern might be that a joint could become neuropathic, effectively a Charcot's joint. Typically, this occurs in peripheral joints, mainly in the lower limbs where all proprioceptive information to the segment is lost. That is not the case following radiofrequency denervation as the innervation to the contralateral joint, the intervertebral joints, and the segmental ligaments would remain intact. There is therefore little reason to believe there is a real or important risk of Charcot's joints developing following radiofrequency denervation.

REPEAT PROCEDURES

If a patient experiences profound and satisfying relief of pain that then recurs, it would appear quite reasonable to perform repeat cervical RFN. There have been no randomized trials of repeated RFN as a long-term treatment but rather extrapolation from a single procedure and uncontrolled case series. These would indicate that patients often achieve useful and reproducible relief of pain with multiple radiofrequency procedures.

CONCLUSION

Cervical RFN offers a useful pain relieving modality for patients with chronic cervical zygapophyseal joint pain. It is a technically demanding procedure that requires a precise knowledge of the radiologic anatomy to be performed safely but at the same time offers a high probability of achieving important and clinically useful pain relief in appropriately selected patients. Its adverse effect profile is favorable but the long-term effects are not known. Although it is axiomatic that further studies would be useful in better delineating the effectiveness and efficiency of the technique, the current state of knowledge is that it has proven efficacy in carefully selected individuals. The procedure itself can be time consuming, but its potential for helping patients would appear to justify this investment.

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37 Chronic Whiplash and Whiplash Associated Disorders

Jerome Schofferman

Nothing either good or bad,
but thinking makes it so.

William Shakespeare, Hamlet Act II, Scene 2

INTRODUCTION

The term *whiplash* is used to describe both a mechanism of injury and the neck pain caused by that injury. For some, the mere mention of whiplash brings to mind a patient wearing a foam cervical collar who is complaining of neck pain and a host of other symptoms while waiting for a cash settlement after a motor vehicle collision (MVC). On the other hand, for many medical professionals, whiplash implies a medical condition that has the potential to cause significant pain and impairment, often has a readily identifiable structural cause for the pain, and is often responsive to treatment.

In light of current knowledge, the term whiplash can be viewed as a generic term that implies an acceleration and/or deceleration injury to the neck caused by a MVC. It is well established that the term whiplash does not accurately reflect occupant biomechanics during a MVC, but the word is firmly and probably irrevocably rooted in medical, legal, and lay vocabularies. Whiplash injury describes the neck pain sustained as a result of whiplash. *Whiplash-associated disorders* (WADs) is the term used to describe other symptoms that may be seen in whiplash injury patients.

Most persons who are in a MVC are not injured. Of those patients who have neck pain soon after a MVC, one-third can be left with some degree of chronic pain, and 2% to 7% become partially or total disabled [1–4]. Whiplash injury remains controversial and sometimes misunderstood despite a plethora of peer-reviewed literature that has provided better understanding of its biomechanical and medical components.

This chapter provides an overview of chronic whiplash injury and WAD—the problems that bring patients to spine specialists. There have been several discussions of whiplash injury published recently from which I have drawn some of my narrative [5–7]. This chapter does not discuss the evaluation and emergency management of the trauma patient.

MECHANISM OF WHIPLASH INJURY

Historically but incorrectly, the biomechanics of whiplash of injury was thought to be an overall hyperextension of the entire neck with subsequent recoil—the "whip." However, we now know that this is not the case. In fact, the overall range of motion of the neck during whiplash injury remains within the physiologic range [8–12]. There is, however, abnormal motion of one or more spine segments, and it is this abnormal segmental motion that causes the injury [8,11].

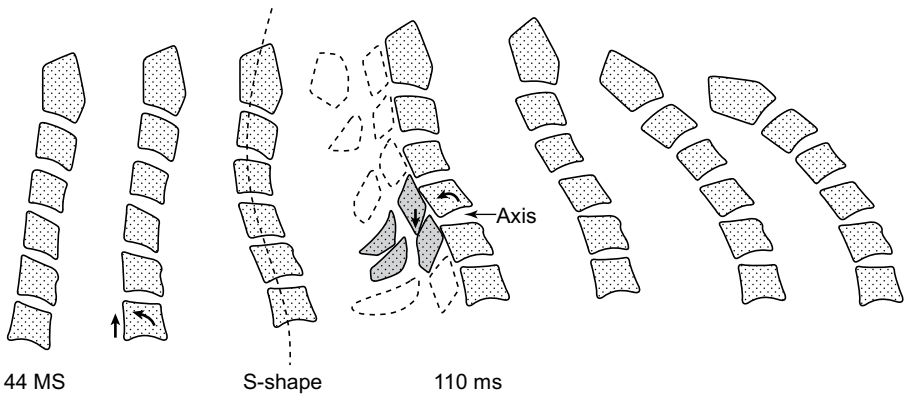

Figure 37.1 Changes in vertebral alignment during rear-end impact.

The biomechanics have been well studied and described [6–13]. As a result of a collision, energy is transferred from the striking vehicle (bullet vehicle) to the body and frame of the struck vehicle. The energy then continues to the seat and then to the torso. As a result, the torso is thrust upward and forward, losing its kyphosis, while the head and neck remain still. This sudden forceful upward and forward motion of the trunk compresses and deforms the cervical spine from below. The lower cervical segments extend and the upper segments flex, forming the so-called S-shaped curvature. As the trunk continues forward and upward, the head drops and the upper segments extend. At this point, the entire cervical spine is extended but still remains within its physiologic limits. The spine then returns to its starting position (Figure 37.1).

Although the overall range of motion of the spine during impact is within the physiologic range, there is abnormal motion of individual spinal segments, which is the cause of injury [8,9,12]. As a result of this pathological motion, the anterior part of the motion segment can separate abnormally while the posterior portion can be compressed (Figure 37.1) and subject to abnormal sheer forces. The facet joint can undergo pinching of the synovial fold and capsular strain [8,10] in addition to the possibility of impact injury to the joint with the possibility of intra-articular contusions and hemorrhage [6]. The abnormal anterior separation can result in strain or avulsion of the anterior annulus [6,13].

NATURAL HISTORY

The natural history and prognosis of whiplash injury have been reviewed many times [1,2,4,14,15]. The percentage of persons involved in a MVC who suffer any neck pain is not known definitively, but has been estimated at about 15% [16]. However, in a small prospective study of 57 patients, Elbel et al. found that 25 (44%) had acute neck pain [17]. Neck pain begins in the first 24 hours in 80% to 90% of patients, whereas arm pain and weakness begin more than a week after the accident in 50% [5,18]. Multiple longitudinal studies have shown that about one-half to two-thirds of patients who develop neck pain after a MVC recover completely [1,2,4,14,15]. Most of the patients who do not recover fully have chronic mild to moderate pain, but about 2% to 4% become severely disabled [2,3]. Radanov prospectively studied 117 patients after whiplash injury [2]. At 3 months, there was full recovery in 56% of patients, at 6 months there was full recovery in 70%, and at 15 months 76% had recovered fully. At 24 months, 18% remained symptomatic including 4% with severe symptoms and five patients who were partially or totally disabled.

Berglund et al. looked at a very large insurance company database to ascertain the long-term sequelae of whiplash injury [19]. They looked at the prevalence of neck pain in four independent groups of patients, all of whom were insured by the same company. In two control groups composed of persons who were not in a MVC, the prevalence of neck pain was 12% and 14%, respectively. In those who had been in a MVC but did not develop neck pain as a result, the prevalence of neck pain 7 years after their MCV was 14%. However, in those who had been in a MVC and had acute neck pain as a result, the prevalence of chronic neck pain was almost 40%, nearly three times greater than the normal population. This is convincing evidence that those who develop neck pain acutely after a MVC are at almost threefold risk of developing chronic neck pain.

Prognostic Factors and Risk Factors

Many studies have looked at whiplash injuries to try to identify prognostic factors for adverse outcome [14,15,20,21]. It is useful to consider crash characteristics and human factors when examining prognosis.

Crash factors include the relative sizes (mass) and speeds of the bullet and struck vehicles (often expressed as change in velocity [ΔV]), damage to the vehicles, position of the head restraint, use or type of seat belt, and direction of the collision. A recent review summarized this evidence and concluded that "collision-related factors are not prognostic of recovery . . ." in WAD [14]. ΔV is a way of describing the amount of force transmitted from the bullet vehicle to the struck vehicle. One might assume that the greater the ΔV, the greater the potential for injury to the occupants, but this relationship is far too variable to be useful clinically or for forensic purposes [5,17].

Bannister et al. stated that a ΔV of 2.5 mph was enough to result in symptoms whereas a ΔV of 8.7 mph was necessary to cause vehicle damage [5]. Elbel et al. assessed the predictive value of ΔV with respect to cervical spine injuries in 57 patients involved in MVCs [17]. They found only a "slight" correlation ($r = 0.55$) between ΔV and neck pain and no correlation between ΔV and severity of injury as graded by the Quebec Task Force criteria. They concluded there was no threshold for ΔV that could be used to determine prognosis. In a study of 105 patients evaluated after MVCs that had a mean ΔV of 6.3 km/h (2.9 mph), 41% of persons had neck pain, 23% had low back pain (LBP), and 14% had thoracic pain at 5 weeks [22]. Relative speeds are not predictive of neck injury [14]. Bannister et al. stated that most MVCs occur at "speeds of less than 14 mph and it is in these that whiplash injuries occur" [5]. The severity of property damage to either vehicle is not a reliable predictor of injury or outcome in low-speed collisions [23,24]. Croft and Freeman, in their literature review, found that a substantial number of injuries occur in crashes of little or no vehicle damage [24]. They also found only a limited correlation between crash severity and injury claims. Finally, it is probable, although not proven, that neck restraints that are positioned too low may increase the risk of whiplash injury [5].

Human factors include the size and weight of the occupants, awareness of the impending collision, direction in which they are facing at impact, gender, and individual tissue tolerances. However, the data for each of these factors are quite contradictory.

The strongest predictor of poor outcome in virtually every study is high initial pain intensity when patients are first evaluated [4,14,15,21]. In one thorough systematic review, it was noted that older age, gender, high initial psychological response, and compensation litigation were not associated with an adverse prognosis [15]. There

was "limited" prognostic value for patients with pain in multiple areas or those with prior psychosocial problems. There was inconclusive evidence regarding the prognostic value of head position at impact, radicular symptoms, cognitive impairments, poor concentration, prior headache, being unprepared for collision, and $\Delta V > 10$ km/h [15]. Most recently, Carroll et al. agreed that high pain intensity is the strongest predictor of poor outcome and reported that the evidence is conflicting regarding the prognosis according to gender or age [14]. They found no evidence regarding the role of preexisting disc degeneration. In a third review, again the strongest predictors of poor outcome were high initial pain intensity and work disability. Female gender, lower level of education, high somatic response, and poor sleep were somewhat predictive [21]. In their review, Bannister et al. found a worse outcome to be associated with rapid onset of pain, severity of neck pain, acute hospital admission, pain radiating to the arms, and headache [5]. They also noted a worse prognosis if there was preinjury significant psychiatric disease, preexisting neck pain or prior whiplash injury, or LBP.

Psychological Predictors of Outcome

A recent systematic review concluded, "We found no scientifically admissible studies examining the effect of psychological or social factors in the onset of WAD. . ." [25]. There is no preinjury personality that renders individuals more likely to develop chronic pain after whiplash injury, nor are there psychosocial factors or personality traits that are significant predictors of chronicity [25]. In their recent systematic review, Williamson et al. found no associations between the development of chronic pain after whiplash injury and personality traits, general psychological distress, well-being, and social support, life control, and psychosocial work factors [26]. They also state the literature was inconclusive.

Radanov et al. recorded symptoms and psychological test scores in 50 patients with acute whiplash and then again at 3 months and 2 years [27]. Psychological testing was normal initially in 82%. At 3 months testing was abnormal in 81% and remained abnormal at 2 years in 69%. There was no correlation between the chronicity of the symptoms and the initial psychological testing. They concluded that psychological problems were a consequence rather than a cause of pain.

Gozzard et al. examined factors that might affect employment and disability after whiplash injury [3]. Forty (7%) of 586 patients had not returned to work. The strongest predictor of prolonged disability was intensity of symptoms. There was no difference in the prevalence of preinjury psychological illness in those who did or did not return to work. Hendriks et al. prospectively followed 141 patients with acute whiplash [21]. At 1 year, 12% were partially or totally impaired or disabled. There was no predictive value for initial psychometric testing, the presence of litigation, age, gender, differences in speeds between vehicles, or type of early treatment.

Recently, the role of coping styles has been examined. Patients with better coping abilities had more favorable outcomes than those who cope poorly [28,29]. The Multidimensional Pain Inventory classifies patients by coping styles and was used to classify 123 early whiplash patients who were then followed for 12 months [28]. In the 48 adaptive copers, one-third were pain-free, one-third had infrequent pain, and one-third had frequent pain. In both the dysfunctional and interpersonally distressed groups, 93% had residual pain and none were pain-free.

Effect of Litigation on Outcome

The effect of litigation on outcome is one of the most contentious aspects of whiplash injury. Although it may be counter-intuitive, in virtually every patient-based longitudinal study and systematic review, there has been no good evidence that personal injury litigation adversely affects outcome [2,5,14,15,18,30,31]. In fact, Bannister et al. go so far as to write, "Medical experts are remunerated for opining that patients with symptoms after three months are likely to recover within two years when this lacks any base in scientific evidence" [5].

The idea that personal injury litigants do poorly is largely based on anecdotes, inherent bias, poor research, and lack of familiarity with the current literature [32–34]. In their narrative review, Bannister et al. remarked the view that symptoms and disability will improve after litigation is settled ". . . is unsupported by the literature" [5]. Bogduk reached the same conclusion [6].

The outcomes of whiplash injury patients currently in litigation have been compared with those who had completed litigation [31]. Active litigants reported more pain than those who had completed litigation," but there were no differences in function or employment status. Patients with more pain and more objective findings were more likely to file claims. There was no evidence for improvement after litigation was settled. There were 39% who improved, 55% who did not change, and 5% who got worse after settlement. Sapir compared the results of radiofrequency neurotomy (RFN) for cervical facet joint pain in litigants and nonlitigants [30]. There were no differences in outcomes between the groups.

In the often cited but certainly not carefully read, Gotten study, only 100 of 219 patients seen many years earlier were interviewed [32]. The investigators found that 88% of patients had "improved" to some degree and attributed improvement to settlement of litigation. The authors did not explain the 12% who had not improved at all nor the 34% with residual chronic neck pain despite settlement.

Persistent neck pain and other WAD symptoms in litigant whiplash patients have been attributed to "accident neurosis." It has been implied that patients are "cured by verdict" [33–35]. The studies cited have very serious methodological flaws and their conclusions are not justified [35].

Several reports have shown a correlation between retaining an attorney and longer time to claim closure or worse outcome [4,36,37]. Consulting a lawyer was associated with less chance of early claim settlement, longer treatment, and slightly worse function, but not with improvement in pain or return to work at 12 months [5]. There was no predictive value to cost of vehicle repairs or the degree of damage to the vehicle, but once again,

severe pain at the initial evaluation was associated with worse outcome. In a no-fault no tort system, there was a shorter time to case closure, although the same authors state ". . . this determination of recovery did not necessarily reflect complete resolution of symptoms," and in fact many patients had to reopen their claims at a later date [14,35]. Most of these data come from insurance databases, not prospective longitudinal studies of patients in MVC. Therefore, no conclusions can be drawn about the effect of treatment.

Cultural factors have been examined with respect to outcome [38]. Some authors suggest that chronic whiplash occurs only in countries that have a "whiplash culture," and that chronic pain after whiplash injury rarely occurs outside the medical-legal context [38]. These authors suggest that in cultures where there is no expectation of financial remuneration for pain, suffering, and lost wages, there is no increase in the prevalence of chronic neck pain after a MVC. Therefore, whiplash injury must be a psychosocioeconomic illness. Haneline examined these arguments. He implied the evidence for such a phenomenon was weak at best, and pointed out that most of the studies were either anecdotal or methodologically flawed. Bannister et al. did not find the evidence for a cultural cause of prolonged pain after whiplash to be convincing [5]. After their literature review, Holm et al. "found no scientifically admissible studies examining the effect of cultural influence on the onset of WAD . . ." [25]. Siegmund et al. quite logically point out that there must be an organic basis for at least some chronic whiplash injury patients because new anti-whiplash car seats reduce the whiplash injury rates by half [8].

CLINICAL SYMPTOMS

Acute Versus Chronic Whiplash Pain

Chronic pain is best defined as pain that persists beyond the expected resolution of the structural injury rather than by an arbitrary duration. After whiplash injury, most patients are better by 3 and certainly 6 months, and therefore it would be appropriate to define chronic whiplash as neck pain that persists beyond 6 months.

Neck Pain

Neck pain is the predominant symptom of whiplash. It can be in the midline or on one or both sides of the midline. Pain is commonly referred to the region of the trapezius, shoulders, interscapular region, arms, and posterior occipital region (often expressed as headache). Occasionally, there may be pain in the face. If there is a lateral disc herniation, there may be radicular symptoms and/or signs.

Headache

Headache is the second most common symptom after whiplash injury. So-called cervicogenic headache (CHA) varies in severity and frequency. It may be confused or coexist with migraine or tension-type headache. CHA can be considered the most cephalad presentation of axial neck pain and as such can arise from an upper cervical facet joint, intervertebral disc, as well as the C1-2 joint. CHA virtually always involves the base of the skull and frequently refers to the crown of the head and frontal regions. It is often unilateral, but the side can vary even in the same patient. It is often precipitated by prolonged static neck position or repeated end-range flexion, extension, or axial rotation. Diagnostic evaluation has been well described [39] (see Chapters 29 and 34).

Whiplash-Associated Disorders

The other symptoms associated with whiplash are often referred to as WAD (Table 37.1). Symptoms include LBP, shoulder pain, visual disturbances, dizziness, ringing in the ears, weakness, generalized fatigue, poor concentration, difficulty with memory, difficulty sleeping, and secondary psychological changes such as depression [6,8,35].

Low Back Pain

Cassidy et al. stated: "Low back pain is a common traffic injury with a prolonged recovery" [40]. In 8124 whiplash claimants, 4473 initially had LBP, which was still present at 6 months in 30% to 42%. Berglund noted a 20% prevalence of chronic LBP 7 years after MVC [19]. In a narrative review, there was a prevalence of 39% to 42% acute LBP and 39% to 42% chronic LBP [35]. The structural causes of LBP after MVC have not been studied specifically, but do not appear different from the usual causes of chronic LBP seen in adults who were not involved in MVCs. Lumbar disc pain, facet joint pain, and sacroiliac joint pain are seen most commonly.

Shoulder and Arm Pain

Pain in the shoulder area can be referred from cervical facets or discs and can also be due to a primary shoulder problem [41–44]. In a retrospective review of 34 patients with chronic pain at the superomedial aspect of the scapula, Gorski et al. found that 24 had been in a prior MVC [41]. All had restriction of cervical range of motion, positive impingement sign, relief of pain after subacromial

Table 37.1 The Incidence of Various Symptoms in Addition to Neck Pain in Patients with WAD

Symptom	Proportion of Patients with Chronic Symptom (%)
Headache	20–88
Shoulder pain	37–80
Arm paresthesia	15–68
Weakness	68
Dizziness	12–68
Visual complaints	2–42
Tinnitus	4–30
Cognitive impairment	26–71
Low back pain	39–42

Modified from Ref. [6]

injection with local anesthetic and corticosteroid, and abnormal shoulder radiographs. Abbassian and Giddins describe subacromial impingement after whiplash injury [42]. Chauhan et al. described a 22% prevalence of shoulder problems in 524 chronic whiplash patients, usually impingement syndrome [43]. Carpal tunnel syndrome has been reported as a cause of shoulder and arm pain after whiplash [44]. It has been postulated that the injury was due to blunt trauma to the median nerve from the steering wheel or dashboard rather than overuse-related carpal tunnel syndrome.

Psychological Disorders

Peebles et al. used the SCL-90-R to compare chronic whiplash patients with patients with other chronic musculoskeletal pain patients and found no differences with respect to the prevalence or types of psychological problems [45]. There was no characteristic chronic whiplash psychological profile. Mayou followed a group of MVC patients for 1 year to evaluate early and late psychological consequences [46]. Initially, almost 20% suffered acute stress syndrome. At 1 year, 5% met criteria for posttraumatic stress disorder, 18% had travel anxiety, and 12% had a mood disorder. Carroll et al. examined the time course of depression after whiplash injury in more than 5000 whiplash injury patients followed for a year by telephone interview [47]. In those with no psychological mental health problems before injury, 42% developed depressive symptoms at 6 weeks, and another 18% developed depression later. Depression was recurrent or persistent in 38%.

If psychological disorders are usually secondary to pain and impairment rather than being causative, they might be expected to improve if pain is effectively treated. In an evaluation of RFN to treat 17 patients with both cervical facet joint pain and psychological abnormalities, all patients who improved after RFN showed improvement in abnormal psychological tests scores [48]. In all but one of those who did not improve after RFN, the psychological abnormalities did not change.

STRUCTURAL ETIOLOGY OF CHRONIC NECK PAIN DUE TO WHIPLASH

There is robust evidence that the most common structural source of chronic neck pain after whiplash injury is one or more facet joints [6,8,49,50]. Less common is disc injury. Some patients may have both [49–52].

In acute whiplash, there can be damage to muscles and/or ligaments, and this acute myofascial damage can be painful [8]. However, as in soft tissue injuries elsewhere in the body, these injuries are expected to heal in 6 to 8 weeks. When pain persists, it is most likely due to other injuries.

To obtain the structural origin of chronic axial neck pain, the history and physical examination are of only limited value. Imaging studies may offer clues but are not specific. Diagnosis often depends on specific diagnostic injections, particularly testing of the facet joints and discs (see Chapters 30 and 34).

Facet Joints

There is compelling biomechanical, autopsy, and clinical research evidence that cervical facet joints can be damaged in a low-speed MVC and become a source of chronic neck pain [8,49,50]. Based on clinical experience, lumbar and thoracic facet joints can be damaged as well, but there are no robust data that confirm this. Pain could be traced to a cervical facet joint in 49% to 54% of whiplash patients with only neck pain and 60% of those with both neck pain and headache [49–52]. In patients with CHA after whiplash, the prevalence of C2-3 facet joint pain was 53% [53]. No structure other than the cervical facet joint has been so well studied and linked so securely to neck pain after MVC.

There are no specific findings for facet joint pain on history, physical examination, or radiological studies. Facet joint pain can only be diagnosed by anesthetizing the putative painful joint using medial branch blocks (MBBs) [49,50]. If pain is significantly relieved each time the joint is anesthetized using two double-blind controlled MBBs, it can be concluded that the joint(s) is the source of the symptoms. A single block may be less definitive because of a respectable false-positive rate from a single block.

Disc Pain

The data implicating the disc as a source of chronic neck pain after whiplash are not as robust as they are for facet joints. Nevertheless, there is anatomical, biomechanical, and autopsy studies that show discs are innervated and can be injured during whiplash [8,9,54]. In addition, uncontrolled clinical studies are consistent with observations in normal volunteers that cervical discs can be a potential source of pain [55]. In addition, anterior cervical discectomy and fusion (ACDF) can relieve pain in about 70% of patients, many but not all of whom had whiplash as the inciting trauma [56,57].

Discography has been used to try to identify painful cervical discs. However, discography is difficult to interpret and there is a high risk of both false positives and negatives. As a result, provocative disc injections are being used less often. Confounding the difficulties is evidence that cervical disc injection can precipitate pain from a facet joint and some patients may have both problems [39]. To maximize the chances of a true positive provocative disc injection, it is necessary to have a negative MBB at the index level. In addition only one or two discs should be painful and injections of adjacent discs should be painless (see Chapter 30).

Myofascial Pain, Sprain/Strain, or Other Soft Tissue Injury

Soft tissue injury means something that is not bone has been injured. It is a nonspecific term that has little clinical value and is best avoided. There is no good scientific basis to attribute chronic neck pain to "chronic strain or sprain" although these terms are used frequently, especially in the medical-legal context. Furthermore, there are no adequate studies that demonstrate soft tissues alone can be a primary cause of moderate to severe chronic neck pain. That

said, muscles may become painful when compensating for a deeper structural abnormality or poor posture and thereby contribute to the overall pain.

TREATMENT: ACUTE NECK PAIN

After serious injury is ruled out, initial treatment should consist of explanation, education, and reassurance that the outcome is usually favorable. In the absence of motor deficits or suspicion of fracture, imaging studies are rarely necessary in this acute phase. There is good evidence that physician reassurance and sense of caring improve outcome [6]. Physicians should explain what is wrong, recommend that patients remain active despite pain, and describe the generally favorable natural history. Patients who are advised to act as usual and maintain normal activity and function tend to do better than those who are advised to rest, take time off of work, and given a cervical orthosis [58]. There are many informational booklets and books available [59]. Exercises should be prescribed and perhaps demonstrated, not just suggested.

In a prospective randomized study, Kongsted et al. compared the outcomes after treating acute whiplash injury patient treated with cervical orthosis, active mobilization, or a recommendation to "act as usual" [60]. At 1 year, there were no significant differences observed except for a higher rate of disability in the cervical orthosis group. At 1 year overall, 48% of the patients reported considerable neck pain, 53% had some degree of disability, and 14% were still off work. Also showing the lack of value of cervical orthosis, Dehner et al. observed no difference in outcome at 2 and 6 months after treatment in patients immobilized for 2 versus 7 days [61].

Although there is no doubt that remaining active produces better outcomes then rest and immobilization, the usefulness of formal physical therapy (PT) is not entirely clear. There are data that suggest patients who utilized more health care in the first 30 days after whiplash injury had slower recovery than did those who used less care [62]. On the other hand, several studies have shown improved outcome with active PT [63–66].

Dehner et al. reported a randomized trial in 70 grade II whiplash injury patients (neck pain plus either decreased range of motion or point tenderness, but no neurological deficit) who were randomized to active versus passive PT and compared both groups with a nonrandomized "act as usual" group [63]. The median reduction in pain was significantly greater in the active PT group than the passive PT group, and both PT groups had better pain relief and a shorter period of disability than the "act as usual" patients.

Amirfeyz et al. found that two-thirds of patients with whiplash injury who began PT within 3 months of the MVC improved significantly [64]. Although there was no control group, the authors felt the improvement was significantly better than literature-based natural history. Therapy consisted of posture advice, graded activities, stretching, range of motion exercises, and strength training.

Vassiliou et al. compared immobilization with a soft collar for 7 days to 10 sessions of PT [65]. Both groups were given diclofenac and ranitidine. The therapy group was given heat, "lymph drainage massage," and active exercises with an elastic resistance band at each visit. They were also assigned home exercises for 20 minutes each day between visits, although self-reported compliance was pretty poor. At 1, 6, and 26 weeks, the therapy group had clinically and statistically better improvements in pain and disability. It cannot be determined if the differences were due to the interactions with the therapists, treatment in therapy itself, or the home exercise program.

Rosenfeld et al. compared active intervention to rest, immobilization, and a soft collar for acute whiplash injury patients [66]. Active treatment consisted of information, postural control, and cervical range of motion exercises which were continued at home. The 63% of patients who were not improving after 20 days received additional treatment using McKenzie principles. Pain intensity and sick leave were significantly reduced in the active therapy group compared with standard care.

Several reviews found no evidence that spinal manipulative therapy (SMT) used alone or in conjunction with modalities was useful [67], but SMT combined with exercise can be beneficial.

In summary then, it appears that the treatment for acute whiplash injury that is most likely to improve outcome is to explain the condition and favorable natural history; recommend remaining active; offer a nonsteroidal anti-inflammatory drug (NSAID); avoid prescribing a cervical collar; and prescribing active and progressive PT that stresses exercise with emphasis on a home or gym program, and accompanied by postural correction. The use of mobilization for symptom control is reasonable for 7 to 10 days.

TREATMENT: CHRONIC NECK PAIN

Rehabilitation is the treatment prescribed most often for whiplash injury, although the evidence is neither strong nor specific. The treatment with the highest level of evidence is RFN for facet joint pain [68]. Although used frequently, there are few indications for epidural corticosteroids or trigger point injections for axial neck pain. The role of SMT remains controversial, but despite its popularity and advocates, the evidence of efficacy in chronic axial neck pain is at best mediocre [69,70].

Rehabilitation for Chronic Neck Pain due to Whiplash Injury

Many experts recommend and most clinicians prescribe exercise and ergonomic training as a first step for patients with chronic whiplash injury. However, the value of rehabilitation for chronic whiplash injury has not been studied sufficiently and experts differ regarding its value. However, all agree that exercise is safe despite pain. Hurwitz et al. felt the evidence was not sufficient to draw a conclusion regarding exercise therapy [67]. Bannister et al. felt that all treatments for late whiplash injury are relatively ineffective [5]. Bogduk felt the data supported the use of exercises, but emphasized that exercise only might reduce the level

of pain, not eliminate it [6]. That said, the clinician seeking to help the patient must rely on the best available evidence, which is sometimes only expert opinion, weigh the potential risks versus possible benefits, and then make treatment decisions. Based on these parameters, it is reasonable guidance to recommend PT for chronic whiplash injury.

There is logic to the prescription of exercise therapy for whiplash patients as there is for all patients with chronic neck pain. There is evidence that neck muscles are weaker in chronic whiplash injury patients than in normal controls, and patients with nonspecific neck pain have similar deficiencies in muscle strength and coordination as whiplash injury patients [71–74]. There is also evidence for increased muscle tone and impaired movement control in chronic whiplash injury patients [74]. Neck muscles provide significant support for deeper structures. Weakened neck muscles may lead to increased stresses on cervical discs or facet joints.

Rehabilitation exercise directed at the cervical spine does appear to help some patients. The mechanism of improvement is probably multifactorial. Exercise has the potential to increase strength, endurance, movement control, and abnormally increased muscle tone. In addition, patients with chronic neck pain may develop a fear-avoidant coping style. Supervised exercises followed by independent gym or home exercise can improve this abnormal fear response and thereby increase function and decrease pain.

Exercise: Whiplash Patients

There are studies in patients with subacute and chronic whiplash injury pain. Stewart et al. compared exercise plus advice with advice alone in 134 whiplash patients with neck pain of 3 to 12 months duration [75]. The exercise was not standardized. Instead it was left to be "individualized by the treating therapist." There is no mention of specific neck strengthening or endurance exercises. Therefore, the exercise group appears to have received a program that might be expected to be less effective. That said, at 6 weeks, the exercise group had significantly better reductions in pain and impairment, but these results were no longer significant at 12 months. Patients with high levels of baseline disability had greater treatment effects at the 12-month evaluation.

Bunketorp et al. compared supervised training with a home training program in 47 patients with subacute whiplash injury [76]. In the 40 patients who completed the study, supervised training was more effective than home training for all parameters studied at 3 months. Results were partially maintained at 9 months. There was no reduction in sick leave.

Exercise: Chronic Neck Pain Unassociated with Whiplash Injury

Chronic neck pain due to whiplash and neck pain unassociated with whiplash injury have been shown to share similar deficits in strength and similar patterns of dysfunction [72,73]. Therefore, it is clinically reasonable to expect that exercise would have the same effect in both sources of neck pain. The details of cervical rehabilitation are discussed elsewhere in this text, and therefore only a brief overview of some recent papers will be presented here.

Ylinen et al. performed a randomized controlled trial to compare strength training, endurance training, a program of stretching, and aerobic exercise in female office workers [77]. Patients were treated in a multimodal program for 2 weeks and then given a 12-month home exercise program [69]. At 1 year, both the strength training and endurance training groups had clinically and statistically significant improvements in neck pain, disability, and range of motion compared with the control group. In a later publication, they showed that the benefits were generally sustained after 3 years despite the fact that exercise compliance was fairly low [78].

Andersen et al. reported a randomized controlled trial that compared 48 women with neck pain treated with 10 weeks of specific neck strength training, general fitness training, or an intervention with no physical component [79]. There was a clinically and statistically significant improvement in pain in the specific neck strengthening group compared with the other two groups.

Griffiths et al. compared specific neck stabilizing exercises with a more general neck exercise program in 74 patients with chronic nonspecific neck pain [80]. Both groups were given postural advice and range of motion exercises. In addition, the stabilization group was given two additional cervical flexion exercises. Both groups improved, and there were no differences between the groups at 6 weeks or 6 months.

Other studies reached similar conclusions. Strength training was shown to reduce pain, improve range of motion, improve function, and decrease disability when compared with placebo therapy [81]. Intensive exercises are more effective than light exercises [82] but not necessarily more effective than ordinary activity [83]. An intensive therapist-directed exercise program with relaxation training and behavioral support proved superior to a neck lecture plus information and instruction for home exercises. Both proved superior to a lecture plus advice to exercise. An uncontrolled study showed that a multimodal behavioral and PT program could reduce pain, analgesic use, disability, and subsequent medical visits, although the reductions in pain were only modest [84]. A tertiary rehabilitation program for injured workers with disabling cervical spine disorders found high rates of return to work and improved long-term outcome in many dimensions [85].

In summary, the literature reflects a strong trend toward the value of neck strengthening and endurance exercise for whiplash injury patients as well as patients with neck pain of other sources regardless of the duration of pain. A single randomized controlled study showed no benefit for exercise in chronic whiplash injury, but it does not appear that the patients were given neck-specific exercises. Multiple observational studies have shown distinct benefit for whiplash patients and patients with nonspecific neck pain treated with neck strengthening exercise.

Expert opinion after literature review is somewhat inconsistent. We have observed reasonably good outcomes for many, but not all, whiplash injury patients [86]. It is clear that there is no risk to the use of supervised and then home exercise. There are few other treatment alternatives that have been proven effective. Therefore, reasonable guidance would suggest using neck strengthening and endurance exercise for treatment.

The literature does not provide sufficient guidance as to the most effective set of exercises. In my opinion, exercise should be directed toward strengthening the muscles that are usually weak in whiplash injury patients—the deep and more superficial anterior muscle groups, the interscapular muscles, lateral neck muscles, and perhaps the posterior muscles. Endurance training is equal to pure strength training. I prefer prescribing isometric exercise, which may be easier for patients to perform on their own without supervision after instructions. In addition, exercise training should be accompanied by training in posture and body mechanics. As mentioned earlier, in addition to increasing muscle strength, exercise serves the very important role of helping patients overcome fear and fear-avoidant behaviors.

Percutaneous Procedures

Radiofrequency Neurotomy

The treatment with the best evidence in neck pain due to whiplash is RFN [68]. In a systematic review, however, Carragee et al. felt the evidence of efficacy for cervical RFN was insufficient [87]. RFN is the heating of the medial branches. It must be noted that they do not claim RFN does not work. Carragee et al. dismiss the RCT by Lord et al. as flawed and find that there is insufficient evidence of proof of efficacy of RFN. They offer no suggestions for other treatment and do not comment that most reviewers, clinicians, and experts feel there is sufficient proof of efficacy, nor has there been any research to show lack of efficacy. Dreyfuss and Baker have offered an elegant and convincing rebuttal of the Carragee et al. opinion [88].

Of course, RFN is applicable only to the approximately 60% of chronic whiplash patients with proven facet joint pain. RFN is discussed elsewhere in this text (see Chapter 36). RFN is the coagulation of the medial branches of the dorsal rami that conduct electrical impulses from the joint, and therefore have the capacity to transmit signals from the affected joint which may be experienced as pain. The single indication for RFN is significant relief of pain following (usually two) controlled blockade of the medial branches of the nerve supply to the joint. In those with good response to MBB, meaningful relief of pain can be achieved in about 70% of patients including those with facet-mediated CHA [39,68]. Relief lasts a median of 270 to 400 days, and when benefits dissipate, repeat RFN is usually successful [68,89,90].

There is no evidence that cervical epidural corticosteroid injections are useful for the relief of axial neck pain. Intra-articular injections of corticosteroids are not effective for the long-term treatment of cervical facet joint pain, but may provide short-term relief during which rehabilitation may be more effective [91].

Medications

Medications can play a role in the treatment of neck pain after MVC [92]. In acute neck pain, the most useful drugs are the NSAIDs, opioid analgesics, and muscle relaxants for short-term use. Opioid analgesics can be helpful to help keep patients active during the acute phase. Intravenous corticosteroids given in the first hours after whiplash injury have been shown to improve outcome. Anecdotally, oral corticosteroids can be helpful for the patient who has not responded to more commonly used medical therapies, but there are no published studies regarding efficacy. I favor 60 mg of prednisone in divided doses with 14-day taper to zero.

In chronic neck pain, the response to NSAIDs is not predictable. It is appropriate to try several NSAIDs for up to 2 weeks each in healthy patients with low risk for systemic side effects. If there is a meaningful response and no significant side effects, continue that drug, but if there is minimal response to three trials, further NSAID trials are not likely to be helpful. Although controversial, opioids have a role for well-selected patients with chronic moderate to severe neck pain who have proven refractory to other treatments. The tricyclic antidepressants have been shown to be useful in chronic axial LBP, and only by inference, chronic neck pain. Drugs such as nortriptyline starting at 10 mg at night with gradual dose increases to a target of 50 to 75 mg may provide analgesia in about one-third of patients. Anticonvulsants are helpful in neuropathic pain, but have not been shown to be helpful in axial pain.

Spinal Manipulative Therapy

In their systematic review, Gross et al. concluded that mobilization and/or manipulation combined with exercise are beneficial for chronic mechanical neck disorders [69]. However, manipulation and/or mobilization without exercise were not beneficial. Hoving et al. compared SMT, exercise therapy, and routine care by general practitioners for patients with mechanical neck pain [69]. At 7 weeks, SMT was somewhat superior to exercise and significantly better than general practice care. However, at 13 weeks and 1 year, there were no significant differences.

Surgery

Surgery is rarely indicated in the treatment of chronic whiplash patients who have predominant axial neck pain, and should only be considered after failure of high-quality rehabilitation, interventions, and medical care have failed. Specific analysis for possible surgery for axial pain requires plain radiographs with flexion and extension views, magnetic resonance imaging scan, and rarely discography. Although controversial, when interpreted in conjunction with history, examination, and other testing, discography may help isolate one or two painful discs. If there is a

negative MBB, concordant pain reproduction at the index level, no pain upon stimulation of adjacent levels, then discography results may be helpful.

The most common indication for surgery in chronic whiplash is severe refractory neck pain or headache. There are no controlled studies regarding efficacy, but several longitudinal and retrospective studies suggest reasonable outcomes [56,57]. These studies do not focus exclusively on whiplash patients, but on axial neck pain in general, although all included patients with whiplash, however.

Garvey et al. reported a 4-year follow-up of 87 (including 25 with chronic whiplash) patients who underwent ACDF for axial neck pain [56]. They obtained 83% good to excellent results with statistically significant improvements in both Oswestry and modified Roland-Morris disability indices. Palit reported 38 patients who had ACDF for axial neck pain, and noted significant improvements in pain and Oswestry disability index, and 79% were satisfied with their outcome [57]. Similar findings were reported for ACDF at C2-3 and C3-4 for discogenic cervical headaches in a study with small numbers [93].

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38 Algorithmic Application to Cervical Radicular Pain

Zacharia Isaac, Elizabeth Beck, and Curtis W. Slipman

INTRODUCTION

Efficient and systematic evaluation of the patient with cervical radicular pain is important for timely, accurate diagnosis and effective treatment. To be effective in the assessment of these patients, the clinician must have extensive knowledge of radicular pain itself and all of its imitators to generate a comprehensive differential diagnosis. The thorough evaluation of cervical radicular pain utilizes historical and examination features, electrodiagnostic (EDx) testing, diagnostic imaging, and diagnostic anesthetization. The objective of the use of the aforementioned tools is to achieve an accurate diagnosis allowing for individualized treatment and therefore better functional outcomes.

Cervical radiculopathy is defined as dysfunction of a cervical spinal nerve root with associated myotomal deficit, reflex change, or an abnormal electromyographic finding. In contrast, cervical radicular pain is simply pain that refers in a dermatomal or dynatomal pattern, without the obligation of a neurologic deficit. As such, radicular pain can be diagnosed without a neurologic deficit, and radiculopathy may be present without radicular pain. The distinction between radiculopathy and radicular pain is illustrated anatomically. Motor deficits arise from involvement of ventral nerve roots carrying motor efferent fibers from the alpha motor neurons in the ventral horn of the spinal cord. Painful sensory symptoms occur because of involvement of dorsal nerve roots carrying primary sensory afferent fibers from cell bodies in the dorsal root ganglion. In practice, both dorsal and ventral spinal roots are generally involved; however, patients can present with predominantly motor or predominantly sensory symptoms.

PATHOPHYSIOLOGY OF RADICULAR PAIN

The major pathway thought to lead to radiculopathy is inflammation from age-associated degeneration. This degeneration begins with the cervical intervertebral disc. Studies in lumbar intervertebral discs have found elevated levels of phospholipase A2, interleukins, and immune cells [1–6]. These chemical pathways may affect cervical disc herniations as well. Biochemical pathways involving these cytokines, cell-mediated immune responses, and the arachidonic acid cascade are involved in the pathophysiology of radicular pain. The inflammatory cascade leads to increased permeability of intraneural blood vessels with resultant nerve root edema. Inflammation of the dorsal root ganglion and chronic nerve root edema and fibrosis lead to increased excitability by decreasing the stimulation threshold. Cervical radicular pain can also occur in the absence of significant degenerative changes in patients with a history of trauma, like whiplash. There is limited literature focusing on nerve root injury secondary to a whiplash event. However, existing data raise the notion that traumatic radiculopathy may not be immune mediated.

CLINICAL ASSESSMENT: HISTORY

Cervical spine symptoms should be divided into three categories: predominantly axial pain, predominantly limb pain, or myelopathy. Making this categorization can be challenging, as most patients present with a combination of the aforementioned symptoms; however, doing so is helpful for generating a differential diagnosis. Cervical radicular pain often presents with upper limb pain that is worse than axial neck pain. Exacerbating positions include sustained neck position, neck extension, neck rotation, and ipsilateral limb abduction with elbow extension. Symptoms are often alleviated by positional shifting, maintaining a neutral posture with avoidance of neck rotation, and overhead abduction with elbow flexion, also known as Bakody's sign [7] (Figure 38.1). Once cervical radiculopathy is clinically suspected, identifying the symptomatic nerve root begins with the patient's description of the areas of pain, traditionally thought to follow a dermatomal pattern. Dermatomal mapping is well described and began in the 19th century by Sherrington [8,9]. However, patients with radiculopathy may have symptoms outside of the dermatome implicated by imaging studies; this is known as dynatomal referral pattern. Dynatomes have been studied for cervical nerve roots C4 through C8 [10] (Figure 38.2). The discrepancy between dermatomal and dynatomal maps is attributed to the high incidence of intrathecal anastamoses between cervical spinal nerve roots [11].

Painful cervical intervertebral discs and facet joints cause predominantly axial neck pain but can refer symptoms into the shoulder girdle complex and upper limb. Generally, facet joint-mediated pain does not refer beyond the elbow (Figure 38.3). In contrast, discogenic pain can

Figure 38.1 Bakody's overhead abduction sign. Patients with cervical radicular pain due to a herniated disc experience relief of discomfort in this position because of less tension on the cervical spinal nerve root.

refer distal to the elbow [12] (Figure 38.4). This pain referral pattern without nerve root compression is termed somatic referral. This referred pain is thought to be mediated by central mechanisms. Patients with this syndrome will often describe axial pain to be greater than limb pain, in contrast to radicular pain that is greater in the limb than the axial pain. Identifying whether the referred symptoms are secondary to radicular pain versus somatic referral can often be ascertained through clinical assessment.

Identification of myelopathy is critical because neurologic outcomes are improved with surgical intervention. Patients with myelopathy can present with different neurologic deficits than those with radiculopathy. In cervical myelopathy, upper motor neuron symptoms predominate secondary to injury to the spinal cord. Transverse lesion syndrome involves all spinal tracts to a roughly equal degree. Anterior cord syndrome results in motor weakness with sensory sparing and is most often due to anterior spinal artery infarction. Central cord syndrome affects sensory and motor in the upper limbs to a greater degree than lower limbs and is seen in syringomyelia or hyperextension trauma. Brown-Sequard syndrome is commonly due to trauma and results in ipsilateral loss of motor strength, vibration, and proprioception with contralateral loss of pain and temperature. Myeloradiculopathy is concomitant radiculopathy and myelopathy.

AN ALGORITHMIC APPROACH

Treatment of cervical radicular pain involves a variety of options which include patient education, activity and postural and worksite modifications, therapeutic exercise, adjunctive modalities, cervical traction, pharmacologic measures, fluoroscopically guided injection procedures, and surgical intervention. Whenever possible, treatment should be individualized to improve patient outcomes

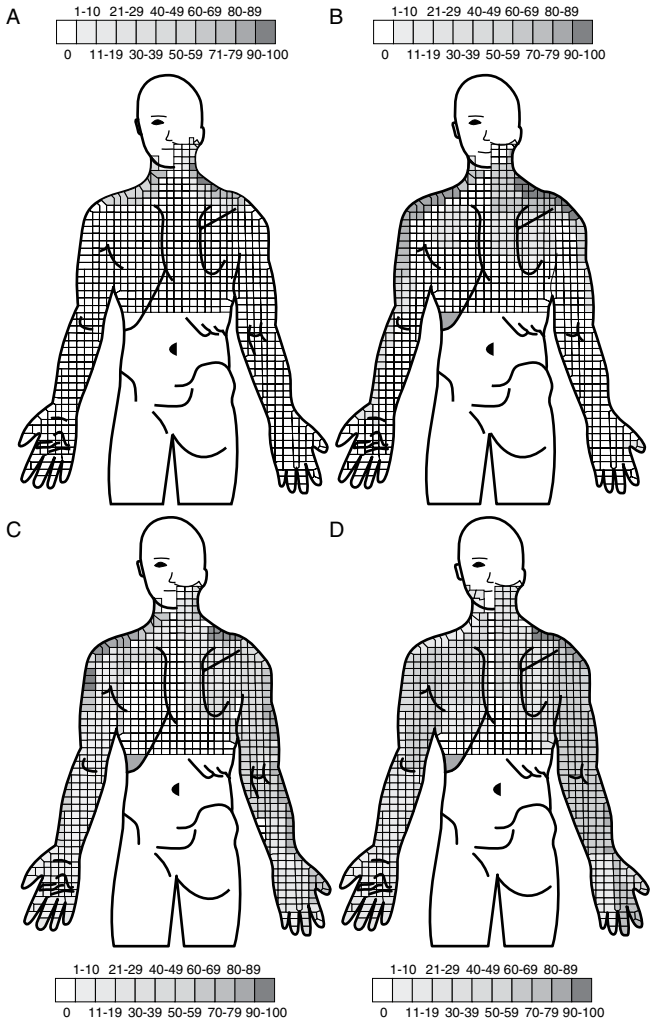

Figure 38.2 Dynatomal maps for cervical spinal nerve roots C4-7: pain referral patterns for cervical spinal nerve roots undergoing provocative stimulation. **(A)** is spinal nerve root C4; **(B)** is spinal nerve root C5; **(C)** is spinal nerve root C6; and **(D)** is spinal nerve root C7.

(Figures 38.5–38.7). When historical and examination features have corroborative imaging, the diagnosis is not a dilemma. In cases where a myotomal deficit or a depressed reflex is present, these are often the most helpful clues to determine the affected spinal nerve root. Sensory disturbances are much less helpful because of the dual dermatomal innervation of a given skin segment. Unfortunately, it is quite common that the affected nerve root is in question, and there is a lack of corroborative imaging abnormalities, or multiple compressive abnormalities are present. In such situations, EDx studies, including electromyography and nerve conduction studies, can help identify physiologic evidence of nerve root injury (Figure 38.7). EDx studies can be helpful to identify the affected nerve root, and differentiate other diagnoses that can mimic radicular pain, such as peripheral nerve entrapments, neuropathy, and plexopathy. When electromyography is also inconclusive, functional testing using injections of local anesthetic can identify the pain generator. Diagnostic selective nerve root

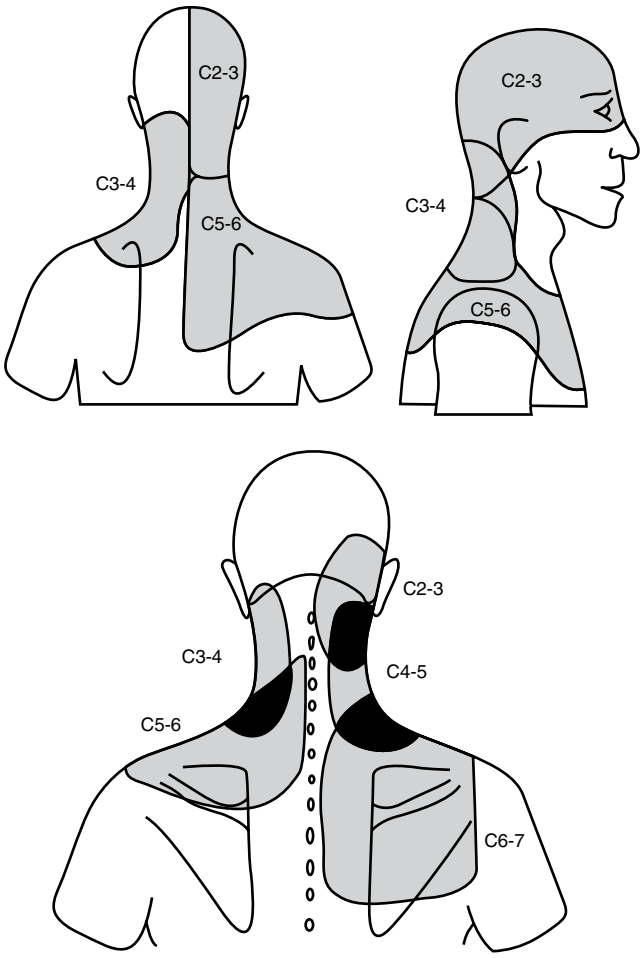

Figure 38.3 Facet joint pain referral patterns for cervical facet joints C2-3 through C6-7.

blocks can confirm radicular pain and help identify the specific root involved (Figure 38.8 and Table 38.1). The pain referral pattern should be compared with known dermatomal and dynatomal maps to help prioritize the most likely affected nerve root.

Patients with cervical spine complaints can be categorized into four groups that are determined on the presence or absence of spondylotic changes and whether or not there is a history of trauma. Four categories emerge: (1) atraumatic spondylotic radicular pain, (2) traumatic spondylotic radicular pain, (3) traumatic nonspondylotic radicular pain, and (4) atraumatic nonspondylotic radicular pain.

Atraumatic Spondylotic Radicular Pain

The incidence of degenerative cervical intervertebral disc changes increases with age with more than half of the adult population demonstrating radiologic evidence of cervical spondylosis [13,14]. Only a minority of these individuals become clinically symptomatic. The etiology of axial neck pain is often secondary to musculoskeletal sprain and strain, discogenic pain secondary to cervical

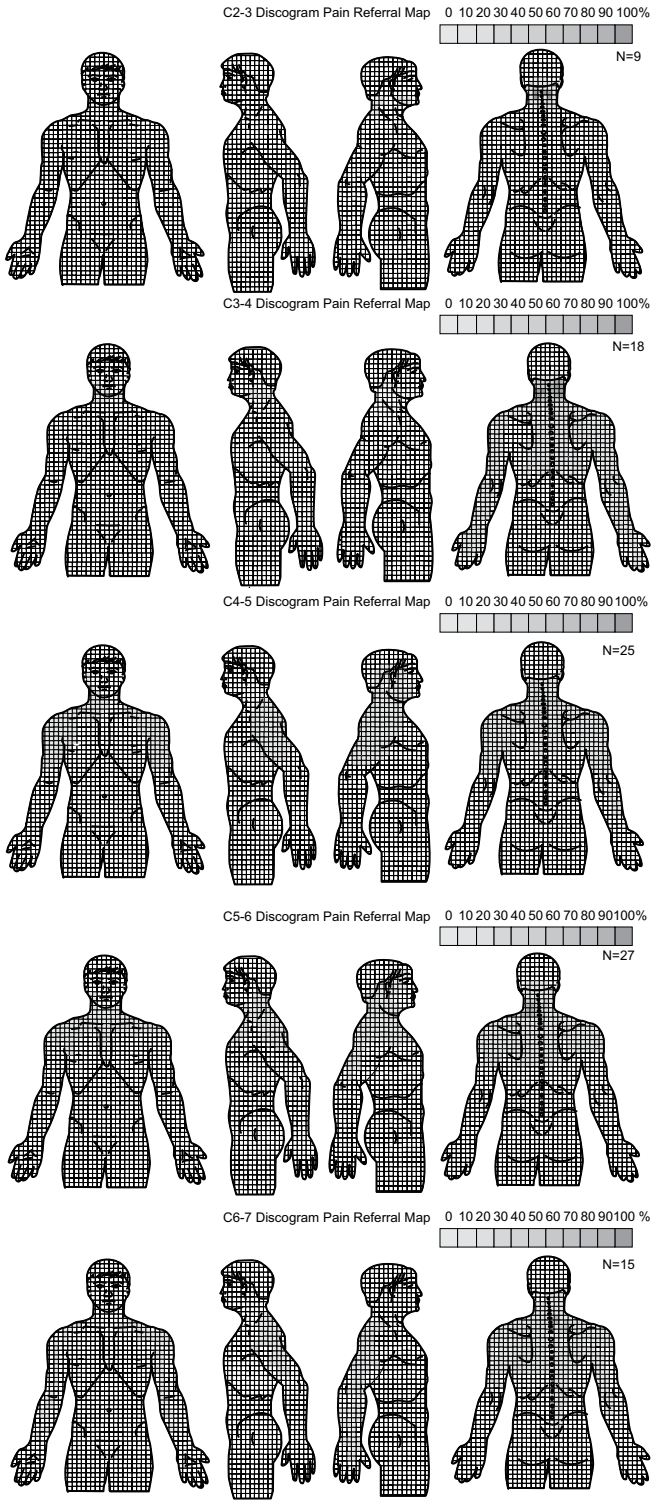

Figure 38.4 Cervical discogenic pain referral patterns based on provocative discography.

internal disc disruption syndrome, or cervical facet syndrome. In contrast, scapular and upper limb symptoms are more commonly attributable to the cervical spinal nerve root or spinal cord itself.

Many patients with atraumatic spondylotic cervical radicular pain present with corroborative imaging findings.

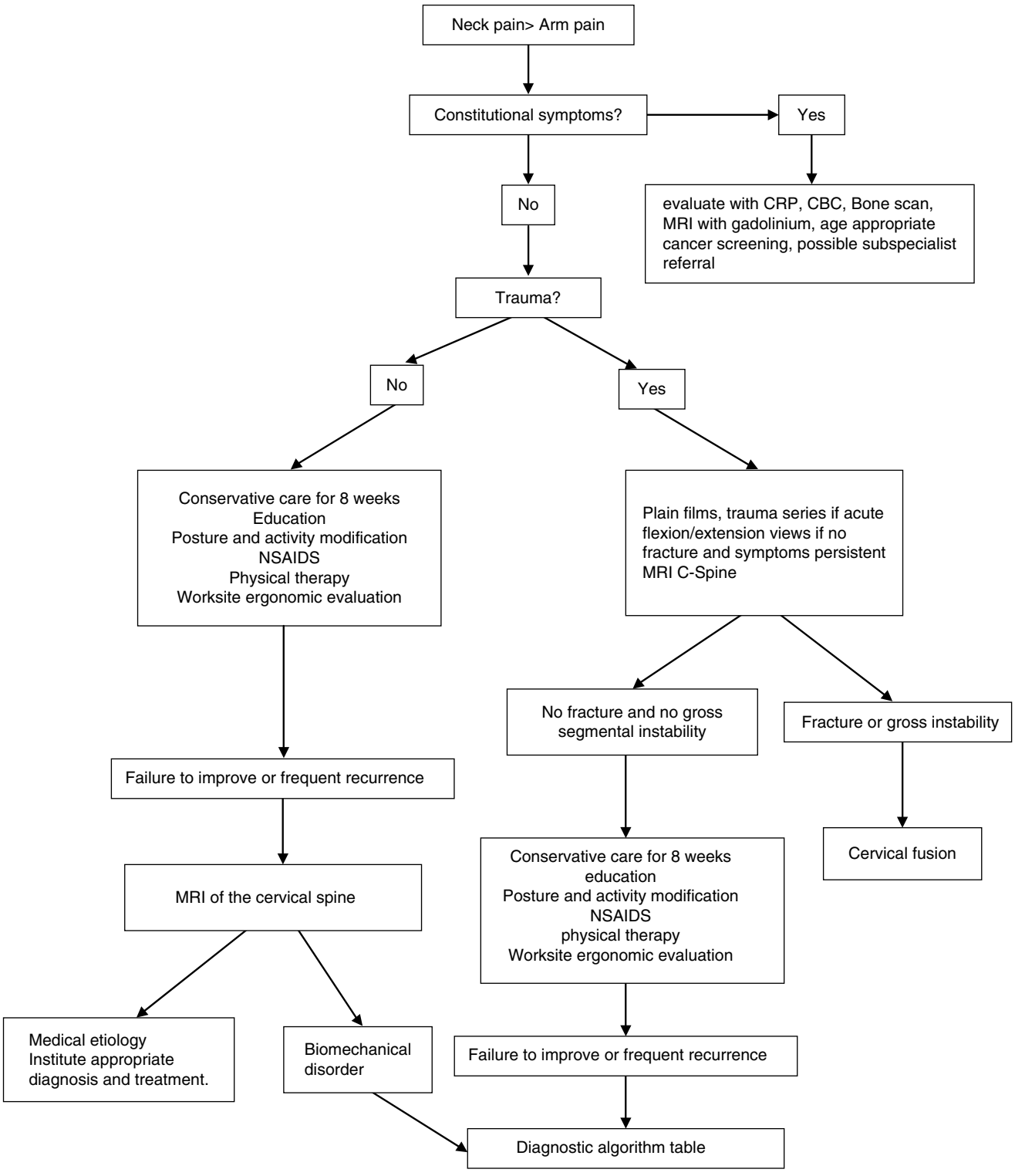

Figure 38.5 General algorithm: Patient with axial symptoms greater than extremity symptoms.

In the presence of severe weakness and/or progressive weakness, surgical decompression is considered the intervention of choice. Patients should be asked as to whether they are aware of progressive weakness or functional deficits relating to the affected limb. In the absence of progressive weakness, conservative options such as pharmacologic measures, physical therapy, and injections targeted toward the affected spinal nerve root can be pursued. If conservative measures fail to bring adequate pain relief and restoration of function, surgical options may be considered.

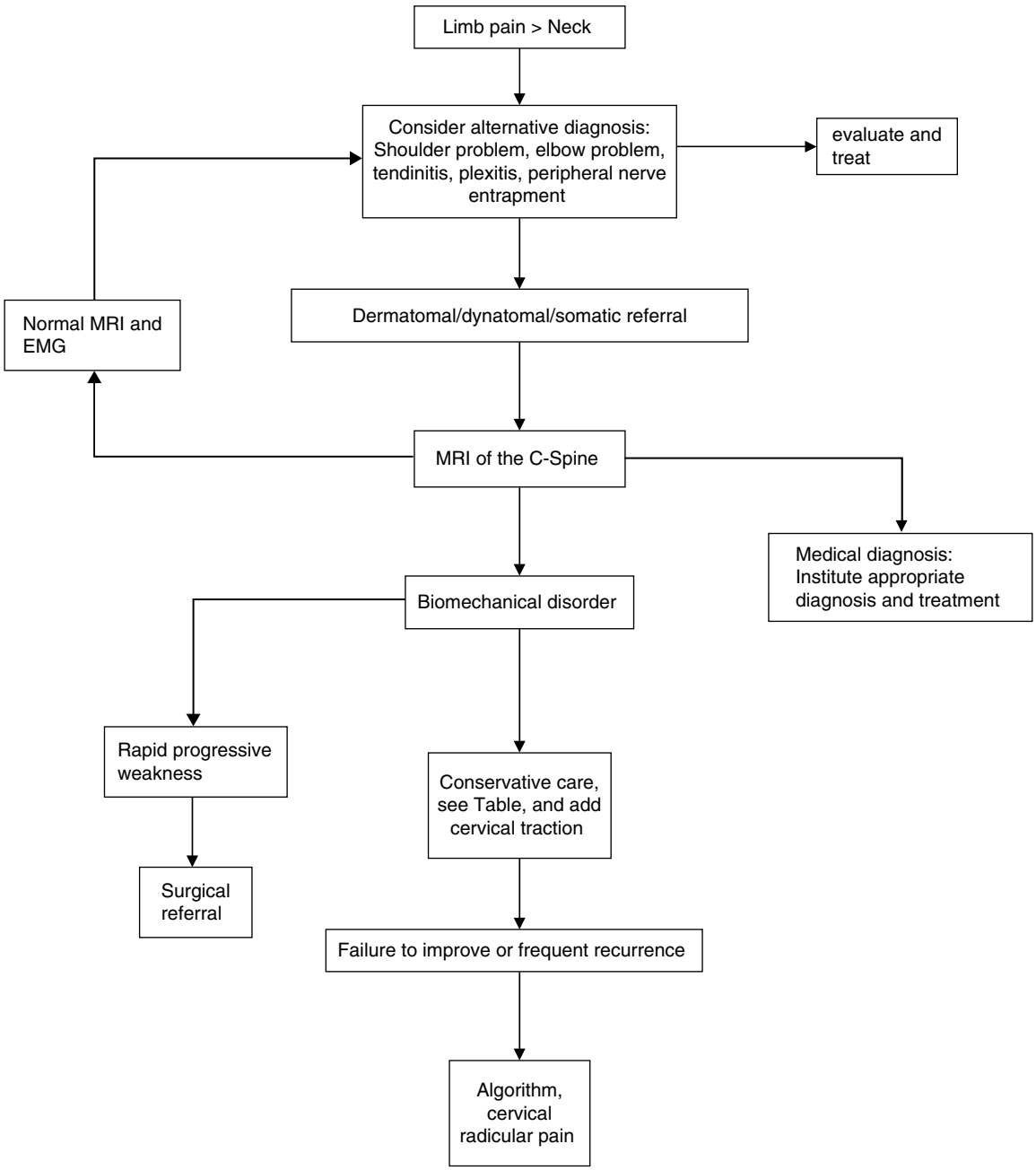

Figure 38.6 General algorithm: Limb symptoms greater than axial symptoms.

Traumatic Spondylotic Cervical Radicular Pain

In patients with traumatic cervical radicular pain, multiple spinal and extraspinal structures may be injured. Degenerative spondylotic changes on imaging sometimes mislead the clinician as to the pain generator because they can predate the injury. A new herniated disc on imaging may explain the symptoms, but this is the case in only a minority of patients. Spinal structures such as the muscles, tendons, and ligaments of the spine, cervical zygapophyseal joints, spinal nerve roots, and the intervertebral disc itself can become injured and cause axial neck pain with or without an limb component which may mimic radicular pain. The shoulder, elbow, and wrist can be concomitantly injured and mimic or be present along with radicular pain. When making a diagnosis, the law of parsimony is applied; however, this rule could lead the spine clinician astray when evaluating a patient who sustained trauma. When evaluating posttraumatic cervical radicular pain, attention should be paid to underlying degenerative changes that may provide clues as to the spinal nerve roots that are prone to injury. The patient's pain referral pattern should be compared with known dermatomal and dynatomal maps, and examination should attempt to identify neurologic findings that

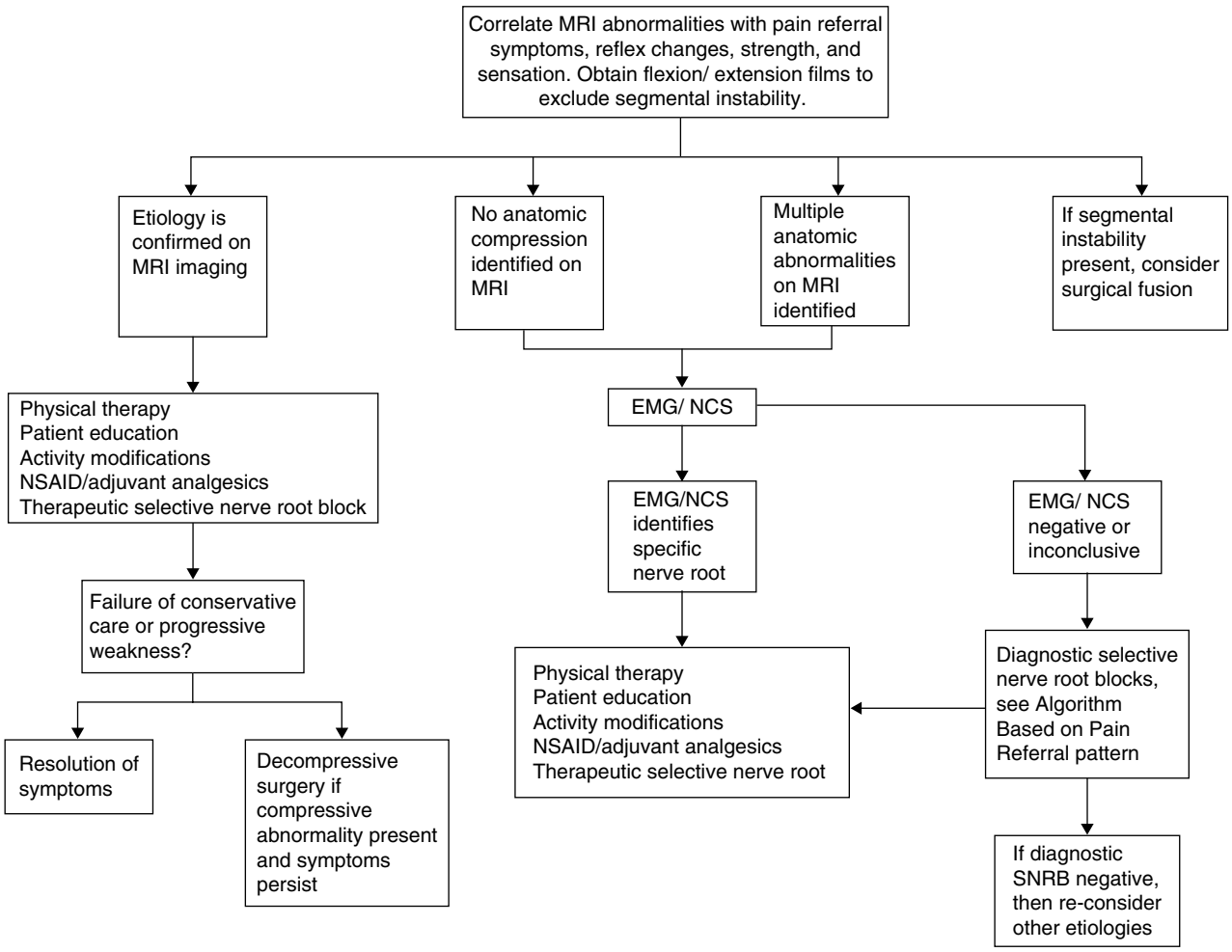

Figure 38.7 Algorithm: Cervical radicular pain.

are of clinical relevance. Electromyographic studies may be helpful to determine if radiculopathy is present; however, in cases where reflexes and myotomal strength is normal, electromyographic studies are often negative. Fluoroscopically guided diagnostic selective nerve root anesthetization may be helpful in establishing a diagnosis of cervical radicular pain when other workup is unrevealing. Upon establishment of a diagnosis, therapeutic interventions such as pharmacologic measures, physical therapy, and therapeutic spinal injections can be implemented. If the affected nerve root has associated spondylotic changes that may be implicated as a compressive abnormality, surgical decompression can be considered in the event of failure of conservative care. However, in cases where posttraumatic radicular pain is present without evidence of an anatomic abnormality on magnetic resonance imaging (MRI) or computed tomography (CT) myelography, no specific surgical intervention is available. These patients may ultimately benefit from neuromodulation (Chapter 43).

Traumatic Nonspondylotic Cervical Radicular Pain

Determining the pain-generating structure in patients with suspected cervical radicular pain without significant cervical spondylotic changes can be difficult. Injury to a variety of spinal and extraspinal structures may occur. In patients with a defined myotomal deficit or reflex change, diagnosing the affected nerve root can be straightforward, but often these examination features are not present. The differential diagnosis is similar to that of spondylotic cervical radicular pain; however, knowledge of pain referral maps for cervical discogenic pain, cervical facet syndrome, and cervical dermatomal and dynatomal maps for radicular pain are often the only clues to defining a diagnosis. MRI and EDx studies are usually negative.

Atraumatic Nonspondylotic Radicular Pain

The differential for patients with atraumatic, nonspondylotic cervical radicular pain is very broad and involves

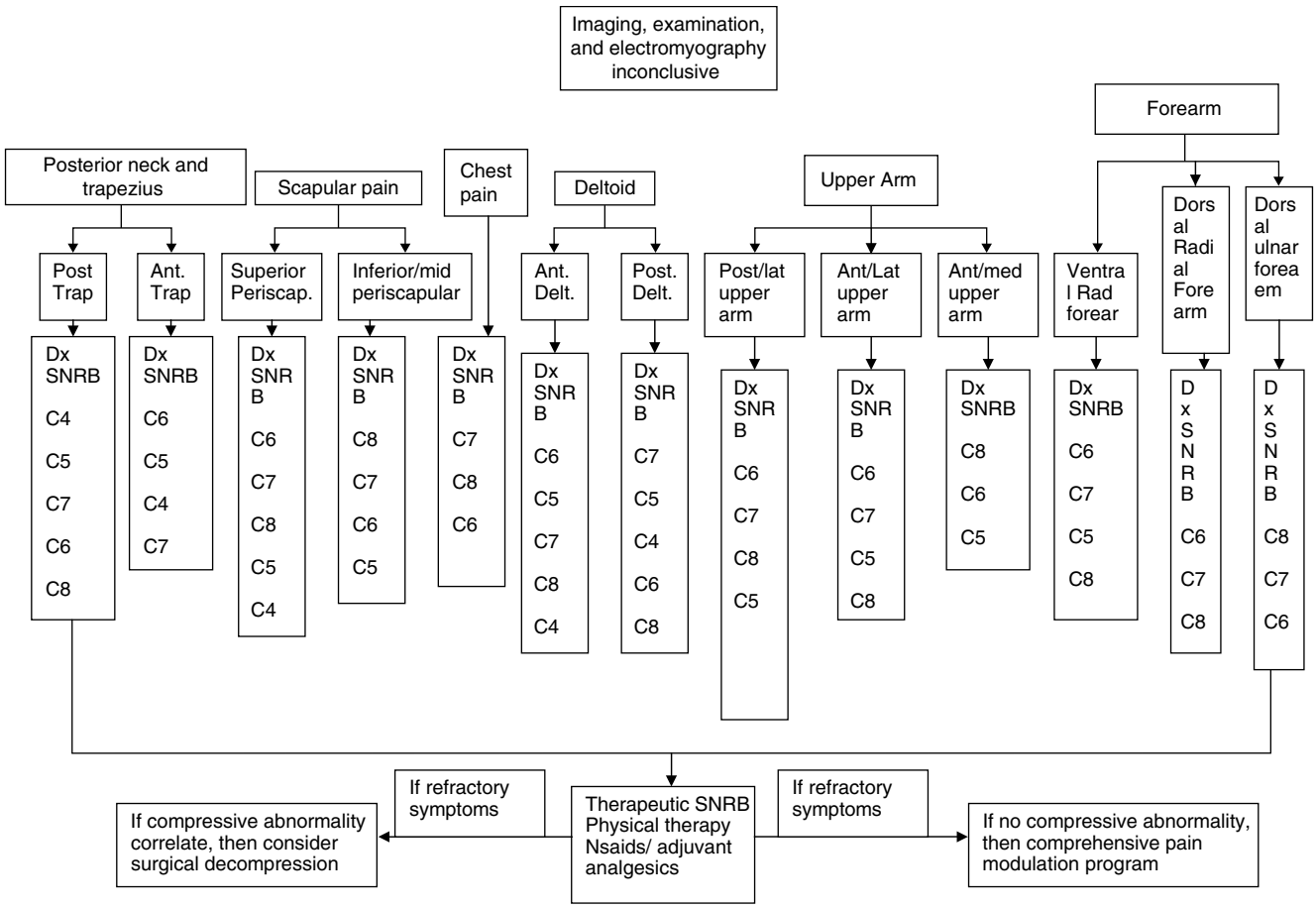

Figure 38.8 Algorithm: Pain referral pattern.

both spinal and extraspinal diagnoses. Peripheral nerve entrapment of the median, radial, or ulnar nerves can cause extremity pain. Brachial plexopathy may occur because of malignancy, radiation, neurotoxic chemotherapeutic agents, viral syndromes (Parsonage-Turner), thoracic outlet syndrome (vascular or neurogenic), or vasculitis. Intrinsic shoulder problems such as impingement syndromes and rotator cuff tears, and suprascapular neuropathy, can cause shoulder, upper arm, and trapezius symptoms. Central nervous system lesions, demyelinating diseases such as multiple sclerosis, cerebrovascular accidents involving the sensory cortex, or the thalamus (Dejerine-Roussy thalamic pain syndrome) can cause limb pain. Complex regional pain syndromes type 1 and type 2 can cause limb pain, hallmarked by allodynia, color changes, edema, and skin and nail changes. Pulmonary, gastrointestinal, cardiac, and vascular etiologies can refer symptoms to the shoulder, scapula, chest wall, and to the extremity.

Pain Referral Patterns

See Table 38.1 for specific referral patterns of individual nerve roots.

Posterior Neck and Trapezius

Symptoms in the region of the posterior neck and trapezius can be seen in medical conditions such as spinal infection and spinal malignancy or referred from a thoracic or abdominal visceral process. Symptoms can also stem from mechanical neck disorders such as facet syndrome, internal disc disruption syndrome (C3-T1) (Figure 38.4), or referred pain from the shoulder. Neurologic disorders including brachial plexopathy, thoracic outlet syndrome, and cervical radicular pain can follow a similar referral pattern. Trapezius symptoms can be referred from nerve roots C4-C8.

Deltoid

The differential diagnosis for deltoid pain again includes the brachial plexus, intrinsic shoulder pathology, mechanical neck pain, and cervical radicular pain from C4 to C8. Intrinsic shoulder pathology including adhesive capsulitis, joint abnormality, fracture, bursitis, tendonopathy, impingement, and scapular instability should all be evaluated. When symptoms distal to the deltoid are present,

Table 38.1 Probability Analysis by Predominant Pain Referral Pattern

Evaluation of scapular pain
Superior periscapular
C6 (51%), C7 (42%), C8 (38%), C5 (29%), C4 (25%)
Inferior/middle scapular
C8 (24/29%), C7 (17/17%), C6(9/16%), C5(7%/7%)
Evaluation of neck and trapezius
Posterior trap
C4 (100%), C5 (92%), C7 (77%), C6 (74%), C8 (52%)
Anterior upper trap
C6 (58%), C5 (50%), C4 (50%), C7 (37%)
Evaluation of deltoid pain
Posterior deltoid
C7 (79%), C5 (79%), C4 (75%), C6 (74%), C8 (71%)
Anterior deltoid
C6 (74%), C5 (57%), C7 (48%), C8 (43%), C4 (25%)
Evaluation of arm pain
Posterolateral arm
C6 (79%), C7 (65%), C8 (62%), C5 (7%)
Anterolateral arm
C6 (58%), C7 (42%), C5 (29%), C8 (14%)
Anteromedial arm
C8 (43%), C6 (30%), C5 (7%)
Evaluation of forearm pain
Ventral radial forearm
C6 (44%), C7 (23%), C5 (14%), C8 (10%)
Dorsal radial forearm
C6 (67%), C7 (58%), C8 (33%)
Dorsal ulnar forearm
C8 (67%), C7 (42%), C6 (23%)
Evaluation of chest pain
C7 (17%), C8 (14%), C6 (7%)

Patients underwent cervical spinal nerve root stimulation and were questioned about regional referral of pain, and corresponding body regions were recorded. The incidence of a particular body region being associated with pain referred from a given spinal nerve is reported in table.

those referral patterns should be used to create a differential diagnosis.

Scapula

The differential of scapular pain is broad and includes visceral etiologies, brachial plexitis, scapulothoracic dyskinesia (SICK scapula syndrome), suprascapular neuropathy, referred shoulder pathology, and referred discogenic or facet pain from the thoracic or cervical spine via somatic referral or radicular pain.

Upper Arm

Symptoms in the arm are caused by shoulder pathology, plexopathy or thoracic outlet syndrome, visceral pain, cervical radicular pain, and humeral osseous pathology. It is not uncommon for a patient to have symptoms solely in the upper arm as a manifestation of cervical radicular pain.

Forearm and Hand

Forearm pain can be the result of musculoskeletal overuse-related tendonopathy such as medial and lateral epicondylitis, peripheral nerve entrapment involving the median or ulnar nerve, or cervical radicular pain. Median neuropathy is the most common upper limb entrapment neuropathy and may be associated with forearm or hand pain and paresthesia depending on the location of entrapment, but classically involves the first through third digits and the lateral one-third of the ring finger. When ulnar neuropathy is suspected, careful examination is necessary to distinguish from C8 radiculopathy, which would also involve median innervated C8 muscles. Musculoskeletal hand disorders including dequervain's tenosynovitis and osteoarthritis can also cause hand pain. These conditions can be distinguished based on examination features, EDx testing, or diagnostic imaging.

Chest Pain

Numerous organ systems can cause chest pain or discomfort. Etiologies include cardiac, gastrointestinal, vascular, pulmonary, and cervical or thoracic radicular pain. Cervical radicular pain rarely refers to the chest, but may be considered in patients with chest symptoms in the absence of a medical etiology. Thoracic radicular pain often follows a band-like distribution from the back to the anterior chest wall.

CLINICAL ASSESSMENT: PHYSICAL EXAMINATION

The physical examination is important in defining the most likely etiology of symptoms prior to proceeding with diagnostic testing. Examination of suspected radiculopathy includes neurologic evaluation, provocation testing, and a selected musculoskeletal examination. Neurologic assessment includes manual muscle testing, muscle stretch reflexes, and sensory examination. The least reliable of which is the sensory examination, because of dermatomal and dynatomal overlap. Detailed knowledge of the spinal nerve roots and peripheral nerve innervating a given muscle is required to determine the causative structure. Although myotomal overlap exists in upper limb muscles, the following muscles are useful to test with manual muscle testing and/or with needle electromyography. The most specific muscle group is italicized. C4 radiculopathy: *levator scapular* and trapezius, clinically patients may have weakness of shoulder elevation (Figure 38.9). C5 radiculopathy: *rhomboid*, deltoid, bicep, and infraspinatus muscles, clinically may be associated with shoulder abduction and external rotation weakness (Figures 38.10–38.12). Additionally, the bicep reflex may be diminished. C6 radiculopathy can easily be confused for C5 or C7 as weakness can overlap with the C5 or C7 muscles. Muscles affected in a C6 radiculopathy include the following: *infraspinatus*, biceps, brachioradialis, pronator teres, and tricep (Figures 38.13–38.15). The bicep or brachioradialis reflex may be diminished. C7 radiculopathy: this is the most common electrophysiologically identified radiculopathy and can result in weakness of the *tricep*, *pronator teres*, and flexor carpi radialis (Figure 38.16). The triceps reflex is supplied

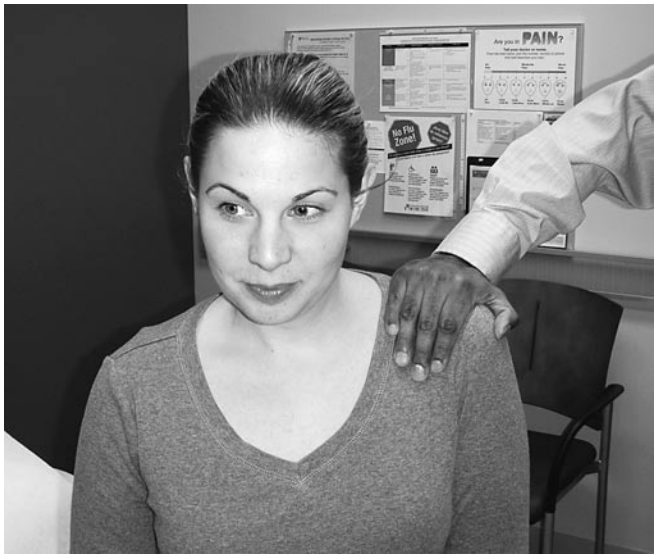

Figure 38.9 Manual muscle testing technique for trapezius C4 myotome. The patient elevates the shoulder against the examiner's downward applied resistance. Commonly, the patient will simultaneously shrug both shoulders, but only one is demonstrated here to illustrate the motion.

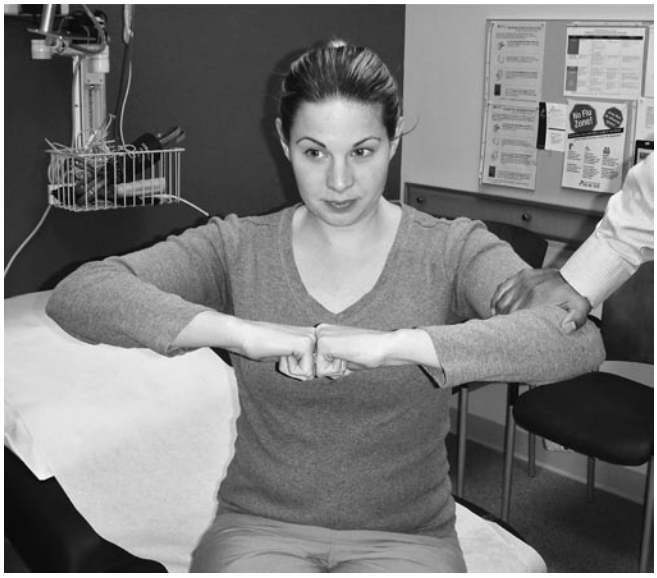

Figure 38.10 Manual muscle testing for deltoid: axillary nerve, C5 myotome. The patient is asked to abduct the shoulder against the examiner's resistance.

Figure 38.11 Manual muscle testing for biceps brachii: musculocutaneous nerve, C5 and C6 myotome. The patient is asked to flex the elbow against the examiner's resistance while the forearm is supinated.

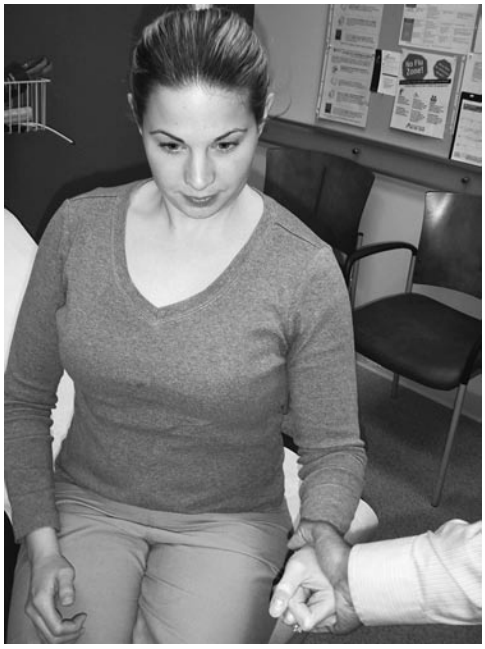

Figure 38.12 Manual muscle testing for infraspinatus: suprascapular nerve, C5 and C6 myotome. The patient is asked to externally rotate the humerus against the examiner's resistance. The examiner must be careful that the patient does not substitute deltoid muscle groups and abduct the shoulder.

by C7. C8 radiculopathy: weakness can be present in the *opponens pollicis*, flexor digitorum profundus, flexor pollicis longus, and hand intrinsic muscles including abductor digiti minimi (Figure 38.17). Clinically, patients present with symptoms similar to an ulnar neuropathy and can have weakness of finger abductors and grip strength as well as a median motor neuropathy. No reliable reflex test is available.

Provocative maneuvers are used with the neurologic examination and attempt to irritate the involved nerve root and reproduce the patient's pain. These maneuvers include modified Spurling's maneuver, Spurling's maneuver, upper limb dural tension, cervical rotation, flexion, distraction, and Bakody's alleviation sign. Select special maneuvers are illustrated in Figures 38.1 and 38.18 to 38.20. There is no single test that is considered

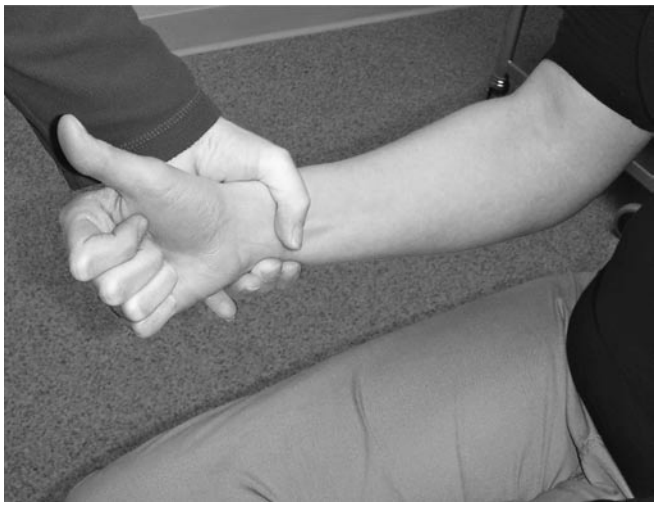

Figure 38.13 Manual muscle testing for brachioradialis, radial nerve, C6 and C7 myotome. The patient is asked to flex the elbow with the radial portion of the forearm pointed upward.

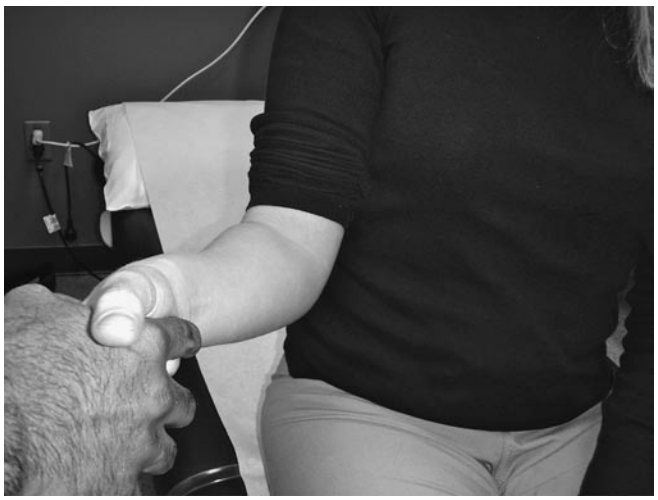

Figure 38.14 Manual muscle testing for pronator teres: median nerve, C6 and C7 myotome. The patient has his/her elbow at approximately 90°, and is asked to pronate the forearm against the examiner's resistance.

Figure 38.15 Manual muscle testing for triceps: radial nerve C6,C7,C8. The patient is asked to extend the elbow against the examiner's resistance. The examiner must be careful not to be fooled by substitution by pectoralis and anterior deltoid muscle groups. The position displayed here helps to isolate the triceps.

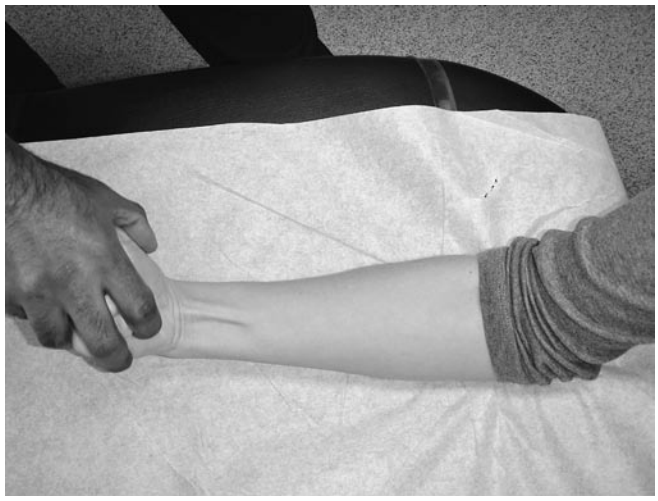

Figure 38.16 Manual muscle testing for flexor carpi radialis: median nerve, C7 and C6 myotome. The patient is asked to flex the wrist against the examiner's resistance. Note the prominent tendon of the flexor carpi radialis.

the gold standard for the diagnosis of cervical radiculopathy, so establishing sensitivities and specificities for each examination maneuver is difficult. A study by Wainner [15] evaluated the correlation of clinical examination abnormalities with electromyographic abnormalities. The investigators found that examination maneuvers associated with acceptable diagnostic accuracy for cervical radiculopathy include the following with respective sensitivities/specificities: Elvey's upper limb tension test A (Figure 38.20) (0.97 sn/0.22 sp), cervical rotation to the involved side less than 60° (0.89 sn/0.49 sp), cervical flexion less than 55° (0.89 sn/0.41 sp), involved bicep muscle stretch reflex (0.24 sn/0.95 sp), neck distraction test reducing symptoms (0.44 sn/0.90 sp), manual muscle testing involved bicep (0.24 sn/0.94 sp), valsalva maneuver exacerbating pain (0.22 sn/0.94 sp), Spurling's test A (Figure 38.18) exacerbating pain (0.50 sn/0.86 sp), shoulder abduction test alleviating pain (0.17 sn/0.92 sp), involved C5 dermatome sensation (0.29 sn/0.86 sp).

The following four tests were analyzed as a test item cluster: Spurling's test A, neck distraction, Elvey's upper limb tension test A, involved cervical rotation less than 60°. The posttest probability for patients with two, three, or four tests positive was 21%, 65%, and 90%, respectively. Another study using EDx examination as the standard by Tong [16] found that the sensitivity of the Spurling's maneuver was 30% with a specificity of 93%. The maneuver

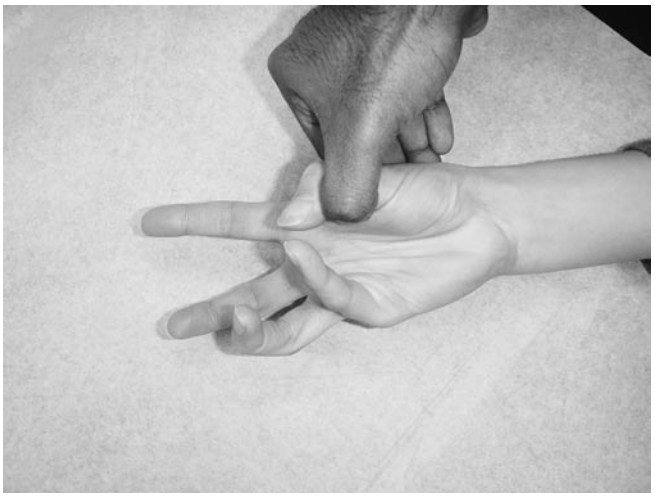

Figure 38.17 Manual muscle testing for opponens pollicis: recurrent branch of median nerve, C8 myotome. The patient is asked to oppose the thumb and bring the thumb toward the little finger against the examiner's resistance.

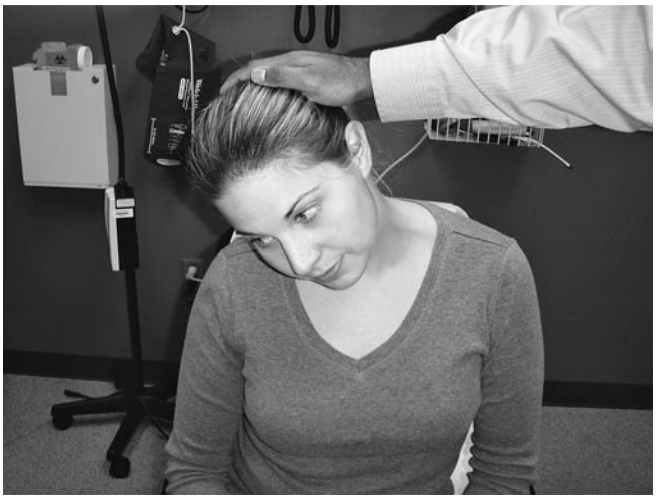

Figure 38.19 Spurling's test. The neck is ipsilaterally flexed while the neck is forward flexed and a sustained axial load is provided. Reproduction of symptoms beyond the shoulder is considered positive and indicative of cervical radicular pain due to a herniated disc or foramenal stenosis.

Figure 38.18 Modified Spurling's test. The neck is extended and the head is ipsilaterally rotated while the spine is axially loaded. Reproduction of symptoms beyond the shoulder is considered positive and indicative of cervical radicular pain due to a herniated disc or foramenal stenosis.

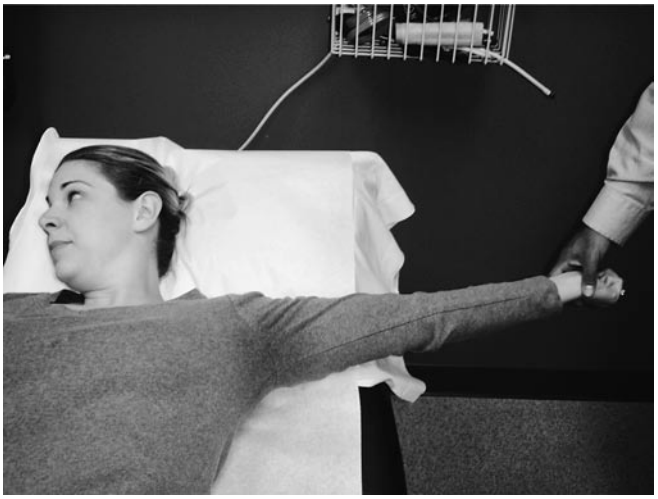

Figure 38.20 Elvey's upper limb root tension test. Abduction of the shoulder, extension of the elbow, and contralateral rotation of the head reproduce limb symptoms.

was considered positive if symptoms referred distal to the shoulder.

As previously discussed, intrinsic shoulder pathology can mimic radicular pain. A targeted shoulder examination is generally indicated. Shoulder impingement syndrome can be assessed with Hawkin's maneuver (Figure 38.21) or with scaption (resisted shoulder abduction in the scapular plane with the elbow extended reproducing familiar shoulder pain). O'Brien's active compression test can be helpful to identify a superior labral anterior and posterior tear (Figure 38.22). Adhesive capsulitis or frozen shoulder presents with a relatively abrupt onset, and on examination the patient has pain at end range of glenohumeral motion. The decrease in range of motion is symmetric in all planes and involves the glenohumeral joint with sparing of scapulothoracic movement. The drop arm test can be seen in full thickness rotator cuff tears of the supraspinatus tendon, and the external lag sign suggests a posterior cuff tear. The empty can test elicits pain or weakness in patients with a rotator cuff tear in the affected shoulder.

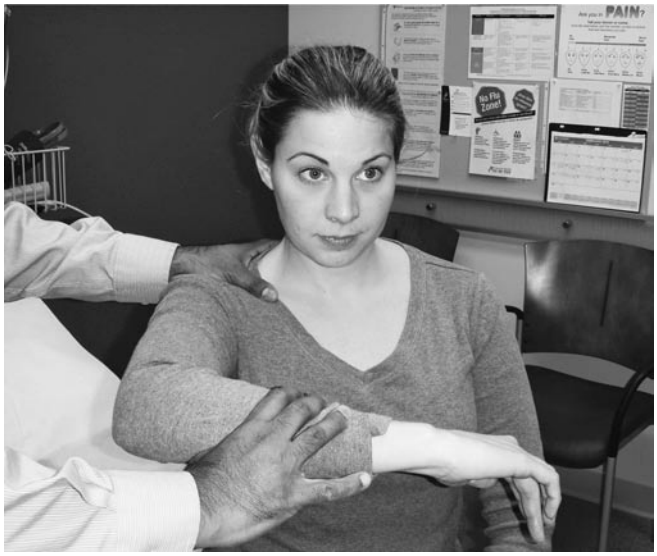

Figure 38.21 Hawkin's sign. The examiner forward flexes the patient's shoulder and internally rotates the shoulder. Reproduction of familiar shoulder symptoms is considered positive and suggestive of impingement of the rotator cuff.

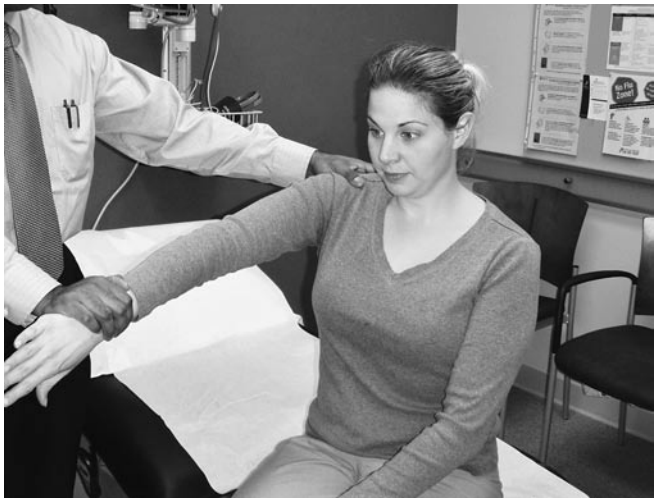

Figure 38.22 Obrien's test. The shoulder is forward flexed with the elbow extended and the forearm is pronated such that the thumb is pointed downward. This patient is asked to resist the examiner's downward force. Reproduction of pain is considered positive and may be suggestive of a SLAP (superior labral tear anterior and posterior) lesion.

CLINICAL ASSESSMENT: DIAGNOSTIC IMAGING

Imaging studies for the evaluation of cervical pain (Chapters 30, 33, and 39) include plain radiographs, MRI, and CT myelogram. Plain radiographs are useful in acute trauma and to assess anteroposterior instability. The trauma series includes anteroposterior, right and left oblique, odontoid view, and cross-table lateral view. All five views in combination have a 92% sensitivity for fracture, but depend upon technique. Flexion and extension films can be used to diagnose gross instability. Greater than 3 mm of translation between flexion and extension or angulation of more than 11° at a single spinal segment may necessitate spinal fusion. Cervical MRI is often the study of choice in patients with radicular and axial neck pain. MRI is indicated when patients have associated constitutional signs, or in patients with prolonged, severe, or progressive neurologic weakness, as further interventions may be necessary. MRI is effective at evaluating degenerative disc changes, central or foramenal stenosis, spondylolisthesis, malignancy, or infection. The addition of gadolinium contrast is useful in the diagnosis of postsurgical epidural fibrosis, infection, or tumor [17–20]. CT myelography can be a complementary tool in the evaluation of cervical radicular pain; however, it is invasive and postdural puncture headaches are among its risks. Using current qualitative methods, MRI and CT myelographic evaluation of cervical spinal stenosis results in significant variation in image interpretation [21]. One study found that CT generally grades disc/bone abnormalities as more severe than those appreciated with MRI [22]. These methods should be viewed as complementary studies, as neither has proven superiority in the diagnosis of all spinal pathology.

Given the high sensitivity of MRI for a wide range of pathology, it has become the study of choice. However, cervical spine anatomic abnormalities such as disk protrusion or extrusion are present in up to 30% of asymptomatic individuals [23,24]. These anatomic abnormalities may lack clinical, not radiologic, specificity for pain complaints. Implicit in that observation is that astute clinical correlation with the history, physical examination, and neurophysiologic and functional tests are necessary to establish a precise diagnosis and ultimately offer specific treatment.

CLINICAL ASSESSMENT: ELECTROMYOGRAPHY

Needle electromyography is the single most useful electrophysiologic test (see Chapter 40) for the identification of cervical radiculopathy. It is performed by percutaneous insertion of a needle into muscles in the upper limb and cervical paraspinal muscles. Muscles with an injury (axonotmesis and/or significant neurapraxia) to its innervating nerve root or peripheral nerve will demonstrate membrane instability, manifested as fibrillation potentials, and positive sharp waves. Needle electrode examination can detect as little as a 4% loss of motor axons [25]. When injury has been present for 4 to 6 weeks, the process of reinnervation will manifest itself as increased duration, polyphasic motor unit potentials. As collateralization progresses, these motor unit potentials mature increasing in amplitude. The pattern of electrophysiologic abnormalities revealed by electromyography can elucidate whether injury involves the cervical spinal nerve root, brachial plexus, or an upper limb peripheral nerve injury. Although needle electromyography is very useful in identifying radiculopathy, it will not identify radicular pain when

motor fiber injury is absent, as cervical radicular pain can exist in the absence of motor fiber injury. Also, in electromyography, six to eight upper limb muscles must be sampled, in addition to cervical paraspinal muscles, to fully assess for radiculopathy. Prior cervical spine surgery can result in lasting denervation and electromyographic findings in the cervical paraspinals, rendering needle study of these muscles less reliable [26]. Another limitation is that the specific nerve root which should be injected or decompressed surgically may not be clear because of myotomal overlap [27,28].

CLINICAL ASSESSMENT: FUNCTIONAL TESTING

Diagnosis may be ambiguous in patients with discordant findings on history, physical examination, diagnostic imaging, and EDx evaluation. Functional testing can play a key role in the diagnosis and management of patients with neck and upper limb pain. Based on the composite of history, examination, and neurophysiologic and imaging studies, a statistical likelihood of the etiology of the patient's pain can be formulated for each individual patient. In a stepwise fashion, proceeding from most likely to least likely, the suspect structures can be anesthetized under fluoroscopic guidance to determine whether symptom relief is obtained. Each patient completes a visual analogue scale (VAS) pre- and postprocedure. The VAS consists of a horizontal bar measuring 100 mm which is marked by the patient to grade the severity of pain. Toward the 100 mm mark represents more severe pain. If the VAS reduction is greater than 80% after a diagnostic block, the block is considered positive and the anesthetized structure is considered to be the etiologic structure. Subsequently, therapeutic injections are offered as treatment if there is a potential benefit from corticosteroid injection. If the diagnostic block does not provide 80% relief, then the next most likely structure is investigated.

Diagnostic selective nerve root blocks have been studied in the cervical and lumbar spine and have demonstrated a high sensitivity and specificity for identifying the pain-generating spinal nerve root. This has been demonstrated in studies correlating positive blocks with identification of an anatomic abnormality at the time of surgery. At least three studies have supported their specificity [29–31]. More extensive studies have been completed on the lumbar spine establishing the sensitivity of diagnostic lumbar selective nerve root block ranging from 87% to 100% and the specificity ranging from 94% to 100% [32–36]. Using methodical technique with low volume of injectate, it is likely that these techniques can also be useful in the cervical spine for the evaluation of cervical radicular pain. It has been demonstrated that higher volumes of injectate will diminish the specificity of the diagnostic selective nerve root block [37].

Technique for Cervical Selective Nerve Root Block

Cervical selective nerve root blocks should be performed by adequately trained physicians with extensive knowledge of the use and role of interventional spine procedures. Numerous case reports have arisen in the literature with the use of particulate steroid resulting in death, cerebrovascular accidents, and spinal cord infarction [38,39]. This chapter predominantly discusses the role of diagnostic selective nerve root blocks where no steroid is used. However, when steroid is used as part of the therapeutic treatment plan, it is advisable to use a nonparticulate steroid such as dexamethasone, use live fluoroscopic imaging with careful attention to the vascular penetrance pattern, use of a 90-second test dose of 2% xylocaine, and the use of digital subtraction angiography if available (Chapter 41).

The patient is positioned in the supine or semioblique position (Figure 38.23). This will be dependent on whether the rotational angle of the fluoroscope is able to reach the optimal angle of entry. If the optimal oblique angle cannot be reached for a given foramen, the patient will need a more oblique positioning on the procedure table. The interventionalist should understand the varying orientation of

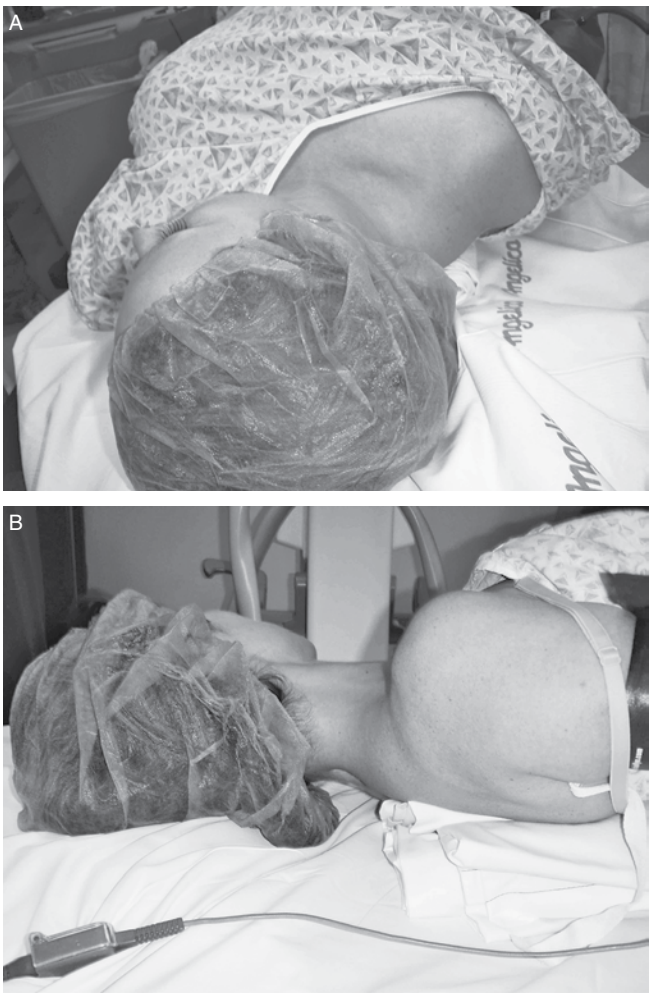

Figure 38.23 Patient is positioned in a supine and semioblique position with a wedge. The need for the semioblique positioning will vary based on fluoroscopy equipment and its rotational angle capability.

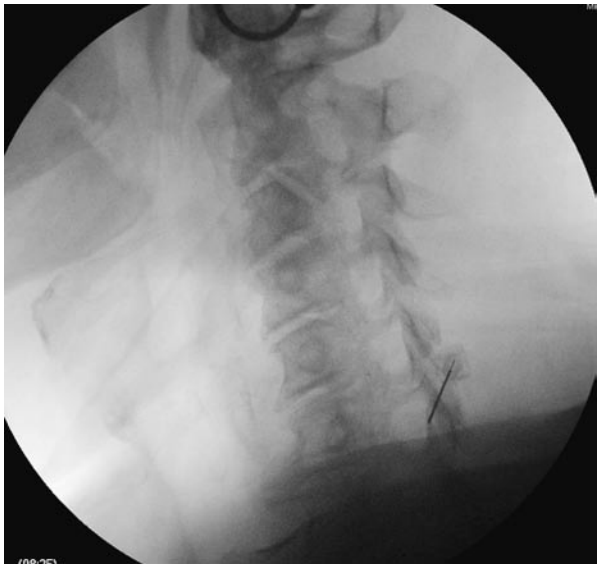

Figure 38.24 The oblique view demonstrates the proper angle of entry for the C6-7 foramen, that is, C7 spinal nerve root's foramen. The figure demonstrates the needle in the posterior aspect of the foramen in order to minimize the risk of trauma to the vertebral artery.

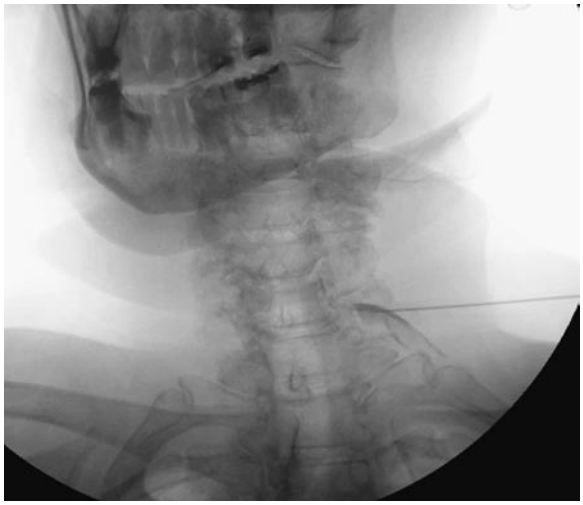

Figure 38.25 Posterior-anterior and oblique fluoroscopic images of the cervical spine during performance of a cervical selective nerve root block. The posterior-anterior view demonstrates the flow of contrast along the course of the spinal nerve without significant central or adjacent level flow.

the cervical foramen. The more cephalad foramen have a more anterior-posterior longitudinal course, and the more caudad foramen adopt a more lateral longitudinal course. The needle is targeted toward the posterior aspect of the foramen at its midpoint in the vertical plane (Figure 38.24). The needle should be advanced slowly using fluoroscopic guidance. Often the bony superior articular process of the zygapophyseal joint can be contacted lightly to minimize the risk of being too ventral. The needle can then be redirected anterior after touching the bone. The depth

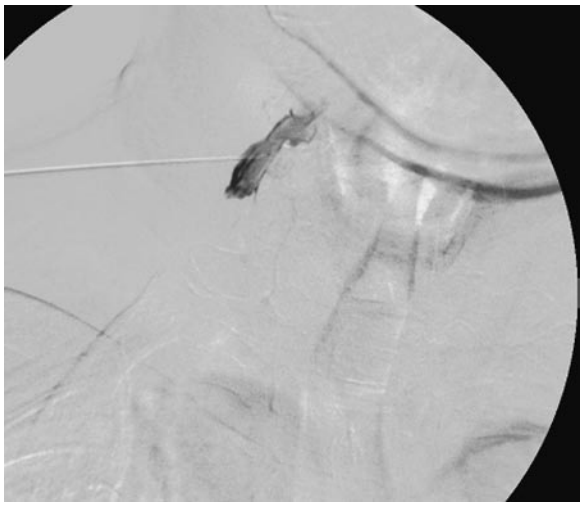

Figure 38.26 The use of digital subtraction angiography confirms the absence of vascular uptake of contrast injectate.

of the needle should be confirmed with posterior-anterior imaging views, and the needle should not at any point stray medial to the articular pillar to avoid dural puncture, and ideally the needle tip should be in the lateral one-half of the articular pillar on the posterior-anterior view (Figure 38.25). This will minimize the risk of vertebral artery penetration and dural puncture. Contrast flow for a selective nerve root block ideally should outline the targeted spinal nerve without adjacent spinal nerve spread or significant epidural flow. If vascular flow is noted, the needle should be repositioned. Use of digital subtraction angiography helps further ensure lack of vascular flow (Figure 38.26). Once satisfactory position is obtained, the anesthetic is injected. For diagnostic selective nerve root blocks, no steroid is used, and a small dose of a potent anesthetic, 0.8 cc of 2% preservative-free xylocaine, is instilled. Larger volumes will cause adjacent level spread, leading to false positives.

Combination with steroid will dilute and increase the volume of injectate, leading to potential false positives and negatives. If steroid is used, a test dose should be performed. This involves injection of 0.8 to 2 cc of 1% preservative-free xylocaine, and 90 seconds of patient observation. The patient should be queried about weakness, numbness, or tingling in the face or mouth, or other neurologic sequelae. After the 90-second time period, the steroid can be administered. A nonparticulate steroid such as dexamethasone should be used.

The patient should complete pre- and postprocedural pain rating scales and pain body diagrams. A reduction of 80% in VAS is considered positive. Careful interpretation of the pain diagrams and VAS is required because patients may misunderstand which pain they are rating, may have multiple nociceptors, may not provoke their symptoms adequately postprocedure, may not have sufficient immediate preprocedural pain, and may have placebo or other nonspecific responses. Adequate informed consent regarding the purpose of the procedure, risk, and expectations is necessary.

CONCLUSION

Treatment of a patient's spine pain and disability should employ a diagnosis-specific approach. Cervical spine complaints should be categorized into axial, radicular, and myelopathic features. Identification of myelopathic features should lead the clinician to consider surgical options to prevent spinal cord compromise. In the absence of myelopathy, most spine disorders can be managed conservatively. Diagnostic workup should be performed taking into account historical provocative factors, neurologic and musculoskeletal examination, electromyography, and functional testing using diagnostic anesthetization of suspect structures (Figures 38.6–38.8 and Table 38.1). Diagnostic anesthetization can be a useful tool to identify the nociceptor in cases where other testing is inconclusive. Treatment options for cervical radicular pain include patient education, activity and postural and worksite modifications, therapeutic exercise, adjunctive modalities, cervical traction, pharmacologic measures, fluoroscopically guided injection procedures, and surgical decompression. Treatment should be individualized and goal oriented to ensure successful patient outcomes.

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39 Diagnostic Imaging of Cervical Radiculopathy

John B. Weigele

INTRODUCTION

Cervical radiculopathy refers to dysfunction of a cervical spinal nerve root and/or spinal nerve typically characterized by pain in one arm and the neck that is associated with varying combinations of the loss of motor function, sensory dysfunction, and abnormal reflexes [1,2]. Diagnostic imaging often plays an important role in the evaluation of cervical radiculopathy; however, careful consideration of when to image and the integration of the imaging findings with the clinical history, physical examination, and other diagnostic testing are essential. This chapter will focus on when to obtain imaging studies, choosing the optimal imaging modality, the essential elements of imaging performance and interpretation, and the predictive value of diagnostic imaging for cervical radiculopathy.

WHEN TO IMAGE

Currently, there are no clearly defined guidelines for the timing of diagnostic imaging for cervical radiculopathy [1,2]. Most cases are caused by spondylosis associated with degenerative disc changes and/or cervical disc herniations [2,3]; some rarer causes are listed in Table 39.1 [4–26]. "Red flags" in the clinical history suggesting an uncommon cause for cervical radiculopathy requiring an urgent diagnosis such as tumor, infection, vertebral artery dissection, or traumatic nerve root injury justify immediate imaging; these "red flags" include unexplained weight loss, a previous malignancy, unremitting pain at night, fever, chills, immune suppression, intravenous drug use, and trauma (Figure 39.1) [2]. Coexisting myelopathy or progressive neurologic deficits are also indications for early imaging.

For the majority of patients, it is reasonable to limit diagnostic imaging to those who remain symptomatic after 4 to 6 weeks of conservative medical management [2]. Many patients will improve within this time frame; imaging is unlikely to influence their clinical care. In addition, anatomic abnormalities of the cervical spine are frequently found in asymptomatic individuals on magnetic resonance imaging (MRI), highlighting the importance of interpreting imaging findings within the context of the clinical presentation [27–29]. Patients who remain symptomatic after 4 to 6 weeks are more likely to require interventional spine procedures or surgery and therefore will require imaging correlation to guide therapy.

CHOICE OF IMAGING MODALITY

Plain Films

Plain films (conventional radiographs) of the cervical spine are often obtained as an initial screening examination for cervical radiculopathy, but typically have limited value [30]. Disc herniations are not detectable, degenerative

Table 39.1 Some Rarer Causes of Cervical Radiculopathy

Neoplasm
Metastases [4]
Lymphoma [5,6]
Multiple myeloma [4]
Primary bone tumors [4,7]
Spinal cord tumors [4]
Nerve sheath tumors [8]
Meningioma [4]
Ganglioneuroma [9]
Pancoast tumor (lung apex) [10]
Other tumors [4]
Infection
Discitis, osteomyelitis, and epidural abscess (bacterial, fungal, tuberculosis) [4,11]
Lyme disease [12]
Viral infection [4]
Inflammatory/autoimmune
Multiple sclerosis [13]
Vascular
Vertebral artery dissection [14]
Vertebral artery loop [15]
Arteriovenous fistula [16,17]
Giant cell arteritis [18]
Cavernous malformation [19]
Trauma
Nerve root avulsion [20]
Fracture [10]
Jumped facet [4,10]
Other
Syringomyelia [4,10]
Synovial chondromatosis [21]
Spontaneous cervical epidural hematoma [22]
Synovial cyst [23]
Varicose epidural veins [24]
Calcifying pseudoneoplasm [25]
Sarcoidosis [26]

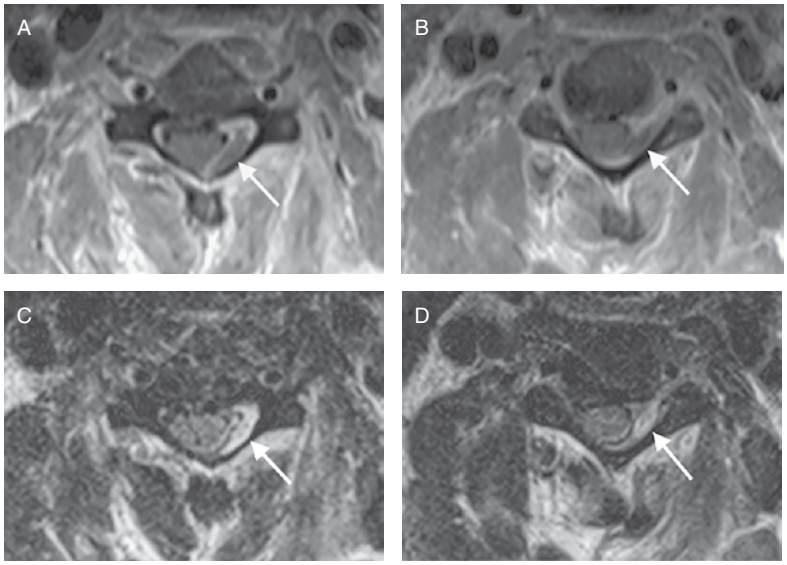

Figure 39.1 Epidural abscess in an intravenous drug abuser presenting with cervical radiculopathy imaged with magnetic resonance imaging. **(A, B)** Postcontrast axial T1-weighted spin echo images demonstrate an isointense, left-sided, dorsal epidural fluid collection (abscess) with surrounding enhancement (phlegmon) displacing the thecal sac (A, arrow), and tracking into the left C4-5 neural foramen (B, arrow). **(C, D)** On axial T2-weighted fast spin echo images the collection is only mildly hyperintense consistent with a high protein content (arrows).

narrowing of the neural foramen is difficult to quantify and correlate with clinical symptoms, and radiographs have a very low sensitivity for unusual causes of cervical radiculopathy such as tumor and infection. In some cases, plain films can clarify whether an abnormality seen on MRI represents an osteophyte or herniated disc; nonetheless, an unenhanced computed tomography (CT) provides this information with much more precise anatomic detail [31]. In one study, the positive predictive value of conventional radiographs for myelographic root sleeve deformities was only 55.5% [32]. Greater than 50% of the vertebral bone volume must be destroyed before it is detectable [30]. Plain films are most useful in limited, selective circumstances such as suspected spinal instability (flexion/extension films) and postoperative assessments.

Magnetic Resonance Imaging

Over the previous two decades, MRI has become firmly established as the primary imaging examination for most cases of cervical radiculopathy [30,33]. Myelography combined with postmyelographic computed tomography (CT myelography) has been relegated largely to a supplementary role. MRI has a greater ability to demonstrate the spinal and paraspinal anatomy in exquisite detail, including the vertebrae, discs, spinal cord, nerve roots, and associated soft tissue structures (ligaments, muscles, arteries, veins, etc.) (Figure 39.2). MRI and CT myelography have comparable sensitivities for surgically confirmed causes for cervical radiculopathy, as discussed later (see predictive value of diagnostic imaging). In addition, MRI has a greater sensitivity than other imaging modalities for the less common causes of cervical radiculopathy, such as infection and tumor (Table 39.1; Figures 39.1 and 39.3) [4,30]. MRI avoids the complications associated with the spinal tap necessary to instill intrathecal contrast for CT myelography [34], and also does not subject the patient to ionizing radiation.

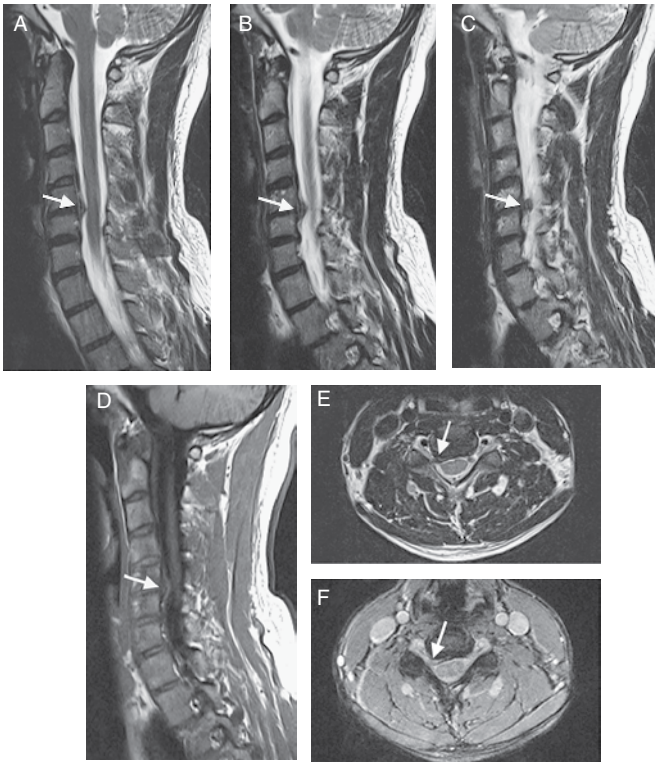

Figure 39.2 Foraminal disc herniation causing a right C6 radiculopathy imaged with magnetic resonance imaging. **(A–C)** Sagittal T2-weighted fast spin echo (FSE) images, **(D)** sagittal T1-weighted spin echo image, **(E)** axial T2-weighted fast spin echo image, and **(F)** an axial T2*-weighted gradient echo image demonstrates a right C5-6 foraminal disc herniation (arrows) impinging on the right C6 nerve root.

Nonetheless, MRI does have diagnostic and practical limitations. A small percentage of symptomatic disc herniations and osteophytes may go undetected on MRI; in the appropriate clinical setting this is an important indication

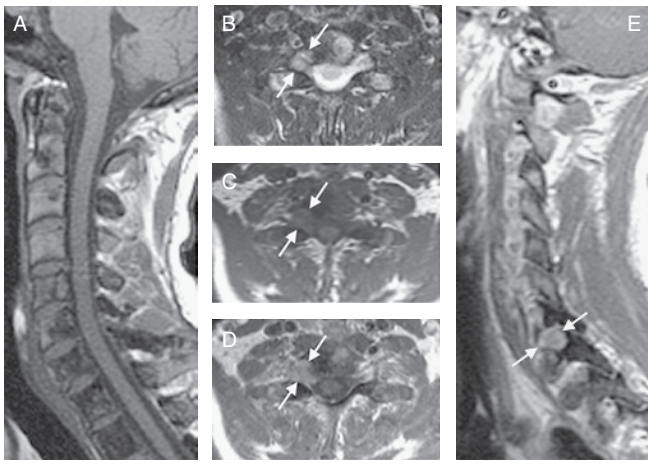

Figure 39.3 Breast metastasis causing a right C8 radiculopathy imaged with magnetic resonance imaging. **(A)** Sagittal T1-weighted spin echo image demonstrates extensive, heterogeneously low T1-signal intensity within the cervical vertebrae consistent with diffuse osseous metastases. **(B)** Axial T2-weighted fast spin echo image shows a mildly T2-hyperintense mass in the right C7-T1 neural foramen (arrows). (C, D) Axial precontrast **(C)** and postcontrast **(D)** T1-weighted spin echo images demonstrate the mass is T1-isointense (C) and has mild, diffuse enhancement (D, arrows). **(E)** Sagittal postcontrast T1-weighted spin echo image outlines the enhancing mass in the right C7-T1 foramen (arrows).

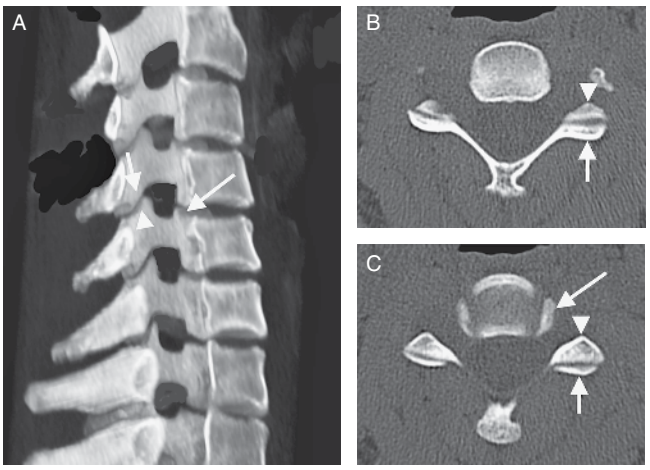

Figure 39.4 Normal bony anatomy of the cervical neural foramina imaged with helical computed tomography. **(A)** Volume-rendered 3D view from inside the cervical spinal canal looking out the left neural foramina demonstrates normal osseous anatomy and normal caliber neural foramina. The uncovertebral joint borders the anteromedial aspect of the neural foramen (long arrow). The superior articular process of C5 (arrowhead) and the inferior articular process of C4 (short arrow) border the posterolateral aspect of the foramen and form the facet joint. (B,C) Axial images through the mid **(B)** and lower **(C)** left C4-5 neural foramen demonstrate the uncinate process (C, long arrow), the superior articular process of C5 (B and C, arrowhead) and the inferior articular process of C4 (B and C, short arrow).

for supplementary CT myelography [35,36]. In addition, the longer imaging times required for MRI may not be tolerated by uncooperative patients who are in pain or claustrophobic; this may result in an aborted or nondiagnostic examination because MRI images are markedly degraded by significant motion. Current-generation MRI scanners have addressed these issues with more rapid scanning protocols and wider, shorter bores. Finally, some patients have an absolute contraindication to an MRI examination, such as a medical implant sensitive to a high magnetic field or a ferromagnetic foreign body that may move and cause tissue damage [37]. Patients in renal failure may have a contraindication to gadolinium-containing MRI contrast agents that can cause nephrogenic systemic fibrosis, but can safely undergo unenhanced MRI examinations [38].

Unenhanced CT

CT without intrathecal contrast plays a relatively limited role in the evaluation of cervical radiculopathy. Unenhanced CT does not detect a significant percentage of herniated cervical discs, despite the fact that it is quite sensitive for lumbar disc herniations [33,39]. A study using high-resolution (1 mm) unenhanced helical CT imaging was only 66% sensitive for surgically proven cervical disc herniations [40]. This was explained in part by the observation that most cervical disc herniations occur at the C5-6 and C6-7 disc interspaces [41]; axially acquired CT images at those levels often include the shoulders and upper torso, decreasing the signal-to-noise ratio and causing image degradation [40]. In addition, unenhanced CT frequently does not demonstrate cervical spine tumors and infections [30].

Nonetheless, current high-resolution multidetector helical CT scanners are unparalleled in displaying the bony anatomy of the cervical spine (Figure 39.4). CT is more accurate than MRI for evaluating the cervical spine for facet (zygapophyseal) joint arthropathy prior to cervical disc arthroplasty [42]. In some cases, it is difficult to distinguish between a "hard disc" (osteophytes) and a "soft disc" (noncalcified disc herniation) on MRI; in this setting, CT can supplement the MRI, precisely depicting the osteophytes (Figure 39.5) [31,36,43].

CT Myelography

In most patients, CT myelography has largely been replaced by MRI for the initial evaluation of cervical radiculopathy. Nonetheless, CT myelography was long considered the gold standard for imaging the soft tissue and bone pathologic changes causing nerve root and spinal cord compression syndromes [44]. Although MRI and CT myelography have comparable sensitivities for surgically confirmed causes for cervical radiculopathy [1], CT myelography adds the discomfort and risks of a spinal tap, as well as ionizing radiation [34]. CT myelography is also much less sensitive than MRI for most of the rarer causes of cervical radiculopathy (Table 39.1) [4,30]. Nonetheless, CT myelography is invaluable in patients with an absolute contraindication to MRI (Figure 39.6), as well as in supplementary or confirmatory roles for MRI in selective circumstances, such as the occasional symptomatic disc herniation or osteophyte that is uncertain or undetected on MRI [35,36].

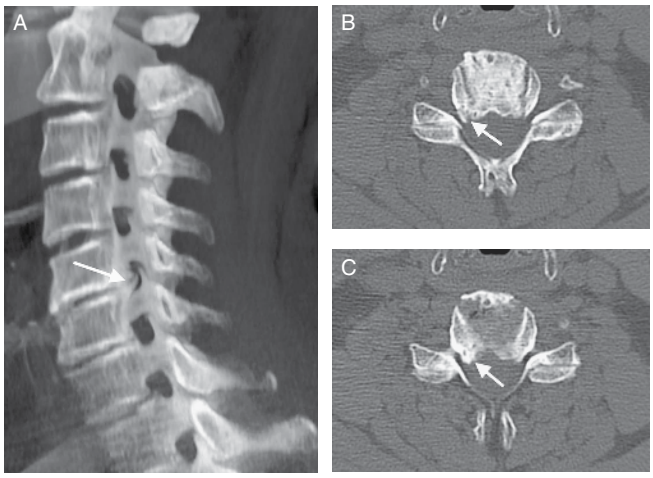

Figure 39.5 Cervical foraminal stenosis caused by uncovertebral joint hypertrophy imaged with helical computed tomography. **(A)** Volume-rendered 3D view from inside the cervical spinal canal looking out the right neural foramina demonstrates severe right C5-6 foraminal stenosis primarily caused by marked uncovertebral joint hypertrophy (arrow) and, to a lesser extent, facet joint hypertrophy. (B,C) Axial images through the mid **(B)** and lower **(C)** right C5-6 neural foramen reveal marked uncovertebral joint hypertrophy and foraminal stenosis (arrows).

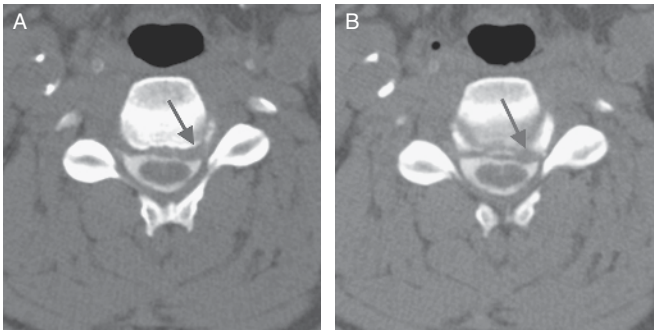

Figure 39.6 Disc herniation causing left C6 radiculopathy imaged with computed tomographic (CT) myelography in a patient with a contraindication to magnetic resonance imaging. **(A,B)** Postmyelographic CT images demonstrate intrathecal contrast outlining a left paramedian C5-6 disc herniation extending into the medial aspect of the left C5-6 neural foramen (arrows) impinging on the left C6 nerve root.

PERFORMANCE AND INTERPRETATION OF DIAGNOSTIC IMAGING

Unenhanced CT

Technique

Current-generation slip-ring, multichannel CT scanners offer helical (spiral) volumetric data acquisitions, providing rapid scanning speed and small, isotropic voxels that can be reconstructed into equally high-resolution images in any plane. In clinical practice, thin-section (e.g., 1 mm) bone algorithm images are routinely reconstructed and reviewed in axial, sagittal, and coronal planes. Axial soft tissue algorithm images are typically reviewed as well, although the CT sensitivity for soft tissue pathology is significantly inferior to MRI. In addition, the data can be transferred to an independent workstation where volume-rendered three-dimensional (3D) images and multiplanar reconstructions can be reconstructed and manipulated in any desired plane (Figures 39.4, 39. 5, and 39.7).

Findings

The osseous anatomy of the cervical neural foramen is exquisitely demonstrated on helical CT (Figure 39.4). The uncovertebral joint (joint of Lushka) arises from the posterolateral aspects of the vertebral bodies and borders the anteromedial aspect of the neural foramen; the superior articular process of the caudal vertebra and the inferior articular process of the rostral vertebra form the facet (zygapophyseal) joint and border the posterolateral aspect of the foramen. Degenerative arthropathy and spondylosis (osteophyte formation) can lead to neural foraminal narrowing because of uncovertebral joint hypertrophy (Figure 39.5), facet hypertrophy, or a combination of both (Figure 39.7). CT is relatively insensitive for the detection of a noncalcified cervical disc herniation ("soft disc") without intrathecal contrast [40]. CT is the best modality to demonstrate a fracture or jumped facet associated with cervical radiculopathy.

Magnetic Resonance Imaging

Technique

Cervical spine MRI optimally is performed on high-field strength (1.5 and 3.0 Tesla) scanners with dedicated surface coils to obtain the best signal-to-noise ratio and spatial resolution. A cooperative patient is essential to minimize motion artifacts; nursing support may be necessary to provide intravenous analgesics for pain and anxiolytic medications for claustrophobia. Occasionally, general anesthesia may be necessary. A typical cervical spine MRI protocol includes sagittal T1-weighted spin echo (SE), sagittal T2-weighted fast spin echo (FSE), axial T2-weighted FSE, and axial T2*-weighted high-resolution 3D gradient echo (GE) images.

An unenhanced MRI examination is adequate to evaluate typical cervical radiculopathy [35]. However, if the clinical presentation or the preliminary unenhanced MRI suggests tumor, infection, or another uncommon cause for cervical radiculopathy (Table 39.1), pre- and postgadolinium contrast-enhanced T1-weighted SE sagittal and axial images often provide important information (Figures 39.1 and 39.3). On occasion, it may be useful to obtain one of the postgadolinium series with fat suppression, because both normal fat and pathologic enhancement appear hyperintense on T1-weighted images.

Findings

Cervical and lumbar spinal nerve roots take different paths out of the spinal canal; in the cervical spine, the

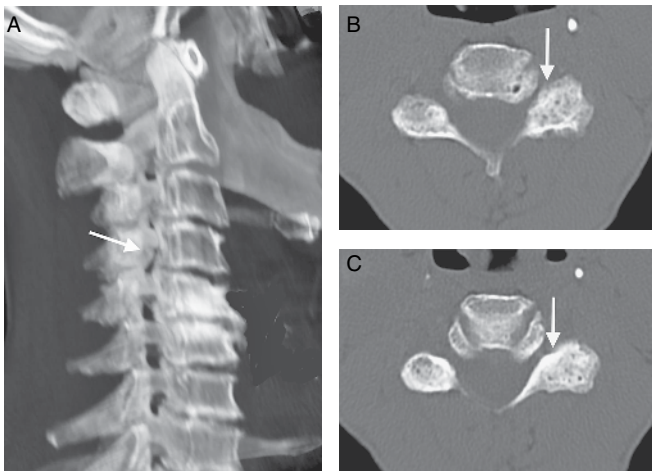

Figure 39.7 Cervical foraminal stenosis caused by facet and uncovertebral joint hypertrophy imaged with helical computed tomography. **(A)** Volume-rendered 3D view from inside the cervical spinal canal looking out the left neural foramina demonstrates a severe left C4-5 foraminal stenosis caused by marked facet joint hypertrophy (arrow) and, to a lesser extent, uncovertebral joint hypertrophy. **(B,C)** Axial images through the mid (B) and lower (C) left C4-5 neural foramen reveal marked foraminal stenosis and facet joint hypertrophy (arrows).

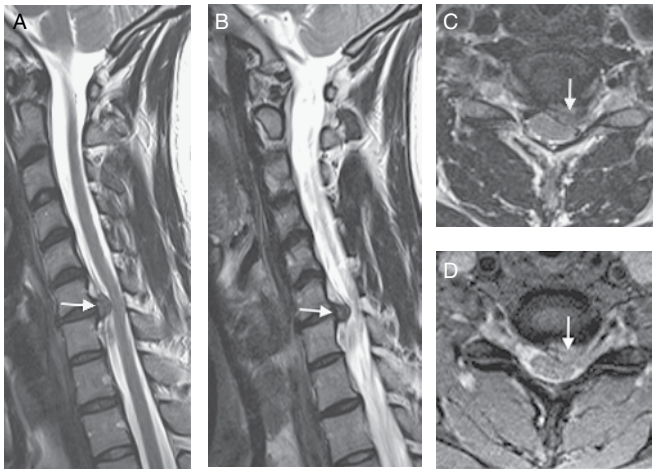

Figure 39.8 Paramedian disc herniation causing a left C7 radiculopathy imaged with magnetic resonance imaging. **(A,B)** Sagittal T2-weighted fast spin images, **(C)** axial T2-weighted fast spin echo image, and **(D)** axial T2*-weighted gradient echo image demonstrate a large left paramedian disc herniation at C6-7 impinging on the left ventral spinal cord surface and impinging on the left C7 nerve root in the exit zone and in the medial aspect of the left C6-7 neural foramen (arrows).

nerve roots for a given level exit above the pedicle of the correspondingly numbered vertebra; therefore, the C7 nerve roots exit the C6-7 neural foramina (the C8 roots exit at C7-T1). In addition, the cervical nerve roots exit the thecal sac at the same level as the disc into the lower portion of the cervical neural foramen. Therefore, both paramedian (Figure 39.8) and lateral (foraminal) (Figure 39.9) cervical disc herniations typically compress a nerve root at the same level (e.g., a C6–7 disc herniation usually compresses the C7 nerve root in the C6-7 exit zone or the C6-7 neural foramen).

On axial T2-weighted FSE and thin-section 3D T2*-weighted GE images, cerebrospinal fluid (CSF) is high signal intensity (hyperintense) and osteophytes are low signal intensity (hypointense; Figure 39.10) [30]. Disc herniations are usually intermediate in signal intensity (hyperintense relative to bone and hypointense to CSF; Figures 39.8 and 39.9); however, the signal intensity of disc herniations is quite variable with differing states of hydration and can be similar to osteophytes [35,43]. The signal in the herniation may also differ from the parent disc. Dense bone contains few mobile hydrogen protons; therefore, it is characteristically hypointense on both T1- and T2-weighted imaging. Degenerative foraminal narrowing demonstrates hypointense osteophytes encroaching on the foramen and effacing more hyperintense fat, vessels, and CSF (Figures 39.10 and 39.11).

Standard 3D thin-section GE techniques provide superior delineation of disc morphology, central canal, and foraminal stenoses (Figures 39.10 and 39.11); however, this technique can be limited by motion and by susceptibility artifacts that overestimated the severity of the foraminal stenosis [45]. Improved hardware (e.g., stronger and faster gradients, self-shielding coils) and the addition

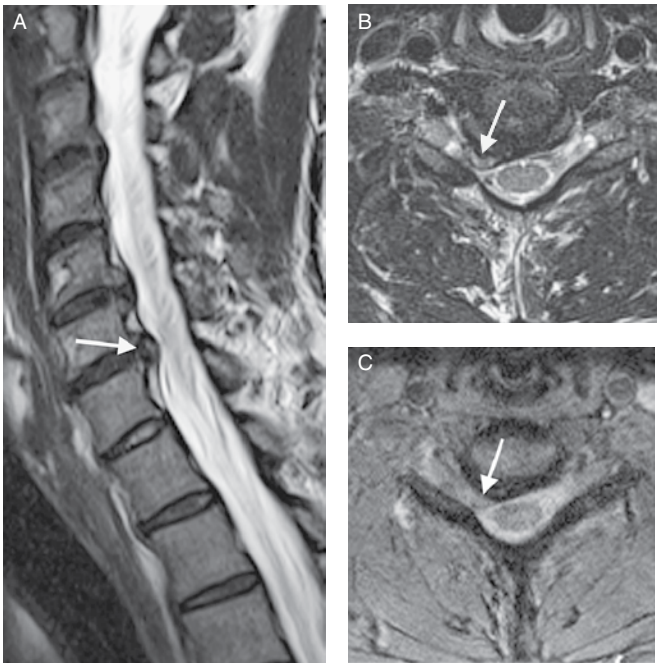

Figure 39.9 Foraminal disc herniation causing a right C7 radiculopathy imaged with magnetic resonance imaging. **(A)** Sagittal T2-weighted fast spin echo image, **(B)** axial T2-weighted fast spin echo image, and **(C)** axial T2*-weighted gradient echo image demonstrate a right C6-7 foraminal disc herniation (arrows) impinging on the right C7 nerve root.

of magnetization transfer at short echo times result in less exaggeration of neural foraminal stenoses [46–48].

Sagittal T2-weighted FSE images are essential to evaluate the cervical discs, as well as potential thecal sac or

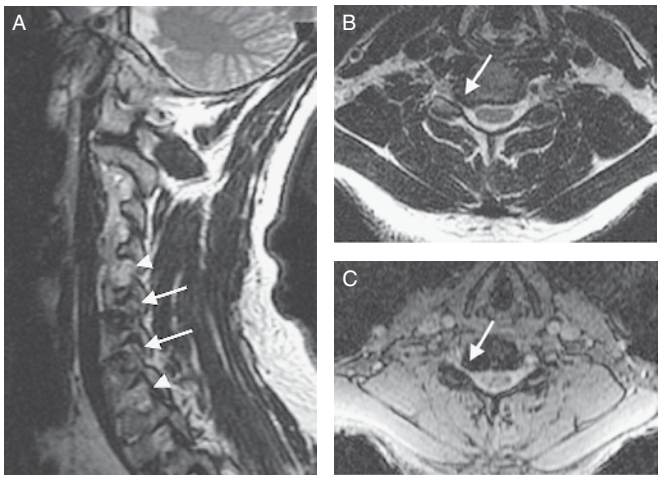

Figure 39.10 Uncovertebral joint hypertrophy causing right C5-6 and C6-7 neural foraminal narrowing presenting with radiculopathy imaged with magnetic resonance imaging. **(A)** Sagittal T2-weighted fast spin echo image demonstrates marked narrowing of the right C5-6 and C6-7 neural foramina (arrows). The right C4-5 and C7-T1 foramina appear normal (arrowheads). (B,C) Axial T2-weighted fast spin echo image **(B)** and axial T2*-weighted gradient echo image **(C)** through C6-7 demonstrate severe uncovertebral joint hypertrophy and foraminal narrowing (arrows).

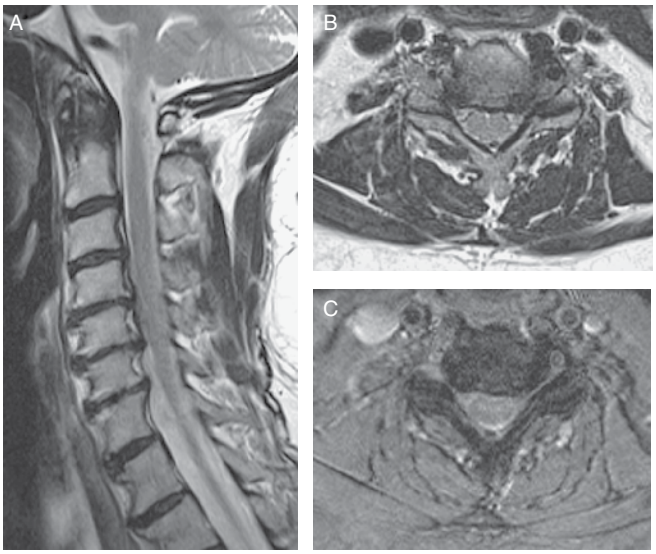

Figure 39.11 Degenerative disc disease, posterior spondylosis, and bilateral neural foraminal stenoses imaged with magnetic resonance imaging. **(A)** Sagittal T2-weighted fast spin echo image demonstrates degenerated discs at C4-5, C5-6, and C6-7 associated with loss of height, decreased signal intensity, and posterior spurs abutting the ventral spinal cord surface. (B,C) An axial T2-weighted fast spin echo image **(B)** and an axial T2*-weighted gradient echo image **(C)** demonstrate severe bilateral C5-6 foraminal narrowing due to facet and uncovertebral joint hypertrophy.

spinal cord compression, and also intrinsic spinal cord pathology. The T2-weighted FSE technique is relatively insensitive to susceptibility artifacts compared with conventional SE and GE techniques; in addition, there is exquisite differentiation between the spinal cord and nerve roots and the CSF because of the differences in signal intensities and edge enhancement (Figures 39.2, 39.8, 39.9, and 39.11) [30]. Although T2-weighted FSE images display good discrimination of soft tissues, one disadvantage to this pulse sequence is that normal fat is relatively hyperintense and may obscure edema, especially in fatty marrow. Occasionally, fat-suppressed T2-weighted FSE or short-tau inversion recovery images may be helpful with cases of suspected inflammation or trauma. Degenerated discs are decreased in height, have lower T2-signal intensity, and are associated with osteophyte formation (Figure 39.11). Degenerative changes in the subchondral bone marrow are often visible reflecting fibrosis and/or edema with inflammation (type I), yellow marrow (type II), or sclerosis (type III) [49]. Sagittal T2-weighted FSE images demonstrate paramedian (Figure 39.8) and foraminal (Figures 39.2 and 39.9) disc herniations, as well as the encroachment of low signal intensity osteophytes into the foramina that causes degenerative narrowing (Figure 39.10) [30].

Sagittal T1-weighted SE images were not found helpful for the evaluation of cervical radiculopathy in one prospective study of 30 patients [50]; nonetheless, unexpected pathology such as replacement of the normal fatty marrow in the cervical vertebrae by tumor may be best appreciated on these images (Figure 39.3).

Postgadolinium T1-weighted SE imaging does not add to the evaluation of neural foraminal compromise by disc herniations or osteophytes [35]; nonetheless, contrast-enhanced MRI often adds to the detection and characterization of more uncommon causes of radiculopathy (Table 39.1), including infection and tumor. Discitis and osteomyelitis typically demonstrate destruction of the bony endplates of the disc interspace; associated with decreased T1-signal intensity, increased T2-signal intensity and marked enhancement in the disc and adjacent vertebral bone marrow reflecting edema and inflammation (Figure 39.12). An epidural phlegmon can be seen as an intensely enhancing mass (Figure 39.12). Most intramedullary, intradural-extramedullary and extradural tumors are T1-isointense, variably T2-hyperintense (an exception: meningiomas often are T2-isointense or T2-hypointense) and enhance (Figure 39.13); involvement of the neural foramina can be demonstrated clearly on postgadolinium SE images (Figures 39.3 and 39.13).

CT Myelography

Technique

Cervical CT myelography begins with the intrathecal administration of nonionic contrast. Both C1-2 and lumbar spinal punctures have been described; although the C1-2 approach usually yields superior quality myelogram films, both approaches provide postmyelographic CT images with equivalent diagnostic quality. Therefore, most practitioners prefer the lumbar approach to avoid the higher morbidity associated with the C1-2 approach [34]. Typically, 10 mL of 300 mg iodine/mL nonionic contrast is instilled intrathecally through a 22-gauge spinal needle

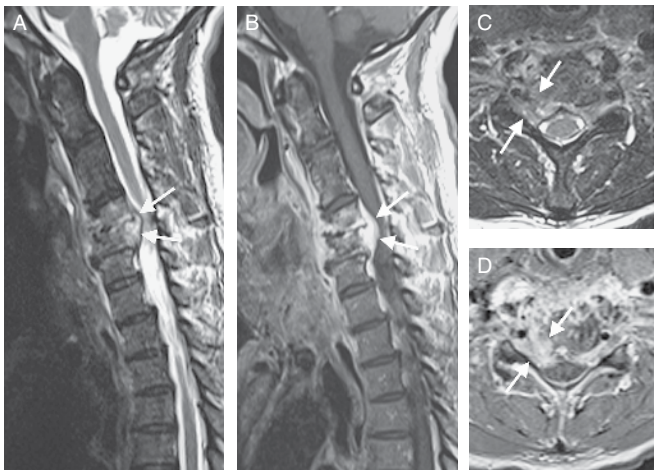

Figure 39.12 Discitis and osteomyelitis in a hemophiliac presenting with neck pain and a right C6 radiculopathy imaged with magnetic resonance imaging. **(A,B)** Sagittal T2-weighted fast spin echo image (A) and contrast-enhanced T1-weighted spin echo image (B) demonstrate destruction of the C5-6 disc, edema (A) and enhancement (B) in the C5 and C6 vertebrae; and an enhancing ventral epidural phlegmon (A and B, arrows). **(C,D)** Axial T2-weighted fast spin echo image (C) and postcontrast T1-weighted spin echo image (D) demonstrate a T2-hyperintense (C) and enhancing (D) phlegmon destroying the posterolateral aspect of the C6 vertebra and filling the right C5-6 neural foramen (arrows).

with the patient in the prone position and the spinal needle is removed. Next, the head is hyperextended to prevent contrast from refluxing into the intracranial subarachnoid space. The patient is then tilted head down to run the contrast into the cervical subarachnoid space and the table is returned to a neutral position. Cervical myelogram films are obtained in anteroposterior, both oblique and lateral projections.

Following the myelogram, the patient is transferred for a postmyelographic CT; this is obtained similarly to the previously described unenhanced CT technique, using slip-ring multichannel CT scanners and helical (spiral) volumetric data acquisitions that provide small, isotropic voxels that can be reconstructed into equally high-resolution images in any plane [51]. Thin-section (e.g., 1 mm) images optimized for intrathecal contrast are routinely reconstructed and reviewed in axial, sagittal, and coronal planes.

Findings

Typically, the mass effect on the intrathecal contrast on the myelogram films is characterized as intramedullary (e.g., intrinsic spinal cord tumor such as an astrocytoma or ependymoma), intradural-extramedullary (e.g., nerve sheath tumor or meningioma), or extradural (e.g., osteophyte or herniated disc). Extradural mass effect on the contrast-filled cervical nerve root sleeves is visualized best on anteroposterior and oblique projections. An indentation in the contour of the opacified nerve root sleeve or complete lack of filling (amputation) reflects significant compression of the medial aspect of the neural foramen, but may not define the exact etiology or anatomy (Figure 39.14). Subsequent postmyelographic CT images precisely delineate the location and degree of foraminal compromise, and differentiate disc herniations with soft tissue attenuation (approximately 80 Hounsfield units) (Figure 39.6) from degenerative spondylosis (Figure 39.14) [40]. In one study, the use of multidetector helical CT improved the visualization of nerve root abnormalities compared with both conventional postmyelographic CT scans and MRI scans [51].

PREDICTIVE VALUE OF DIAGNOSTIC IMAGING

Epidemiology of Cervical Radiculopathy

An epidemiologic population-based study of cervical radiculopathy in Rochester, Minnesota found annual incidence rates of 107.3 per 100,000 for men and 63.5 per 100,000 for women. The age at presentation ranged from 13 to 91 years, with an age-specific peak incidence rate of 202.9 per 100,000 for the 50- to 54-year-old age group [3]. Another study found a prevalence of 3.5 cases per 1000 individuals, with a peak in the 50- to 59-year-old age group [52].

In the Rochester study, physical exertion or trauma preceded symptom onset in only 14.5% of the cases. The most common presentation was a C7 monoradiculopathy, followed by C6. Spondylosis, a disc herniation, or both were related to the radiculopathy in 68.4%. Most patients were treated conservatively, without surgery. Recurrence, defined as the reappearance of symptoms after a symptom-free interval of at least 6 months, occurred in 31.7%. At last follow-up, 90% were asymptomatic or only mildly incapacitated due to cervical radiculopathy [3].

Imaging Abnormalities in the Asymptomatic Population

Degenerative changes in the cervical spine (including the discovertebral complex, uncovertebral, and facet joints) are an inevitable part of the normal aging process and are often asymptomatic; one early study found degenerative changes in 75% of asymptomatic individuals in the seventh decade of life [53]. In a prospective MRI study of the cervical spine in 63 asymptomatic volunteers, 14% of individuals younger than 40 years had significant abnormalities (disc herniation in 10%, foraminal stenosis in 4%). In those older than 40 years, 28% demonstrated significant abnormalities (disc herniation in 5%, disc bulge in 3%, and foraminal stenosis in 20%) [28]. In another MRI study of asymptomatic volunteers, degenerative changes in the cervical spine increased linearly with age. The most common finding was disc degeneration, found in 17% of men and 12% of women in their twenties; this increased to 86% of men and 89% of women older than 60 years. Disc herniations impinging on the spinal cord were present in 7.6% of the subjects, mostly in those older than 50 years. In addition, 5.9% of the neural foramina were narrowed, also primarily in individuals older than 50 years [29].

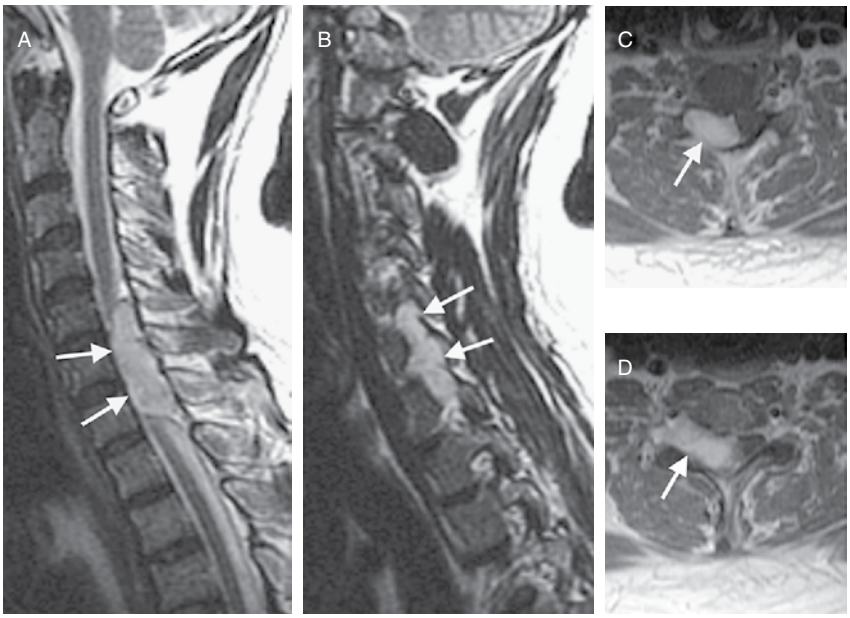

Figure 39.13 Low-grade neuroectodermal tumor presenting with pain and cervical radiculopathy imaged with magnetic resonance imaging. **(A,B)** Sagittal T2-weighted fast spin echo images demonstrate a mildly hyperintense extradural mass (A, arrows) extending into the right C5-6 and C6-7 neural foramina (B, arrows). **(C,D)** Axial postcontrast T1-weighted spin echo images show the enhancing mass within expanded right C5-6 (C) and C6-7 (D) neural foramina (arrows).

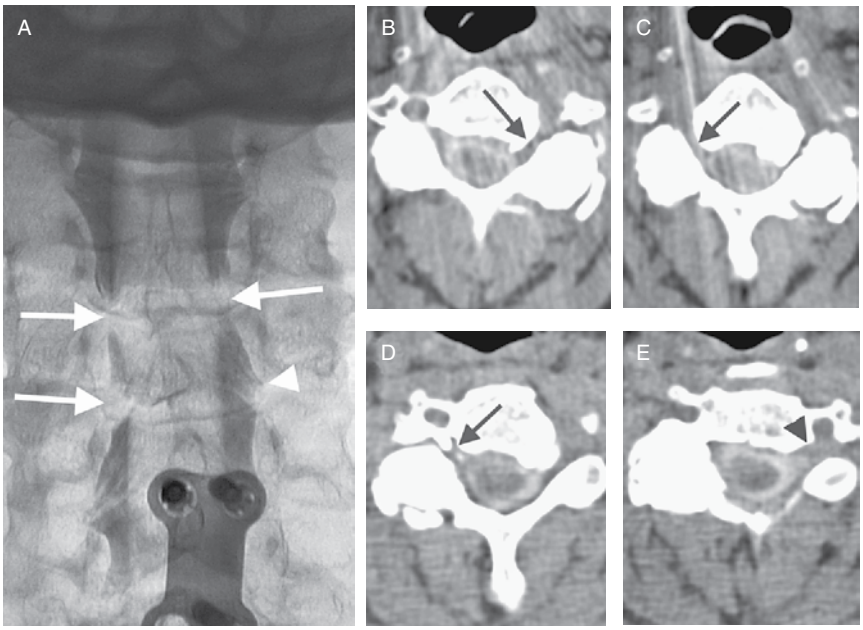

Figure 39.14 Cervical radiculopathy following anterior discectomy and fusion imaged with myelography and postmyelographic computed tomography (CT). **(A)** Anteroposterior cervical myelogram radiograph with intrathecal contrast reveals marked amputation of both C3-4 and the right C4-5 nerve root sleeves (arrows). The left C4-5 nerve root sleeve is only mildly compressed (arrowhead). **(B–E)** Axial postmyelogram CT images reveal intrathecal contrast and marked foraminal narrowing caused by uncovertebral and facet joint hypertrophy on both sides at C3-4 **(B,C)** and on the right at C4-5 (**D**, arrow and **E**). The left C4-5 foramen is only mildly narrowed (E, arrowhead).

Imaging Abnormalities in the Symptomatic Population

The most common imaging finding in cervical radiculopathy (70%–75% of cases) is bony narrowing of the neural foramen encroaching on the spinal nerve root by varied combinations of degenerative spondylosis of the uncovertebral joint anteriorly and the facet (zygapophyseal) joint posteriorly, often associated with a degenerative decrease in the disc height. A herniated disc is present in 20% to 25% of the cases [2].

A prospective study of patients with cervical radiculopathy published in 1986 compared myelography, postmyelographic CT, and MRI with the surgical findings. Surgical evaluation confirmed 67% of the myelographic findings, 85% of the postmyelographic CT findings, and 74% of the MRI findings [31]. However, MRI was just as sensitive as postmyelographic CT in identifying the abnormal level although less accurate in differentiating between a disc herniation and an osteophyte. This older study did not utilize modern FSE or 3D T2*-weighted GE techniques that may have improved the ability to make this distinction. In another study, Brown et al. found MRI for cervical radiculopathy and myelopathy detected 88% of surgically detected lesions, whereas myelography detected 58% and postmyelographic CT detected 81% [33].

Additional studies found similar accuracies for MRI and CT myelography in the evaluation of cervical radiculopathy. Larsson et al. found an accuracy of 73% for CT myelography and 77% for MRI [54]. Wilson et al. found

84% accuracy and 92% sensitivity for MRI in detecting surgical lesions in 40 patients [55]. CT myelography yielded comparable results but was only performed in 13 patients. Neuhold et al. found that MRI for cervical radiculopathy and myelopathy detected 100% of 30 surgically detected lesions; CT myelography detected 28 of 30 lesions [56]. Both Wilson et al. and Neuhold et al. concluded that MRI was the only preoperative examination necessary in most cases of cervical radiculopathy.

It is worth noting that MRI hardware and software has markedly improved since the time these comparative studies were performed in the late 1980s and early 1990s [57]. It highly likely that the diagnostic accuracy of MRI for cervical radiculopathy using current technologies has surpassed CT myelography; however, MRI has been so firmly established as the primary imaging modality it is unlikely that comparison studies will be repeated.

In one prospective study, CT myelography detected surgically confirmed traumatic cervical nerve root avulsions with 85% accuracy, compared with only 52% accuracy for MRI [20]; however, in a more recent study, the sensitivity for detecting cervical nerve root avulsions was the same for both modalities (92.9%) when overlapping thin-section T2-weighted MRI images were obtained [58].

Clinical Significance of Imaging Findings

Cervical radiculopathy is primarily a clinical diagnosis; diagnostic imaging and electrophysiological testing can only provide supporting evidence [1,3]. Unfortunately, there are no well-defined clinical diagnostic criteria for cervical radiculopathy; the existing data suggest that the neurologic examination is not very precise [1,3]. Surgery is also an imperfect "gold standard" for cervical radiculopathy; in one prospective, multicenter study, 26% of patients reported their worst pain remained "horrible or excruciating" following surgery, raising the question whether the cause was truly identified in those patients [59].

Another important caveat of the studies comparing imaging and surgical findings is that these represent a small subset of all patients with cervical radiculopathy and therefore contain significant selection bias; thus, these results cannot be extrapolated to all patients with the disorder. Hence, no "standard reference criterion" for cervical radiculopathy has been established to which diagnostic imaging can be compared [60].

The epidemiologic data and the studies of the imaging abnormalities in asymptomatic individuals emphasize the low specificity (high false-positive rate) of diagnostic imaging for cervical radiculopathy in the general population. Nonetheless, the current literature suggests that diagnostic imaging in selective symptomatic patients can be carefully integrated with clinical information to improve the management of cervical radiculopathy.

CONCLUSIONS

Diagnostic imaging often plays an important role in the evaluation of cervical radiculopathy; nonetheless, it must be carefully evaluated within the context of the clinical history, physical examination, and other diagnostic testing. Patients with "red flags" for an unusual cause for cervical radiculopathy should undergo immediate imaging; those with a typical clinical presentation can undergo conservative management with subsequent imaging if they do not experience significant improvement. Diagnostic imaging can clarify clinical uncertainty and guide interventional spine procedures or surgery. MRI is the preferred diagnostic imaging modality for most patients; however, plain films, CT, and CT myelography continue to play important roles in selective circumstances. Imaging studies must be interpreted within the context that asymptomatic individuals frequently have anatomic abnormalities in the cervical spine, including those that may be associated with cervical radiculopathy. Finally, MRI and CT myelography detect the large majority (but not all) of the anatomic lesions surgically proven to be causing cervical radiculopathy.

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40 Electrodiagnostic Evaluation of Cervical Radiculopathy

Timothy R. Dillingham and Diane W. Braza

INTRODUCTION

Cervical radiculopathies are conditions involving a pathologic process affecting the spinal nerve root. Commonly, this is a herniated nucleus pulposis that anatomically compresses a nerve root within the spinal canal. Another common etiology for radiculopathy is spinal stenosis resulting from a combination of degenerative spondylosis, ligament hypertrophy, and spondylolisthesis. Inflammatory radiculitis is another pathophysiologic process that can cause radiculopathy. It is important to remember, however, that other more ominous processes such as infiltration by malignancy, granulomatous tissue, and infection (epidural abscess) can manifest the same symptoms and signs of radiculopathy as the more common causes.

This chapter discusses the clinical approach used in an electrodiagnostic laboratory to evaluate a person with neck pain or upper limb symptoms which are suggestive of radiculopathy. The indications for referring for testing as well as the limitations of testing are discussed to give a greater understanding of this important diagnostic evaluation.

Given the large differential diagnosis for upper limb, neck, and shoulder symptoms, it is important for electrodiagnosticians to develop a conceptual framework for evaluating these referrals with a standard focused history and physical examination and a tailored electrodiagnostic approach. Accurately identifying radiculopathy by electrodiagnosis whenever possible provides valuable information that guides treatment and can better direct utilization of diagnostic and therapeutic strategies.

SPINE AND NERVE ROOT ANATOMY: DEVIATIONS FROM THE EXPECTED

From an electrodiagnostic perspective, several specific anatomical issues merit further discussion. At all levels, the dorsal root ganglion lies within the intervertebral foramen [1]. This anatomical arrangement has implications for clinical electrodiagnosis of radiculopathy, namely that sensory nerve action potentials are preserved in most radiculopathies as the nerve root is affected proximal to the dorsal root ganglion.

There are many anatomic variations regarding the cervical nerve roots and the brachial plexus. Perneczky [2] described an anatomic study of 40 cadavers. In all cases, there were deviations from accepted cervical root and brachial plexus anatomy. Levin et al. [3] examined the pattern of abnormalities on electromyography (EMG) in 50 cases of surgically proven cervical root lesions. A range of needle EMG patterns was found with EMG demonstrating less specificity for the C6 root level, but more specificity and consistent patterns for C8, C7, and C5 radiculopathies. In those subjects with C6 radiculopathy, half of the patients showed findings similar to those with C5 radiculopathy and the other half demonstrated C7 patterns. This surgical group was more severely affected than patients who did not require surgical interventions, and this pattern may not hold for less symptomatic patients. These findings underscore the limitations of precise localization for root lesions by EMG and the need for complementary spinal imaging. When the imaging and EMG findings are concordant, it is more compelling evidence for a radiculopathy. The electrodiagnostician should maintain an appreciation of these anatomic variations to better convey the level of certainty with respect to diagnostic conclusions.

PHYSICAL EXAMINATION

The electrodiagnostic examination is an extension of the standard clinical examination. The history and physical examination are vital initial steps in determining what conditions may be causing the patient's symptoms. Most radiculopathies present with signs and symptoms confined to one limb. Multisegmental radiculopathies such as those seen in cervical spinal stenosis may present as numbness, pain, and weakness in both upper limbs. A focused neuromuscular examination that assesses muscle strength, reflexes, and sensation in both the affected limb and the contralateral limb is important, providing better detection of subtle weakness if present and also providing a conceptual framework for electrodiagnostic assessment.

For upper limb and neck symptoms, the differential diagnosis is large and includes brachial plexopathy (brachial neuritis), radiculopathy, shoulder impingement syndrome, lateral epicondylitis, deQuervains tenosynovitis, and median and ulnar neuropathies. An astute clinician can sort these conditions out to a large extent before the

study, but as you see later, musculoskeletal disorders can coexist with radiculopathy.

Cannon and colleagues [4] examined the ability of physical examination and identification of musculoskeletal disorders (myofascial pain, shoulder impingement, lateral epicondylitis, deQuervain's tenosynovitis) to predict the outcomes of electrodiagnostic testing in persons with neck and upper limb symptoms referred to an electrodiagnostic lab. They found that the total prevalence of musculoskeletal disorders as identified by physical examination was 42%. The prevalence in those with a normal EMG study was 69%, compared with 29% in those with cervical radiculopathy ($P < 0.0001$) and 45% in those persons who demonstrate another diagnosis ($P = 0.02$) by electrodiagnostic testing. Those patients who ended up showing an electrodiagnostically confirmed cervical radiculopathy had lower prevalences of musculoskeletal disorders than did other patients, yet still 29% showed one of these musculoskeletal conditions. This means that patients frequently had both radiculopathy and one or more musculoskeletal conditions. Although the prevalence of certain musculoskeletal disorders made having a normal electrodiagnostic evaluation significantly more likely, the high prevalence among both patients with normal studies and those with cervical radiculopathy and other disorders limited the usefulness of this information in precisely predicting study outcome. Therefore, the presence of musculoskeletal disorders should not preclude electrodiagnostic testing when otherwise indicated.

Lauder and colleagues [5] examined the effectiveness of the patient's history and physical examination findings for predicting electrodiagnostic outcomes in suspected cervical radiculopathy. If a reflex was lost, there was a 4.32 significant odds ratio for increased likelihood of finding a cervical radiculopathy by EMG. For any weakness, the increased likelihood was 4.2 times (significant odds ratio). A combination of reflex changes and weakness resulted in a ninefold (odds ratio = 9.15) greater likelihood of finding electrodiagnostically confirmed cervical radiculopathy [5]. Haig et al. [6] found that electrodiagnosis substantially alters clinical impressions in 42% of patients, and confirmed the clinical diagnoses in 37% of patients studied.

ELECTRODIAGNOSTIC EVALUATION

The American Association of Neuromuscular and Electrodiagnostic Medicine (AANEM, formerly American Association of Electrodiagnostic Medicine) guidelines recommend that for an optimal evaluation of a patient with suspected radiculopathy, a needle EMG screen of a sufficient number of muscles and at least one motor and one sensory nerve conduction study should be performed in the involved limb [7,8]. The nerve conduction studies are necessary to exclude polyneuropathy. The sufficiency of the EMG screen and a recommended number of muscles is discussed in detail later. An EMG study is considered diagnostic for a radiculopathy if EMG abnormalities are found in two or more muscles innervated by the same nerve root, and different peripheral nerves, yet muscles innervated by adjacent nerve roots are normal [7]. This assumes of course that other generalized conditions such as polyneuropathy are not present.

EMG study of bilateral limbs is often necessary, particularly if a single limb shows EMG findings suggestive of radiculopathy and the patient has symptoms in both the studied and the contralateral limb. If bilateral limbs are involved, then the electrodiagnostician should proceed by studying selected muscles in an upper limb (if the lower limbs are abnormal on EMG) or a lower limb (if both upper limbs are abnormal), to exclude a generalized process such as polyneuropathy or motor neuron disease. Likewise, additional nerve conduction studies are appropriate to exclude other suspected conditions (median or ulnar neuropathies) and the electrodiagnostician should have a low threshold for expanding the study.

Motor and Sensory Nerve Conduction Studies

Standard motor and sensory nerve conduction studies are not helpful in identifying a cervical radiculopathy; however, they should be performed to screen for polyneuropathy and exclude common entrapment neuropathies if the patient's symptoms could be explained by a focal entrapment. It is important to remember that based upon the anatomy of the dorsal root ganglion, sensory responses should be normal in most radiculopathies. If they are found to be absent, this should raise suspicion for another diagnosis such as polyneuropathy or plexopathy. Motor nerve conduction studies are frequently normal in cervical radiculopathies, unless significant axonal loss has occurred resulting in reduction of the compound muscle action potential amplitude. Such compound muscle action potential reductions are infrequently seen, however, because of dual nerve root innervation of most muscles. Additionally, incomplete nerve root compression occurs in most cases of cervical radiculopathy [7].

Plexopathies often pose a diagnostic challenge as they are similar to radiculopathies in symptoms and signs. To distinguish brachial plexopathy from cervical radiculopathy, sensory responses which are accessible in a limb should be tested. In plexopathy, they are likely to be reduced in amplitude, whereas in radiculopathy they are generally normal. Sensory responses may be normal in the case of a cervical root avulsion due to trauma. The distal motor latencies and conduction velocities are usually preserved as they reflect the fastest conducting nerve fibers, but you may have some slowing if substantial axonal loss has occurred in the case of a severe plexopathy [7].

Late Responses

In patients with upper limb symptoms suggestive of cervical radiculopathy, H-reflexes and F-waves are not useful in diagnosis but rather help exclude polyneuropathy as an underlying cause of symptoms. Although H-reflexes can be elicited, they are more challenging than in the lower limb. One study by Miller and colleagues [9] examined the H-reflexes in the upper limb and found they were somewhat helpful and complementary to needle EMG. Although these findings suggested a possible role for these upper

limb H-reflexes, they are highly specialized, time consuming, and difficult to consistently elicit. Further studies are necessary to clarify whether these findings can be duplicated in other centers [9].

F-waves are late responses involving the motor axons and axonal pool at the spinal cord level. They can be assessed and classified by using the minimal latency, mean latency, and chronodispersion or scatter [9]. As in the case of H-reflexes, F-waves demonstrate low sensitivities and are not specific for radiculopathy, rather they are a better test to screen for polyneuropathy. Published sensitivities in radiculopathies range from 13% to 69%; however, these studies suffer from many of the shortcomings as are found in the H-reflex studies [10–13].

Needle EMG

The need for EMG, particularly in relationship to imaging of the spine, has been highlighted [14]. Needle EMG is particularly helpful in view of the fact that the false-positive rates for spine magnetic resonance imaging (MRI) are quite high. The false-positive rate for cervical MRI is approximately 19% of subjects demonstrating an abnormality, but only 10% showing a herniated or bulging disc [14,15]. Radiculopathies can occur without structural findings on MRI, and likewise without EMG findings. The EMG only evaluates motor axonal loss or motor axon conduction block. For these reasons, a radiculopathy affecting the sensory root will not yield abnormalities by EMG. If the rate of denervation is balanced by reinnervation in the muscle, then spontaneous activity is less likely to occur and be identified by needle EMG. However, if clinical weakness is notable on physical examination, EMG may detect an increased motor unit recruitment frequency.

The sensitivity of EMG for detecting cervical radiculopathy has been examined in a number of studies and reduced motor unit recruitment. The results of some of these studies are tabulated in Table 40.1 [16–21]. Table 40.1 lists the "gold standards" for diagnosis against which these EMG findings were compared. Studies using a clinical standard may reflect a less severe group, whereas those using a surgical confirmation may indicate a more severely involved group. The sensitivity for EMG is unimpressive, ranging from 49% to 92% in these studies. EMG is not a sensitive test, yet likely has higher specificity. The issue of specificity and its value in electrodiagnosis was underscored by Robinson [15]. It is apparent that EMG is not a very good screening test. In terms of screening tests, MRI is better for identifying subtle structural abnormalities, with EMG to assess their clinical relevance and to exclude other disorders.

Identification of Radiculopathy

The concept of a screening EMG encompasses identifying the possibility of an electrodiagnostically confirmable radiculopathy. If one of the muscles in the screen is abnormal, the screen must be expanded to exclude other diagnoses, and to fully delineate the radiculopathy level. Because of the screening nature of the EMG examination, electrodiagnosticians with experience should look for more subtle signs of denervation, and if present in the screening muscles then expand the study to determine if these findings are limited to a single myotome or peripheral nerve distribution. If they are limited to a single muscle, then the clinical significance of the findings is less certain.

Table 40.1 Selected Studies Evaluating the Sensitivity of EMG Relative to Various "Gold Standards" for Diagnosis of Cervical Radiculopathy

Study	Sample Size	Gold Standard	EMG Sensitivity (%)
Berger [16]	18	Clinical	61
Partanen [17]	77	Intraoperative	67
Leblhuber [11]	24	Clinical + myelogram	67
So [18]	14	Clinical	71
Yiannikas [19]	20	Clinical and/or radiographic	50
Tackman [20]	20	Clinical	95
Hong [21]	108	Clinical	51

Unless otherwise stated the EMG parameters used in sensitivity calculations were fibrillation potentials.

EMG, electromyography.

Modified from Dillingham TR. Electrodiagnostic approach to patients with suspected radiculopathy. *Phys Med Rehabil Clin N Am.* 2002;13:567–588, with permission.

Quantifying Nerve Root Dysfunction

The standard EMG protocol in a radiculopathy screen includes assessment of distal, proximal, and paraspinal muscles. Proximal and distal muscles innervated by the same myotome should be sampled to exclude a distal-proximal pattern of abnormalities such as a polyneuropathy. Standard assessment includes observation of insertional activity, presence of abnormal spontaneous activity (positive sharp waves, fibrillation potentials, complex repetitive discharges [CRDs]), and quantification of such abnormalities. EMG abnormalities are conventionally graded on a scale from 0 to 4, with 0 being none present and 4 being full and sustained interference pattern of potentials in all four quadrants. It is important to know that fibrillations can be seen with direct muscle trauma such as a traumatic injury, arthroscope sites, or where surgical fixators are placed with damage to underlying muscle tissue. Such areas should be avoided whenever possible.

Observation of motor unit morphology, stability, amplitude, duration, recruitment pattern, and firing characteristics and the myotomal distribution of such abnormal findings is critical in the assessment of radiculopathy [22].

Chronology of Nerve Root Injury

The specific electrodiagnostic abnormalities seen with a radiculopathy have conventionally been used to assess chronicity of the radiculopathy. Myotomal fibrillations and positive waves in the absence of change in motor unit configuration suggested a recent onset of injury [23]. It was a commonly held notion that in acute radiculopathies, the paraspinal muscles denervated first followed by proximal and then distal limb musculature, and that reinnervation starts with paraspinal muscles and then limb

muscles. This paradigm was recently addressed with a series of investigations [24,25]. Symptom duration had no significant relationship to the probability of finding spontaneous activity in paraspinal or any limb muscles. One could postulate that radicular pain can exist without or before onset of radiculopathy marked by measurable disrupted neurophysiology. The pathophysiology of radiculopathy is not well explained by this simplistic symptom duration model.

Based upon the investigations cited earlier, there is no evidence of a relationship between the duration of a patient's symptoms and the probability of finding fibrillations in paraspinal or limb muscles. This simplistic explanation, although widely quoted in the older literature, does not explain the complex pathophysiology of radiculopathies. Electrodiagnosticians should not invoke this relationship to explain the absence or presence of fibrillations in a particular muscle [24,25].

Paraspinal Muscle Examination

With respect to paraspinal muscle assessment, the only relevant findings are fibrillations, positive sharp waves, CRDs, myokymia, or myotonia. There are no normative values to which polyphasicity or motor unit morphology of cervical paraspinal muscles can be compared. Motor unit morphology is therefore uninterpretable and without well-derived norms to which a patient's findings can be compared. Electrodiagnosticians should not identify radiculopathies solely on the basis of paraspinal polyphasicity, reduced recruitment, or increased insertional activity. Paraspinal muscles should be considered either normal or positive if they have fibrillations, positive waves, or other specific discharges (CRDs, myokymia, or myotonia). Care must be taken to have the patient relax these muscles to ensure optimal evaluation of insertional and spontaneous activity. A particular issue that the electrodiagnosticians should be aware of is that the trapezius muscle is a large triangular-shaped muscle that is superficial to the cervical paraspinal muscles. Persons with a spinal accessory cranial neuropathy due to trauma may complicate radiculopathy assessment as there might be membrane irritability in some parts of the cervical paraspinal muscle examination. It is important to examine the deepest cervical paraspinal muscles for membrane irritability and the clinician should use caution when placing significance on the paraspinal examination in this clinical circumstance.

Cervical paraspinal muscles should be examined in most patient evaluations with suspected radiculopathy [26]. Recruitment pattern findings and motor unit potential morphology for these muscles have not been established. Paraspinal muscles either show spontaneous activity and other discharges as described earlier and therefore localize the lesion to the root level—or they do not. There is likely overlap in cervical paraspinal muscles with single roots innervating fibers above and below their anatomic levels. For this reason, the level of radiculopathy cannot be delineated by paraspinal muscle EMG abnormalities alone, but rather is based upon the root level that best explains the distribution of limb muscles demonstrating EMG changes.

The Cervical Radiculopathy Screen

Dillingham et al. [27] conducted a prospective multicenter study evaluating patients referred to participating electrodiagnostic laboratories with suspected cervical radiculopathy. The sample consisted of persons with electrodiagnostically confirmed cervical radiculopathies. The analyses were directed at determining the extent of testing, by means of modeling various radiculopathy screens, that one must perform to be sure of not missing an electrodiagnostically confirmable radiculopathy. This is a different paradigm than sensitivity and involves determining when an EMG of the upper limb and neck can be confidently stopped. A standard set of muscles were examined by needle EMG for all patients. Those with electrodiagnostically confirmed cervical radiculopathies, based upon EMG findings, were selected for analysis. The EMG findings in this prospective study encompassed the following neuropathic findings: (1) positive sharp waves, (2) fibrillation potentials, (3) CRDs, (4) high-amplitude, long-duration motor unit action potentials, (5) increased proportion of polyphasic motor unit action potentials, or (6) reduced recruitment of motor unit potentials. A cervical radiculopathy subject in the sample was considered successfully identified when one or more muscles in our modeled screens were positive for that subject. There were 101 patients with electrodiagnostically confirmed cervical radiculopathies representing cervical root levels C5 to C8. When paraspinal muscles were one of the screening muscles, five muscle screens identified 90% to 98% of radiculopathies, six muscle screens identified 94% to 99% (Table 40.2), and seven

Table 40.2 Six Muscle Screen Identifications of the Patients with Cervical Radiculopathies

Muscle Screen	Neuropathic (%)	Spontaneous Activity (%)
Without Paraspinals		
Deltoid, APB,FCU, triceps, PT, FCR	93	66
Biceps, triceps, FCU, EDC, FCR, FDI	87	55
Deltoid, triceps, EDC, FDI, FCR, PT	89	64
Biceps, triceps, EDC, PT, APB, FCU	94	64
With Paraspinals		
Deltoid, triceps, PT, APB, EDC, PSM	99	83
Biceps, triceps, EDC, FDI, FCU, PSM	96	75
Deltoid, EDC, FDI, PSM, FCU, triceps	94	77
Biceps, FCR, APB, PT, PSM, triceps	98	79

The "neuropathic"column indicates the identification rates when looking for all types of subtle neuropathic findings. The spontaneous activity column indicates identification rates when only fibrillations or positive sharp waves are considered.

APB, abductor pollicus brevis; EDC, extensor digitorum communis; FCR, flexor carpi radialis; FCU, flexor carpi ulnaris; FDI, first dorsal interosseus; PSM, paraspinal muscles; PT, pronotor teres.

Identification criteria, and definitions are described in text. Adapted from ref. [27] with permission.

muscle screens identified 96% to 100%. When paraspinal muscles were not part of the screen, eight distal limb muscles recognized 92% to 95% of radiculopathies.

Six muscle screening including paraspinal muscles yielded consistently high identification rates and studying additional muscles led to marginal increases in identification. In some instances, a particular muscle cannot be studied because of wounds, skin grafts, dressings, or infections. In such cases, the electromyographer can use an alternative muscle with similar innervations in the screen with equally high identification. These findings were consistent with those derived from a large retrospective study [28].

Limitations of the EMG Screen

If one of the six muscles studied in the screen is positive, then there is the possibility of confirming electrodiagnostically that a radiculopathy is present. In this case, the examiner must study additional muscles to determine the radiculopathy level and to exclude a mononeuropathy. If the findings are found in only a single muscle, they remain inconclusive and of uncertain clinical relevance. If none of the six muscles are abnormal, the examiner can be confident of not missing the opportunity to confirm that a radiculopathy is present, and the painful needle examination can be curtailed. The patient may still have a radiculopathy of mild degree (affecting only a few axons or sensory nerve roots), but other tests such as MRI will be necessary to confirm this clinical suspicion. It is important for examiners to realize that a purely sensory nerve root involvement will yield a normal EMG screen.

These cervical muscle screens are not intended to substitute for a clinical evaluation and differential diagnosis formulation by the electrodiagnostic consultant. Rather, information from investigations described earlier allows the electrodiagnostician to streamline the EMG evaluation and make more informed clinical decisions regarding the probability of missing an electrodiagnostically confirmable radiculopathy when a given set of muscles are studied. Performing a focused history and physical examination is essential, and these screens should not supplant such clinical assessments or a more detailed electrodiagnostic study when circumstances dictate considerations of diagnoses other than radiculopathy.

It is important to remember that the EMG screens for cervical radiculopathies were validated in a group of patients with limb symptoms suggestive of radiculopathies. These screens will not provide sufficient screening power if a brachial plexopathy is present or if a focal mononeuropathy such as a suprascapular neuropathy is the cause of the patient's symptoms. The electrodiagnostician should always perform EMG on weak muscles to increase the diagnostic yield. These screens do not sufficiently screen for myopathies or motor neuron disease. It is incumbent upon the electrodiagnostician to formulate a differential diagnosis and methodically evaluate for other diagnostic possibilities when clinically indicated. Structuring the examination as data are acquired is an important aspect of electrodiagnostic medicine and one that distinguishes such consultations from other diagnostic tests.

Implications of an EMG-Confirmed Radiculopathy

It is important that the electrodiagnostician not forget that EMG does not indicate the exact cause of the radiculopathy, only that nerve root injury has occurred. A spinal tumor, herniated disc, bony spinal stenosis, chemical radiculitis, or severe spondylolisthesis can all yield the same EMG findings. This underscores the need to image the spine with MRI (including gadolinium) to assess for significant structural causes of electrodiagnostically confirmed nerve dysfunction. A negative EMG test should not prevent obtaining an MRI if clinical suspicion for radiculopathy is high. Given the low sensitivities of needle EMG, it is not an optimal screening test, but rather a confirmatory test.

Saal et al. reported on the overall favorable natural history for cervical radiculopathy secondary to disc herniations treated nonoperatively. In this cohort of patients with herniated cervical discs, 92% were managed successfully using pain management strategies that included medications, therapy, and occasionally epidural steroid injections for pain control [29]. No correlation with electrodiagnostic testing was made however [29]. Trials comparing electrodiagnostic findings and outcomes after cervical spinal surgery using validated outcome measures are scarce. Alrawi et al. studied the value of neurophysiologic and imaging studies in predicting the surgical outcome for patients with cervical radiculopathy. Patients with preoperative evidence of cervical nerve root dysfunction on EMG had better outcomes following discectomy and anterior fusion that those without EMG evidence of nerve root abnormality [30]. Bednarik and colleagues [31] examined 66 persons with mild cervical stenosis by MRI yet who were asymptomatic. This group found that 20% of scanned individuals eventually developed clinical signs and symptoms of myelopathy. The investigators discovered in those persons who had clinical radiculopathy symptoms and EMG showing motor axonal loss in two myotomes that EMG predicted with *90% accuracy* the 20% of the sample who progressed from mild asymptomatic cervical stenosis to clinically symptomatic myelopathy within 2 years (odds ratio 12.5 [$P < 0.001$] for EMG). This underscores the value of EMG at discerning physiologic axonal loss and its usefulness in outcome prediction [31].

SUMMARY

This chapter reviews the electrodiagnostic approach to evaluation and testing patients with upper limb or neck symptoms suspected of having a cervical radiculopathy. One cannot overemphasize the importance of the clinical evaluation and differential diagnosis formulation by the electrodiagnostic medical consultant to guide testing. Musculoskeletal disorders are quite common and frequently coexist with entrapment neuropathies and cervical radiculopathy. The needle EMG examination is the most useful single electrodiagnostic test, but is limited in its sensitivity. EMG screening examinations using six muscles optimize identification yet minimize patient discomfort. Nerve conductions studies and F-waves are not very useful for confirming radiculopathy. They are useful,

however, to exclude polyneuropathy or mononeuropathy. Electrodiagnosticians should understand the strengths and limitations of electrodiagnostic testing to effectively use this important diagnostic tool when evaluating patients with suspected radiculopathy.

ACKNOWLEDGMENT

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41 Epidural Steroid Instillation for Cervical Radiculopathy

Todd M. Reiter, Ray M. Baker, and Michael J. DePalma

INTRODUCTION

Cervical spine disorders have been estimated to affect 9% to 12% of the general population, rivaling their lumbar counterpart as a common presenting complaint to the health care practitioner [1]. The average annual age-adjusted incidence rates for cervical radiculopathy have been documented as 83.2 per 100,000 [2]. The pathophysiologic basis for radicular pain or radiculopathy rests upon three proposed mechanisms: biomechanical [3], biochemical/inflammatory [4–21], and neovascularization [22–24].

Although clearly distinct, the terms radiculopathy and radicular pain are often used interchangeably. For the diagnosis of radiculopathy to be made, a loss of sensation, loss of myotomal strength, or a loss of muscle stretch reflex is required. Radicular pain, on the other hand, refers to pain in the normal distribution of a spinal nerve, with or without a loss of neural function. To avoid repeating the phrase "radiculopathy or radicular pain" we will use the term radicular pain throughout this chapter, unless the data presented are specific to radiculopathy.

Biomechanical compression of cervical nerve roots has been clearly established as a source of radicular pain [3]. In contradistinction to the lumbar spine, soft disc protrusions are a less common cause of cervical radicular pain, accounting for only 21.9% of all patients with radicular pain. Spondylotic foraminal encroachment, resulting from decreased disc height, degenerative changes of the uncovertebral joints, and zygapophyseal joints degeneration, accounts for 68.4% of patients with radicular pain (Figure 41.1). The most affected nerve root is C7, followed by C6 [2]. Spinal ganglion lesions, infectious processes, and tumor-associated soft tissues are less common causes of compressive cervical radicular pain [25,26].

Biochemical/inflammatory sources are a second cause of radicular pain and may be present even when herniated disc material is not in direct contact with a nerve root [26]. Histobiochemical and clinical studies have shown that inflammatory mediators can account for much of the pathophysiology of radicular pain and can accelerate disc degeneration [4–6,8–21,27,28].

The clinical course of radicular pain due to herniated intervertebral disc is often associated with gradual improvement over a period of a few weeks to months [2,29–34]. Over this period, 50% to 60% of patients will improve clinically, irrespective of radiographic findings [33,35–37]. Given that asymptomatic disc herniations have been documented in the cervical spine [38–40] in 5% to 57% of normal subjects, increasing with the age of the subject, the extension of nuclear material through a rent in the annular fibers represents a potentially reversible abnormality responsible for limb pain owing to nerve root insult. Such an injury may result in acute biochemical and/or biomechanical harassment of the spinal nerve root. However, the biomechanical or biochemical insults can abate over time, allowing for resolution of signs and symptoms of nerve root injury. In this sense, the pathophysiologic components of the disc herniation are effectively reversible.

The true natural history of cervical radicular pain is more complex, however. Although certainly many patients will improve, with or without treatment, over the course of several months, cervical radicular pain tends to involve a relapsing, remitting course over time. Indeed, recurrence of symptoms occurs in almost one third of patients over a 5-year period [2].

If acute symptoms persist beyond 6 weeks, despite appropriate care including physical therapy, oral anti-inflammatory medications, and a tincture of time, fluoroscopically guided epidural corticosteroid or selective nerve root injections are an appropriate next step in the treatment algorithm [29–31,33]. Symptoms will often improve with one to four injections as the inflammatory response of the herniation is rendered inert [32,41,42].

Effective cervical epidural steroid injections are based upon an understanding of cervical spine anatomy, the pathophysiology of radicular pain, and the location of steroid instillation. Commonly, one of two techniques is utilized to deliver injectate into the cervical epidural space. The cervical interlaminar epidural steroid injection (CILESI) approach involves advancing a needle between adjacent lamina, through the ligamentum flavum, and into the dorsal epidural space. The cervical transforaminal epidural steroid injection (CTFESI) approach places the injectate at the middorsal aspect of the intervertebral foramen by advancing a needle behind the exiting nerve root. This chapter will review the many tools and techniques that have been developed to increase the accuracy of needle placement, the safety profiles, and predict outcomes of both CILESI and CTFESI together. However, each of these two techniques is separate and distinct in their relevant anatomy, outcomes, and relative pros and cons, so these topics will be described separately.

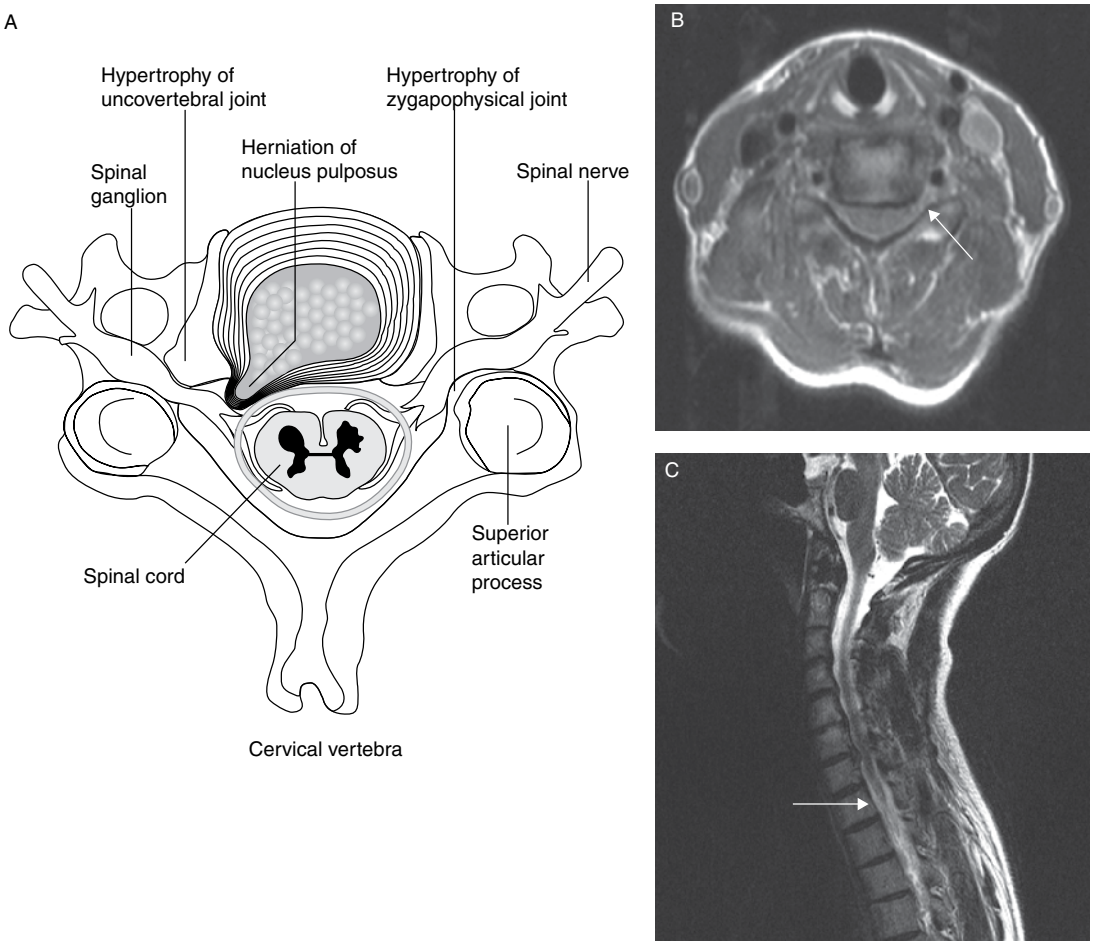

Figure 41.1 Causes of cervical radicular pain. Foraminal encroachment of the spinal nerve from degenerative changes in the uncovertebral and zygapophyseal joints and herniation of the nucleus pulposus are the two most common causes of cervical radicular pain **(A)**. T(2)-weighted magnetic resonance imaging in a sagittal view **(B)** and axial view **(C)** shows a herniated disk and an osteophytic spur at C6-C7 paracentral to the left side with compression of the exiting C7 nerve root. There is no evidence of spinal cord compression. Courtesy of Ray Baker, MD.

CERVICAL TRANSFORAMINAL EPIDURAL STEROID INJECTIONS

CTFESIs may be performed with the patient lying in a supine, oblique, or a lateral decubitus position (see Figure 38.19, Chapter 38), depending on operator preference and patient comfort. The position must allow adequate visualization of the cervical intervertebral foramina in anteroposterior (AP), lateral, and oblique planes.

The critical first step is to obtain a correct oblique view of the target foramen as viewed in Figure 41.2. This usually involves adding 10° to 15° of caudal to cranial tilt to the fluoroscope, and then rotating the beam laterally until the foramen is maximally opened, and the ventral wall of the superior articular process projects sharply onto the silhouette of the lamina. Through a puncture point just ventral to the superior articular process, a needle is passed toward the foramen. The needle tip should always lie over the ventral half of the superior articular process maintaining a maximal distance from the vertebral artery as noted in the diagrammatic representation in Figure 41.3. When the needle has reached the superior articular process, the tip should be readjusted to enter the foramen tangential to its dorsal wall, opposite the equator of the foramen.

Using an AP view, noted in Figure 41.2, the tip of the needle should be adjusted to lie opposite the sagittal midline of the articular pillar. The needle should never be placed beyond the uncovertebral joint, and care should be taken if the needle is placed beyond the sagittal midpoint as the dural sleeve can be encountered with subsequent subarachnoid spread of injectate. The final needle position should be checked and recorded on an oblique view, which documents placement against the posterior wall of the foramen, and on an AP view, which documents depth of insertion. Both the oblique and AP views should also be viewed and recorded after injection of contrast.

Under direct, real-time fluoroscopy in the AP view, a small volume of nonionic contrast medium is injected through microbore tubing connected from the needle hub to a syringe. The contrast should outline the dorsal root ganglion and spread centrally toward the epidural space as in Figure 41.4. Real-time fluoroscopy is essential to check for inadvertent vascular injection, which may occur even if the needle is correctly placed. Intraarterial

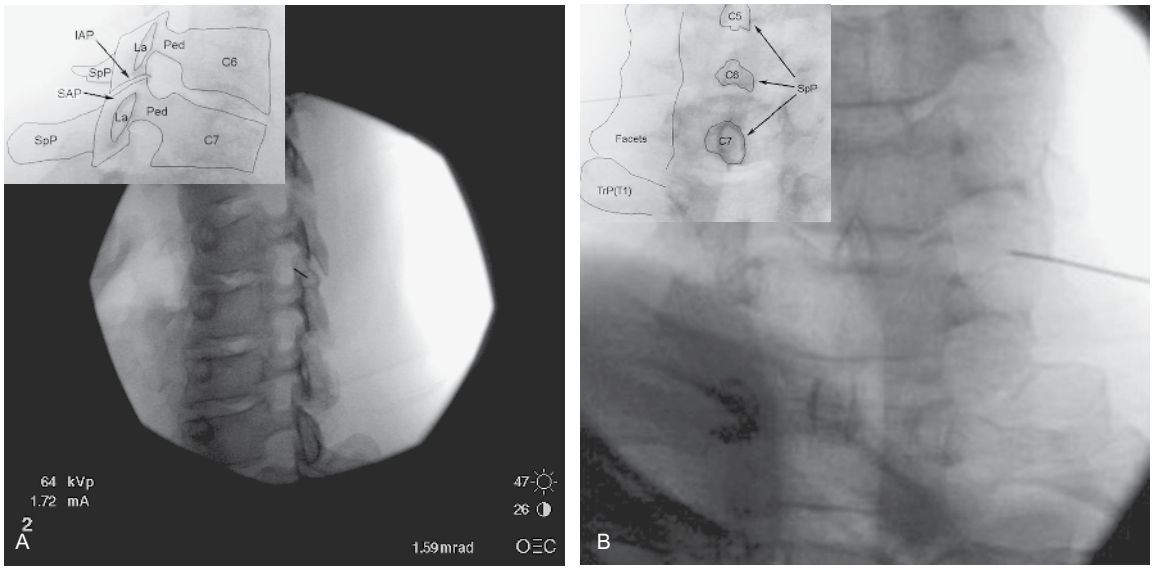

Figure 41.2 **(A)** Right anterior oblique radiograph demonstrating a needle in position along the posterior aspect of the right C4-C5 intervertebral foramen. *Inset* of midportion of image with bony structures labeled: C4 = C5 vertebral body; C5 = C5 vertebral body; IAP = inferior articular process; LA = lamina; Ped = pedicle; SAP = superior articular process; SpP = spinous process. **(B)** Anteroposterior radiograph demonstrating needle in final position within the right C6-C7 intervertebral foramen. The needle lies halfway between the medial and lateral borders of the articular pillars. *Inset* of midportion of image with bony structures labeled: Facets = medial and lateral aspect of the facet column; SpP = spinous processes of C6, and C7; TrP (T1) = transverse process of T1. Courtesy of Ray Baker, MD.

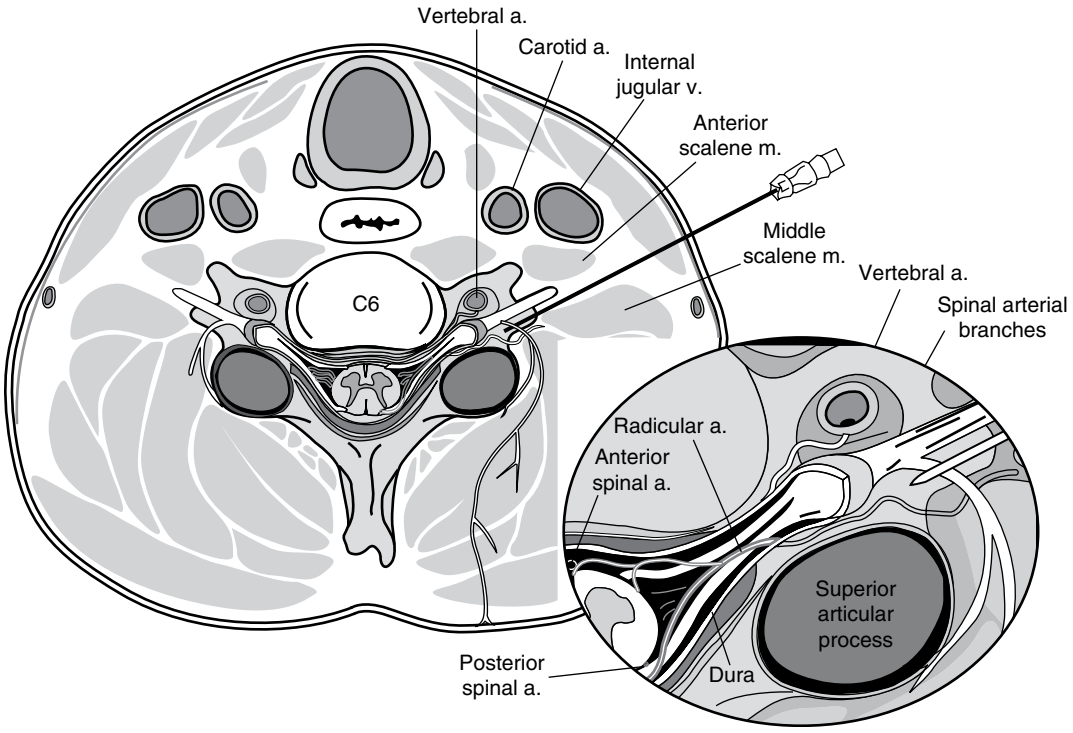

Figure 41.3 Illustration of an axial view of the cervical intervertebral foramen and adjacent structures at the level of C6 with a needle inserted parallel to the axis of the foramen along its posterior wall. Note the proximity of adjacent structures: C6 = vertebral body of C6. Redrawn with permission from Ref. [55].

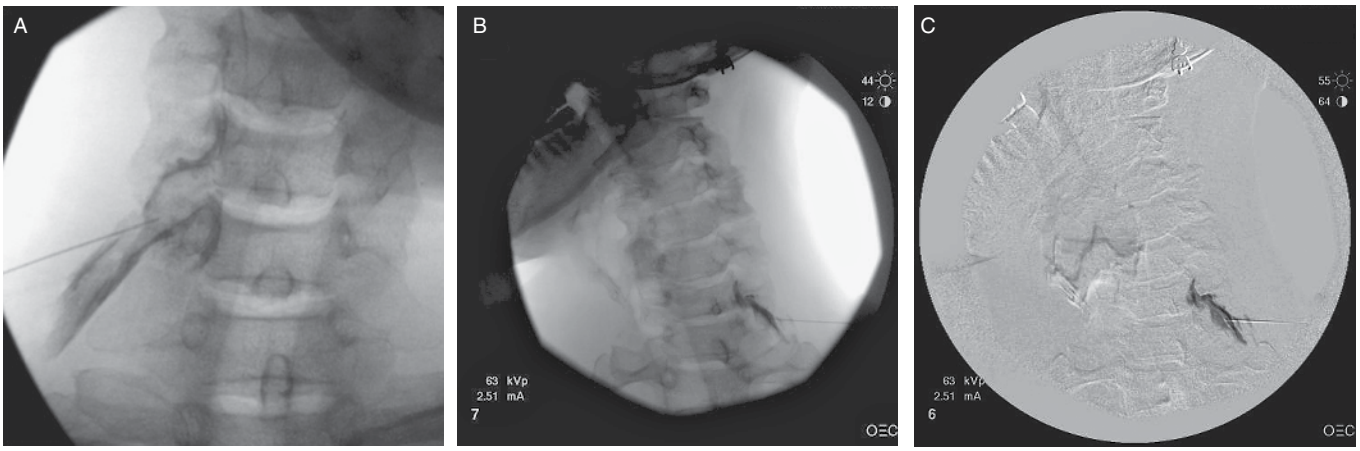

Figure 41.4 An anteroposterior view of a CTFESI after injection of contrast medium, before planned transforaminal injection of corticosteroids. **(A)** Image as seen on fluoroscopy. The needle lies in the left C5-C6 intervertebral foramen no further medially than its mediolateral point. Contrast medium outlines the exiting nerve root. **(B)** The radicular artery appears as a thin thread passing medially from the site of injection. **(C)** Digital subtraction angiogram after pixel-shift re-registration reveals that the radicular artery extends to the midline to join the anterior spinal artery. Courtesy of Ray Baker, MD.

injection is manifest by rapid clearance of the injected contrast. Injection into a vertebral artery results in a rapid ascending vertical flow, whereas injection into a radicular or medullary artery is typically horizontal toward the midline. These smaller caliber arteries do not cross the midline, and are characterized by a smaller caliber relative to veins, and by a tortuous course with distal tapering. Contrast medium commonly fills epiradicular veins, which are recognized by the slow clearance of the contrast and by an irregular course that might ascend laterally or even flow across the midline before draining inferiorly as noted in the angiogram and digital subtraction angiography (DSA) in Figure 41.4.

Although only a small volume of contrast medium (1.0 mL or less) is usually required to outline the dorsal root ganglion, sufficient contrast should be injected to allow for confirmation of appropriate flow and to exclude aberrant flow into a vessel or subarachnoid space. As contrast spreads into the lateral epidural space, it assumes a linear configuration. Rapid dilution of the contrast medium implies subarachnoid spread, which may occur if the needle has punctured the thecal sac or a lateral dilatation of the dural root sleeve in the intervertebral foramen. After contrast challenge and negative aspiration, provided adequate periradicular and epidural spread is achieved, an anesthetic challenge is performed by injecting a small volume of anesthetic (e.g., 0.8 mL of preservative-free 2% xylocaine). After a minimum of 75 seconds have elapsed, the patient should be queried for symptoms of light-headedness, dizziness, metallic taste, tinnitus, or sensorimotor changes. If the patient affirms any of these symptoms or demonstrates any sensorimotor impairment, corticosteroid should not be injected, and further contrast should be injected to verify a safe flow pattern. Digital subtraction imaging (DSI) can be especially useful in these cases to distinguish previously injected contrast from a more recent reinjection. Additionally, the initial symptoms of a severe allergic reaction can partially mimic symptoms of a vascular injection, as patients might experience light-headedness, dizziness, or an "odd feeling" well before pruritus, hive formation, or cardiovascular changes occur.

CTFESI Outcomes

Studies of CTFESIs have demonstrated positive results for all the prospective nonrandomized studies in long and/or short-term pain relief (Table 41.1) [43–51]. The Anderberg [43] study, which is the only randomized prospective study to date, revealed no difference at 3 weeks between subjects in the control group who received a single transforaminal injection of 0.5 mL mepivacaine 1% and 1 mL saline versus the active group which received 0.5 mL mepivacaine 1% plus 1 mL methylprednisolone (40 mg/mL). Although the addition of methylprednisolone did not improve outcomes over Mepivicaine alone, both groups improved after one injection. In addition, only one injection was explored rather than multiple successive injections.

Given the paucity of controlled prospective trials and conflicting outcomes of retrospective studies a definitive statement about the efficacy of CTFESIs remains pending. However, these interventions appear effective in ameliorating or eliminating disabling cervical radicular pain in many patients and are therefore meritorious and offer a vital treatment option. More rigorous scientific study of the efficacy and safety of CTFESIs is both necessary and warranted given the conflicting nature of current published studies.

TFESI Pros and Cons

Pros

- Target specific allowing delivery of corticosteroid closest to the site of pathology.

Table 41.1 CTFESI prospective and retrospective outcome studies demonstrating long and/or short-term pain relief results.

Study/Methods	Method	Participants	Intervention(s)	Timing/ Outcome(s)	Result (s)	Conclusion (s)
Kumar, 2008 [44]	Retrospective	33 patients with radicular pain due to cervical disc disease and/or foraminal stenosis with correlative MRI findings	Two needle transforaminal technique with fluoroscopic guidance	Timing: 6 week, 1 year, and 2 years. Outcome measure: Neck Disability Index and VAS	28 showed good to excellent clinical response	Positive short-term and long-term relief
Anderberg, 2007 [43]	Prospective randomized	40 patients with one-sided cervical radicular pain, radicular arm pain, positive diagnostic transforaminal SNRB at the level of correlative MRI findings	Single transforaminal injection: Control with anesthetic agent only, study group with anesthesia and steroid under fluoroscopic guidance	Timing: Immediately after procedure and 3 week post. Outcome measure: Questionnaire	Control group: six patients reported remaining effect at 3 weeks. Study group: six patients reported remaining effect at 3 weeks	No differences in treatment results in the two patient groups
Kim, 2007 [45]	Prospective	19 consecutive patients presenting with radiating pain to the shoulder or arm had CT or MRI scan findings compatible with cervical herniated disc or foraminal stenosis	Cervical transforaminal steroid injection using multislice CT fluoroscopy guidance up to three times with a minimal interval of 2 weeks	Timing: 2, 4, 8, and 16 weeks. Outcome measure: VAS	Significant pain improvement by VAS score at the second, fourth, eighth, and sixteenth week compared with week 0	Positive short-term and long-term relief
Dreyfuss, 2006 [46]	Prospective	38 consecutive patients with single-level, unilateral radicular pain with advanced imaging demonstrating single-level neural compression	Patients received a single cervical transforaminal epidural injection with either dexamethasone or triamcinolone	Outcome measure/ timing: VAS and verbal integer scale at 4 weeks	Pain relief by VAS > 50%: dexamethasone group 60%; triamcinolone group 67%	Positive short-term relief. Long-term not tested
Lin, 2006 [47]	Retrospective	70 patients with MRI confirmed herniated cervical discs with nerve root impingement, offered a trial of cervical transforaminal epidural injections while awaiting surgery	Cervical transforaminal with local anesthetic and steroids	Pain relief by Odom's criteria and avoidance of surgery	Of the 70 treated patients, 44 (63%) had significant relief of their symptoms and did not wish to proceed with surgical treatment	Positive short-term and long-term relief
Kolstad, 2005 [48]	Prospective	21 patients with either cervical disc herniation or spondylosis confirmed by advanced imaging awaiting cervical disc surgery	Patients received two transforaminal cervical epidural injections 2 weeks apart	Timing: 6 weeks and 4 months. Outcome measures: VAS, Odom's criterion, and surgical intervention	5 of the 21 patients canceled their surgery because of improvement in pain	Positive short-term and long-term relief
Cyteval, 2004 [49]	Prospective	30 patients with cervical radicular pain, 16 patients with foraminal degenerative stenosis, 14 patients with disk herniation	Periradicular foraminal steroid (dexamethasone) infiltration under CT control	Timing: 2 weeks, 6 months. Outcome measures: VAS	Greater than 50% pain relief was reported in 60% of patients. There was no rebound of pain at the 6-month follow-up	Positive short-term and long-term relief
Vallee, 2001 [32,50]	Prospective	32 patients with 34 cervical radicular pain foci with correlative radiographic findings	Periradicular foraminal steroid infiltration under fluoroscopic guidance	Timing: 14 days, 3, 6, 12 months. Visual pain scale	Radicular pain in 22 of 34 cervical radicular pain had fair to excellent relief at 12 months	Positive short-term and long-term relief
Bush, 1996 [33]	Prospective	68 patients with neck pain and cervical radicular pain with neurologic signs (except one) and advanced imaging correlating with signs/symptoms (except one)	Patients received an average of 2.5 transforaminal cervical epidural steroid injections	Timing: 1 month to 1 year. Outcome measures: VAS and neurologic examination	None of the patients required surgery. 93% of the patients were reported to have good pain relief lasting for 7 months. 7% lost to follow-up	Positive short-term and long-term relief

- May offer diagnostic utility in multilevel disc herniation.
- May determine a primary pain generator in the cervical shoulder syndrome or cervical and peripheral nerve involvement.
- May determine the symptomatic root in patients with documented postoperative fibrosis.

Cons

- The cervical intervertebral foramen is near the vertebral artery and other vascular structures, and injection confers the risk venous or arterial injection.
- Because of the proximity of the needle to the spinal nerve root and the spinal cord, there is a possibility of neural trauma.
- The level of evidence for the efficacy of CTFESIs is inconsistent with only one randomized controlled trial.
- Transforaminal approach may be difficult for patients with previous fusion and/or hardware.

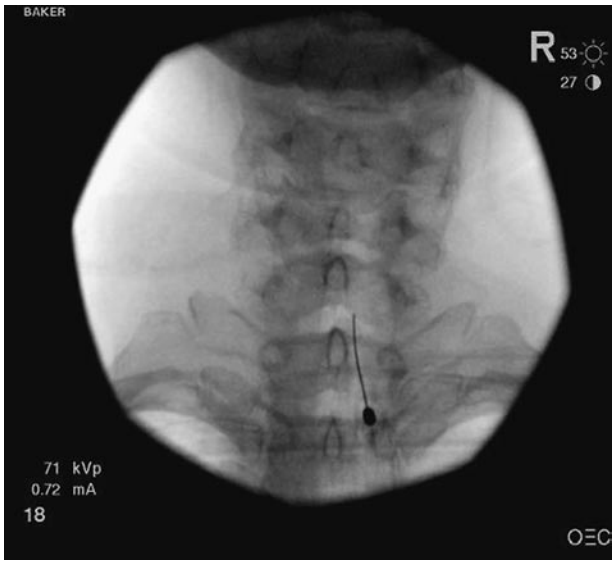

Figure 41.5 Demonstrates proper needle placement during an anterior to posterior radiograph of a C7-T1 interlaminar epidural steroid technique.

CERVICAL INTERLAMINAR EPIDURAL STEROID INJECTIONS

Although a sitting position is still used by some practitioners, most have adopted variations of the prone position described earlier. Advantages of the prone position include a more secure patient with less chance of movement, particularly if the patient is sedated. Additionally, the fluoroscopy table represents a more stable platform for the performance of emergency maneuvers in case of an allergic reaction, vasovagal event, or respiratory or cardiovascular embarrassment. Pillows, blankets, and other makeshift pads have been replaced by fluoroscopically friendly radiolucent positioning devices with articulating, multipositional face rests.

Using an AP fluoroscopic view, the desired cervical interlaminar space is located (Figure 41.5). Currently, the C6-7 or C7-T1 interspaces are felt to be the safest, while not compromising efficacy. Not only is the ligamentum flavum more consistent in the lower cervical and upper thoracic spine, but, should inadvertent injury occur such as a direct needle injury or injection into the spinal cord, the resulting neurologic deficits will be less devastating. Goel [51] showed that injection of 2 to 4 mL of contrast reliably spreads rostral to C3 when interlaminar injection is performed at C7-T1. Further, there have never been any data to indicate that interlaminar injection above the C6-7 level is associated with superior outcomes. In any case, the level chosen should be checked using advance imaging to ensure that there is adequate room around the cord and an adequate epidural space to perform the procedure safely.

After anesthetizing the skin with 1% lidocaine, a Tuohy or other suitable needle (gauge 18–25) is inserted either midline or paramedian on the side of the patient's symptoms using a coaxial (tunneled, down the beam) view. During advancement of the needle, frequent lateral or contralateral oblique images are obtained to determine the needle depth, and AP images are taken as needed to ensure correct needle trajectory. If a lateral fluoroscopic view is used to monitor depth, the needle is advanced just dorsal to the so-called spinolaminar line—the radiographic line that appears as the posterior spinous process transitions into the lamina as noted in Figure 41.6B. If a contralateral oblique image is used to monitor needle depth, the needle is advanced to a point dorsal to the "string of pearls" created by the elliptical appearance of the lamina when viewed in radiographic cross-section. In all instances, the epidural space is then located by using loss of resistance (LOR) to air, saline, or contrast.

After LOR is encountered, and needle aspiration tests are negative for heme or cerebrospinal fluid, and accuracy of needle placement is assessed by the injection of a small volume of nonionic contrast medium under a lateral or contralateral oblique fluoroscopic view. The contralateral oblique view is particularly helpful in patients with large shoulders or a thick, short neck in whom lateral fluoroscopic imaging is difficult. A typical "dorsal stripe" is noted using either view. Figure 41.6 demonstrates a diagrammatic representation and oblique cervical radiographic view of proper needle position. In the event that the operator is not satisfied that the contrast medium spread is consistent with epidural injection, the needle is repositioned and the process is repeated until adequate contrast medium spread is obtained. If there is suspected dural puncture or if a paresthesia is elicited during needle placement, the procedure is aborted and the patient monitored.

Following the initial injection of a small volume of contrast, a larger volume of contrast medium is injected to confirm epidural placement, rule out partial venous uptake, and to determine any aberrant flow patterns. AP and lateral or contralateral oblique radiographs are once again obtained. Fat globules are outlined on AP imaging,

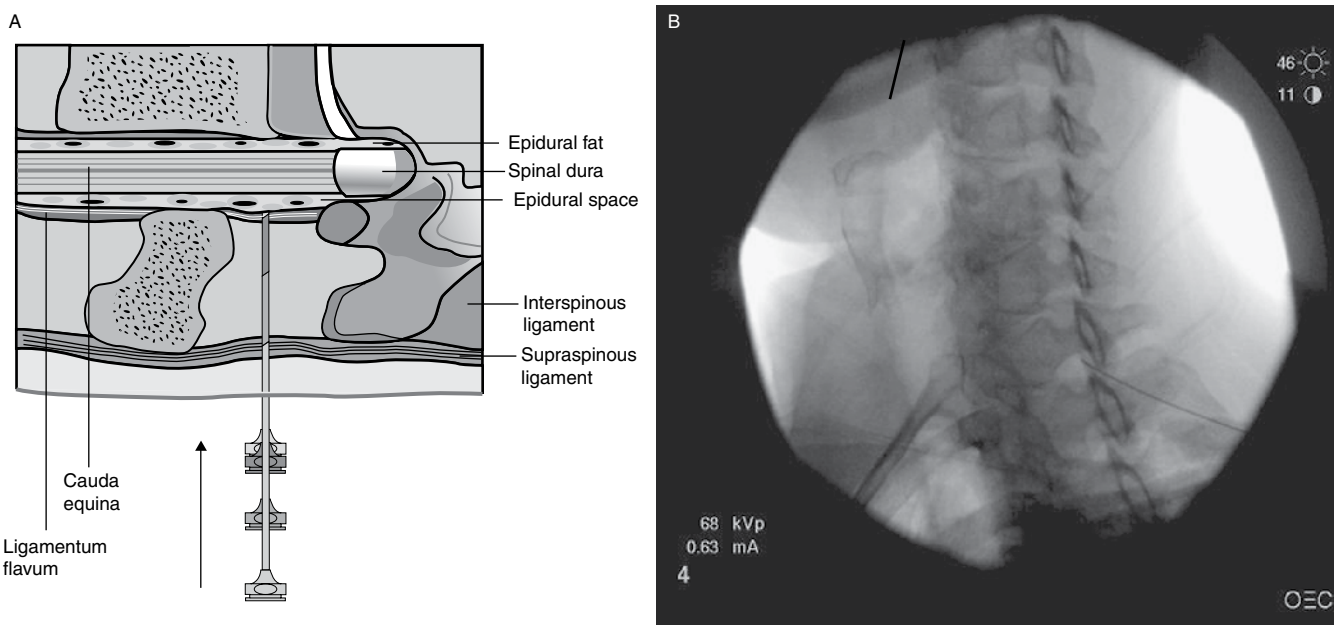

Figure 41.6 **(A)** Is a diagrammatic representation of a cervical interlaminar injection from the lateral view. **(B)** Is an oblique cervical epidurogram of proper needle position at T1-2. Courtesy of Ray Baker, MD.

and contrast will be seen to outline the medial aspect of the pedicle. A short run of real-time fluoroscopy can often detect a partial vascular injection that is missed with spot images. If local anesthetic is used as a part of the injectate, a small volume of a short acting local anesthetic can be used as a test dose. However, as often as not, a test dose to rule out subarachnoid injection merely results in a false sense of security, as manifestations of spinal blockade do not occur immediately. The first manifestation of cervical spinal blockade is often a drop in blood pressure coupled with a decrease in heart rate as a result of early sympathetic blockade. Sensorimotor changes can take several minutes to fully develop.

CILESI Outcomes

There is a relative paucity of randomized prospective clinical trials for the performance of ILESIs for cervical radiculopathy. This review will include only studies that have a cervical radiculopathy and will exclude those ILESI studies for neck or myofascial pain only. There are two randomized prospective clinical trials performed by Stav and Castagnera [52,53]. These are small studies; however, both revealed good long- and short-term results. Stav's [52] 1993 study evaluated 50 patients with chronic resistant cervicobrachialgia who were randomly divided into two groups. Twenty-five patients (group A) were treated with cervical epidural steroid/lidocaine injections and 17 patients (group B) were treated with steroid/lidocaine injections into the posterior neck muscles. One to three injections were administered at 2-week intervals according to the clinical response. Pain relief was evaluated by the visual analogue scale (VAS) at 1 week and 1 year after the last injection. Pain relief was very good or good in 76% of the patients in group A, as compared with 35.5% of the patients in group B at 1 week, and 68% in A and 11.8% in B at 1 year.

Castagnera's [53] prospective randomized study was even smaller and was designed to evaluate the long-term effectiveness of a single CILESI performed with or without morphine. Twenty-four patients had cervical radicular pain absence of isolated facet syndrome and motor weakness and at least 12 months of medically treated pain without surgical indications. They were randomly assigned to two groups and pain relief was assessed at day 1, at months 1, 3, 6, 8, and then yearly for up to 4 years posttreatment (mean follow-up 43 ± 18.1 months). Group S (n = 14) received an equivalent volume of 0.5% lidocaine plus triamcinolone acetonide (10 mg/mL); group S + M (n = 10) received the same combination plus 2.5 mg of morphine sulphate. Pain relief was assessed as the percentage of pain decrease on a 100 mm VAS. A decrease between 51 and 100 mm was considered a success. Long-term results did not differ between groups, with success rates of 78.5% in group S and 80% in group S + M, providing pain relief of 86.8 ± 14.7% and 86.9 ± 17.9%, respectively. Importantly, pain relief did not deteriorate over time. These results suggest that a single CILESI can produce long-lasting pain relief, but that the addition of morphine does not provide additional benefit when combined with steroid. Weakness of the paper includes its failure to reveal how the subjects were randomized or reported if there were any subjects that dropped out of the study. In addition, this study was prospective and did not include a control arm without triamcinolone acetonide.

In 2007, Kwon [45] retrospectively evaluated outcome predictors for fluoroscopy-guided CILESI in 91 patients with neck pain and/or cervical radicular pain. Therapeutic effects were evaluated 2 weeks after the administration of a single CILESI. Outcome measures included VAS and a five-point outcome scale: 0 (aggravated), 1 (stationary), 2 (improved), 3 (much improved), and 4 (no residual symptoms). Of the 76 who met full criteria, 55 (72.4%) experienced a successful outcome, defined as an outcome scale score of 3 or more and a VAS reduction of more than 50%. Patients with herniated discs had significantly better results than patients with spinal stenosis (86.1% vs 60.0%) ($P < 0.05$).

Strub [54] retrospectively assessed categorical factors that could help predict clinical outcome of CILESI for localized neck or radicular pain in 161 patients (average age 58 years; range 26–82 years) who had failed treatment with oral pain medications or physical therapy. The average duration of symptoms was 18.2 months. Up to three CILESIs (mean 1.74) were allowed. Patients were assessed by telephone 10 days after the procedure to determine efficacy and were rated on a four-point pain relief scale: none, minor, some, or substantial. Of the 280 total injections, 233 (83%) resulted in at least minor pain relief, with 40% or patients showing substantial relief. Patients were more likely to experience pain relief if they presented with multilevel degenerative changes (odds ratio [OR] = 4.13, $P = 0.0055$), had radicular symptoms in the hand and/or finger (OR = 2.72, $P = 0.0011$), or underwent injection at the C7-T1 level (OR = 2.44, $P = 0.0034$). Patients who required narcotics for their symptoms before the procedure showed lower odds of pain relief (OR = 0.80, $P = 0.4367$).

Based upon the two studies of CILESI for pain relief lasting longer than 1 year reviewed, it may be concluded that those with cervical radicular pain receiving CILESI experiencing good or very good pain relief was greater than 68%.

Pros and Cons of CILESIs

Pros

- Efficacy extensively studied.
- May be useful in the setting of multilevel pathology or bilateral pathology to obtain wider coverage.

Cons

- Increasing number of complications are occurring relative to CTFESIs [55].
- Risk of spinal cord trauma, either directly from needle puncture or indirectly from epidural hematoma.
- Less target specific and not diagnostic.
- Risk of dural puncture.

IMAGING METHODS

Many tools available to the operator can improve accuracy of the needle location and reduce the risk of vascular or neurologic insult. Cervical epidural steroid injections are being performed with blind LOR, fluoroscopy with epidurography, computed tomography (CT) guidance [56], DSA, and most recently with ultrasound (US) [57–59].

Epidurograms

Fluoroscopically guided epidurography, in conjunction with epidural steroid injections, enhances the safety and accuracy of therapeutic injections and is associated with an exceedingly low frequency of untoward sequelae [60–62]. In one study, epidurograms of 38 interlaminar cervical epidural steroid injections in 31 patients were reviewed [61]. Unilateral epidural contrast spread was found in 51% and ventral spread was found in 28% of cases. The effects of age, gender, magnetic resonance imaging (MRI) results, previous cervical laminectomy, and the operator's level of training correlated with results. In addition, Goel [51] showed that injection of 2 to 4 mL of contrast reliably spreads rostral to C3 when interlaminar injection is performed at C7-T1. Another study by Kim evaluated epidurography contrast patterns in fluoroscopic-guided CILESI using the midline approach at the C6-7 level. Epidurography was performed with 1, 2, or 3 mL of nonionic contrast medium. The findings concluded that 2 mL of contrast solution can provide optimal dispersion of contrast in a ventral and longitudinal spread [62]. Despite a paucity of methodologic examination, CTFESIs reliably deposit contrast and subsequent corticosteroid within the anterior epidural space on the side of symptoms.

CT-Guided Epidurography

The effect of volume on transforaminal epidural contrast spread was investigated in nine patients using multislice CT and three different volumes of injectate (0.6, 1.1, and 1.7 mL) [63]. Postinjection CT scans revealed nonselective spread to adjacent nerve roots with larger volume. Local anatomy (size of foraminal area) was also a factor. In all the patients, perineural, intraforaminal, and extraforaminal distribution was observed. The length of perineural distribution of contrast varied from 18 to 49 mm (mean 36 mm) and was not correlated to the volume injected. Only five of the nine injections met criteria for selective nerve spread, including all three of the injections using 0.6 mL volume and two of the three injections using 1.1 mL volume.

Another study evaluated the safety and efficacy of multislice CT fluoroscopy (Figure 41.7 showing ideal needle placement for transforaminal steroid injection) in the performance of transforaminal epidural steroid injections. Nineteen consecutive patients, presenting with radiating pain to the shoulder or arm and CT or MRI findings compatible with cervical herniated disc or foraminal stenosis, underwent up to three cervical transforaminal steroid injections at intervals of at least 2 weeks. At 16-week follow-up, there were no serious complications during or after the procedures [56]. However, CT does not allow real-time visualization of contrast flow patterns. Such imaging characteristics are critical to ensure the absence

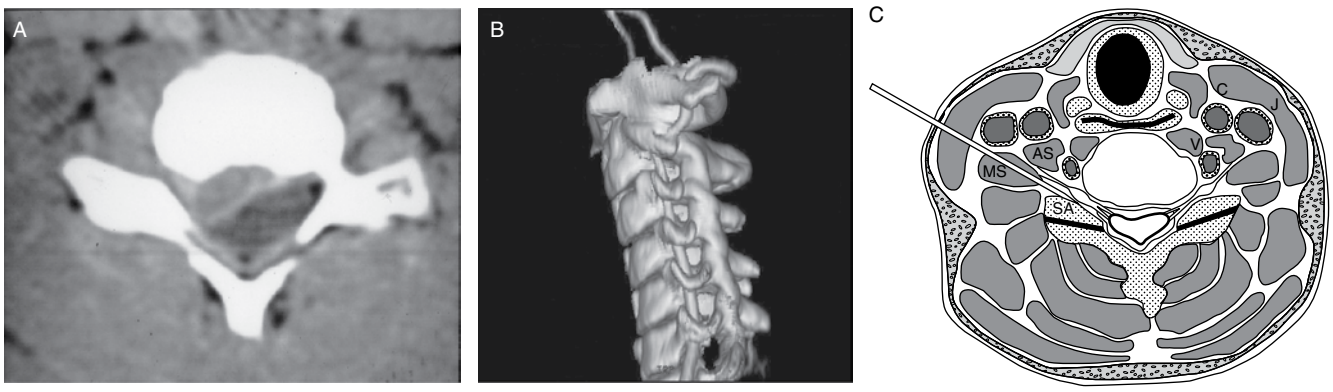

Figure 41.7 Diagnostic CT scan image of C5-6 level shows a herniated disc at the right intervertebral foramen **(A)** CT reconstruction image of oblique cervical spine **(B)** and the ideal needle path for the transforaminal steroid injection which is modified from Rathmell's Ref 55 **(C)**. AS, anterior scalene; C, carotid artery; J, internal jugular vein; MS, middle scalene; SA, superior articular process; V, vertebral artery. Courtesy of Ray Baker, MD.

of intravascular uptake [64]. Although CT studies are useful in demonstrating anatomy and injectate flow patterns, this should not imply that CT is a safe alternative to fluoroscopic guidance. Although vascular complications of CTFESIs are rare, studies involving low numbers of patients are not sufficient to prove safety, and in addition to multiple cases sub judice, at least one complication involving injection into a vertebral artery under CT guidance has been reported [65].

Digital Subtraction Angiography

DSA consists of injecting small amounts of water-soluble contrast medium into an artery or vein, then electronically collecting x-ray signals for computerized alignment, forming images that highlight vascular flow patterns. When used to detect vascular flow when intravascular injection is not intended, the term *digital subtraction imaging* is more appropriate. The technology used in DSA or DSI is available on newer fluoroscopic units and is a useful tool for documentation of needle placement and contrast flow, especially when previously injected contrast or hardware placement obstructs adequate visualization. It might also be helpful in the detection of intravascular injection during interventional techniques [66–69]. Despite appropriate care and accurate technique, it is possible for injectate to find its way into a radicular or medullary artery [69]. In this case study, the authors believed that DSI, with the use of contrast medium, detected the filling of a radicular artery that passed to the spinal cord during a TFESI. The procedure was abandoned, and the patient suffered no ill effects (Figure 41.8).

US Guidance

US is currently being investigated on cadavers as seen in Figure 41.9 and correlated with CT to determine if sonography may be used as a safe and effective tool to replace the radiation exposure of both CT and fluoroscopy [58,59] .In addition, a study has been performed using sonographic estimation of needle depth for cervical epidural blocks on 50 patients at C6–7 [57]. The cervical epidural block was successfully performed on 48 patients (96%). There were two incidents (4%) of dural puncture with no bloody taps, postprocedure complications, or hemodynamic instability related to cervical epidural blocks occurred. Further work will be needed to determine whether or not US can safely and effectively replace existing imaging methods in the performance of CTFESIs or CILESIs. Not only must target accuracy be validated, but the ability of US to determine appropriate injectate flow patterns must also be validated, particularly with regards to recognizing arterial flow of injectate.

SAFETY PROFILE

Although both the CILESI and the TFESI provide effective outcome evidence, a careful consideration of their safety profile is critical. It is essential to understand both the character and frequency of complications related to the CILESI and TFESI as they pertain to general spinal procedures. Accessibility to this information is critical in reviewing one's own protocols and in reducing complications. A patient's understanding of the relative risks, benefits, and alternatives is equally critical for informed choice. Possible impediments to gaining an exact numerical determination of complication rates relative to cervical epidural procedures include litigation, inaccurate or unpublished case reports, and patient reluctance, or refusal, to authorize the release of their records for publication [69–71]. Moreover, interpretations of the literature must reflect recent innovations in technique, and a more comprehensive view of the anatomy.

Complications common to both TFESI and ILESI include infections, arachnoiditis, direct injury to either the spinal cord or nerve root, and spinal hematomas. In fact, infections of the spine have been reported in nearly every type of spinal injection including discitis, intradural/subdural abscess, epidural abscess, and meningitis [72]. In addition, local non-neural effects may involve intrathecal injection of

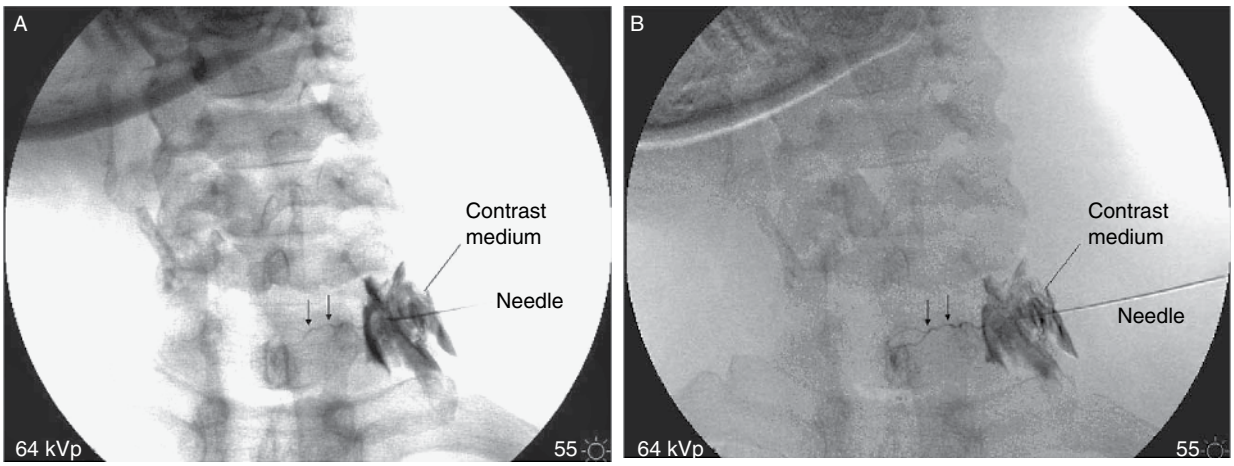

Figure 41.8 An anteroposterior view of an angiogram obtained after the injection of contrast medium, prior to the planned transforaminal injection of corticosteroids. The needle lies in the C6-7 intervertebral foramen no further medially than its mediolateral midpoint. The intervertebral foramen contains contrast medium. The arrows indicate the artery that was filled, and which passes medially to the spinal cord. **(A)** Conventional fluoroscopic exposure. **(B)** Digital subtraction view. Courtesy of Ray Baker, MD.

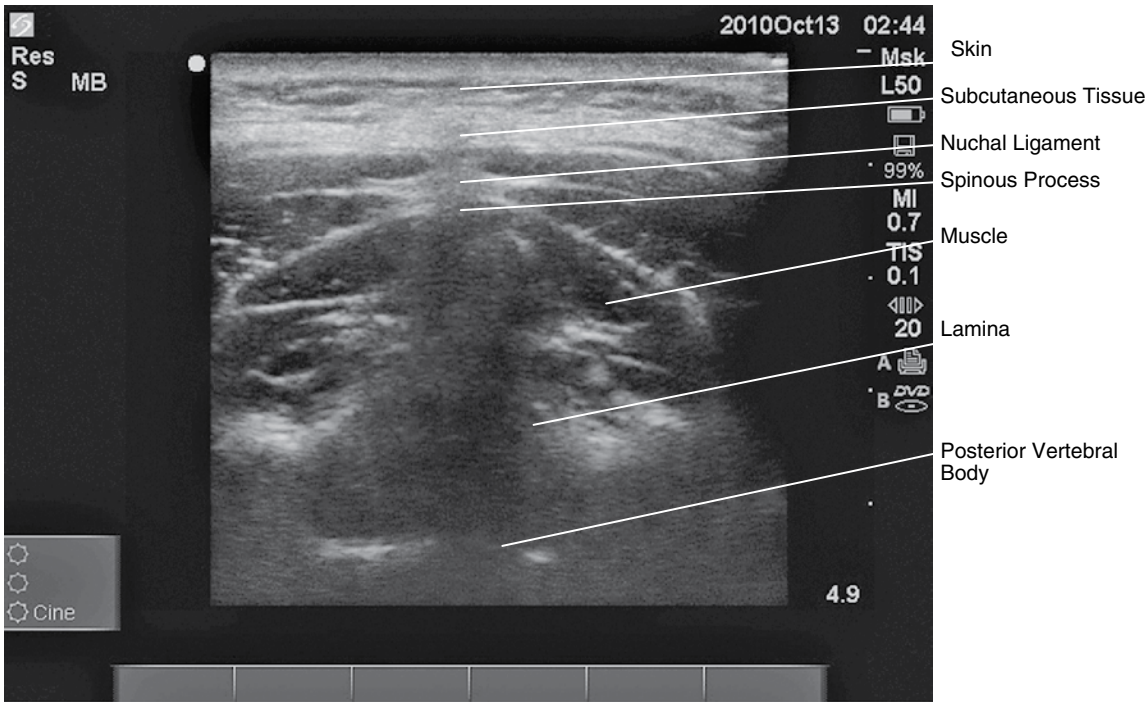

Figure 41.9 Ultrasound imaging of cervical vertebra 6–7. Transverse view shows the skin, subcutaneous tissue, nuchal ligament, spinous process, muscle, lamina, and posterior vertebral body. Courtesy of Gulf Coast Ultrasound Institute and Todd Reiter, MD.

corticosteroids, which may pose the threat of arachnoiditis because of the chemicals contained in the preparations [73]. Risk of intrathecal injections may best be reduced with the use of fluoroscopy with contrast dye, as it is possible to have negative aspiration because of the bevel being partly through the dura and have the injectate still enter the subarachnoid space.

Both CILESI and TFESI generate the risk of contacting and injuring the spinal cord or spinal nerve with the needle tip, resulting in complex regional pain syndrome, local nerve root pain, dysesthesia, nerve root injury, or funicular spinal cord injury [74–76]. Theoretical risk reduction of contacting the neural structures with the needle tip include avoiding levels with large disc protrusions due to their mass effect, possibly compressing the spinal cord into the already very small epidural space [74]. Because of ligamentum flavum discontinuity in the upper thoracic and cervical spine, sole reliance on LOR technique must

be avoided [77,78]. Some have warned against sedation for cervical epidurals because of the patients being unresponsive to pain that would otherwise warn the physician of a compromised spinal cord or nerve root [76,79]. Unusual and rare risks may occur even with strict adherence to the "standards of care," including visual complications [80,81] and pneumocephalus [82].

Spinal hematomas are very rare in any spinal procedure (613 cases reported from 1826 to 1996) but have been documented [83,84]. A study of more than 1 million epidural anesthetics and subarachnoid blocks yielded only 20 cases of hematomas [85]. Even though spinal hematoma may occur in a young healthy person not on anticoagulation therapy, a review of the patient's medication list for anticoagulants should be performed preprocedurally [84].

A 2007 literature review of CILESI revealed a complication rate range from <1% to 16.8% [50,52,86–89]. The differences in the literature and much of the difficulty identifying an accurate complication rate is based upon the definition of "complication" which ranges from mild to major adverse outcomes [87]. Minor complications may be any adverse event resolved relatively quickly (usually within 24 hours) without long-term sequelae. These include increased axial neck pain [88,90], nonpositional headaches [88], facial flushing, vasovagal episodes [88,89,91–93], vomiting [90], fever the night of the procedure (0.3%) [88], 24 hours of subjective upper limb weakness [90], insomnia during the night of injection (1.7%) [88], and superficial infection at the injection site [92]. Major complications may include epidural hematoma, subdural complications, neuropathic symptoms, permanent spinal cord injury, and cervical epidural abscess.

The largest known anonymous survey study to date of TFESIs [67] included 287 US physicians of the American Pain Society. In all, 78 complications were reported, including 16 vertebrobasilar brain infarcts, 12 cervical spinal cord infarcts, and 2 combined brain/spinal cord infarcts. Thirteen cases resulted in a fatal outcome. Major complications primarily result from vascular injuries to the vessels supplying the nerve root or spinal cord, infections and, steroid-related effects [55,67,69,71].

During TFESI, embolism as a result of injection of particulate steroids (e.g., triamcinolone or methylprednisolone) may be reduced or eliminated by the use of nonparticulate steroids (dexamethasone or prednisolone) even when administered directly into the vertebral artery in animal studies [46,94,95]. A prospective randomized study of fluoroscopically guided TFESIs was performed using 32 subjects with single-level radicular pain, and corresponding unilateral nerve root compression on advance imaging, to determine the effects of particulate (triamcinolone) versus nonparticulate (dexamethasone) steroids [46]. All patients completed a phone interview 4 weeks after treatment. A Visual Analog Pain Scale was used preprocedurally, and a verbal integer scale was used at 4 weeks to assess the severity of the patients' radicular pain. Both groups exhibited statistically and clinically significant improvements in pain at 4 weeks with no statistically or clinically significant difference between the two groups. It appears that using a nonparticulate steroid preparation for CTFESIs (dexamethasone) is reasonable to minimize risk of embolic insult and achieve therapeutic benefit.

Review of the cervical spine anatomy earlier revealed the close connection of the spinal nerve roots to their vascular supply. A prospective study of 337 patients and a total of 504 TFESI revealed fluoroscopically confirmed intravascular-contrast injections of 19.4% [96]. In a study of 10 embalmed cadavers, 95 intervertebral foramina were dissected and 2 were carried down to the spinal cord level, demonstrating the anterior spinal, radicular, and segmental medullary arteries [97]. This study specifically sought to determine if the ascending or deep cervical arteries supplying radicular or segmental medullary arteries were potentially susceptible to cannulation or needle trauma (22-gauge spinal needle is 0.711 mm outside diameter) during transforaminal injections. The study revealed that in 21 of the 95 cervical foraminal areas examined, the parent ascending or deep cervical artery, or large branch of it, was within 2 mm of the needle path for TFESI procedure. Thirteen of the 21 vessels could potentially be penetrated only if the needle was not sufficiently advanced into the foramen. Also, those 13 vessels did not wholly advance into the foramen, did not contribute major spinal branches, and had no demonstrable communication with the spinal circulation except smaller twigs to the ventral rami. Transforaminal epidural steroid injections have been associated with anterior spinal artery syndrome [69,98].

Risk reduction for TFESI may be obtained with appropriate needle placement [41], use of a blunt needle [99], fluoroscopic or CT guidance [100], DSA, a test dose of local anesthetic followed by a brief exam [69], minimal-volume extension tubing to reduced needle movement during change of syringes [69,100], use of nonparticulate steroids [46], and use of nonionic versus ionic contrast medium for epidurography [101]. Aspiration-prior injection has a very low sensitivity of 45.9% but a high specificity of 97%; therefore, it is not a good screening tool [96].

PREDICTING OUTCOMES

Diagnostic Selective Nerve Root Blocks

Although advanced imaging techniques can diagnose areas of narrowing or potential neural impingement, they cannot discern if those areas are symptomatic [39]. In 1971, MacNab [3] demonstrated the value of diagnostic, selective nerve root blocks (SNRBs) in the preoperative evaluation of patients with negative or inconclusive imaging studies and clinical findings of nerve root irritation. Since then, nerve blocks have been used to diagnose the source of radicular pain when imaging studies suggested possible compression of multiple nerve roots [3,102–112].

Bogduk [113] suggests that, for a diagnostic spinal nerve block to be positive, it should completely relieve a patient's radicular pain or should produce numbness in the territory in which a paresthesia was previously felt. If the symptom is numbness, anesthetizing the responsible nerve should produce no change in the numbness or perhaps accentuate the numbness. He further postulated

that if a nerve is anesthetized that is not responsible for the patient's symptoms, pain will not be relieved and numbness will occur in a distribution that does not correspond to the distribution in which the patient ordinarily experiences pain.

Anderberg et al. [114] performed a prospective study on 20 patients with cervical, SNRBs and a comparison of postsurgical response. Of the 20 patients studied, 18 had a positive cervical SNRB that corresponded to MRI findings and complete postsurgical pain relief.

Slipman et al. [115] examined the effects of biomechanical stimulation on 87 patients and examined 134 cervical nerve roots. Patients then described to an independent observer the referred symptoms (dynatome) on a pain diagram. That dynatomal map, compared with classic dermatomal maps, showed distribution similar to classic dermatomal maps, but frequently overlapped other dermatomes and had a larger distribution. This study emphasized the potential importance and advantage of a SNRB in evaluating patients with equivocal imaging and radicular pain.

In a 2005 study comparing MRI findings in patients with subacute (mean 4.7 months) cervical or lumbar radicular pain with SNRBs and surgical outcomes [116], the majority of patients did not undergo SNRB prior to surgery; SNRBs were reserved for patients with symptoms refractory to conservative treatment of at least 6 weeks and with discrepancies between presenting examination and radiologic imaging. A positive outcome was defined at ≥95% pain relief following injection of 0.5 to 0.75 cc of 2% lidocaine. A total of 101 patients underwent SNRBs: 91 (90%) were positive and 10 were negative at the level operated. Ninety-one percent of the patients with a positive SNRB had good surgical outcomes, whereas 60% of the patients with a negative SNRB had good outcomes. When findings between SNRB and MRI differed (n = 20), surgery at a level consistent with the SNRB was more strongly associated with a good surgical outcome. SNRBs were superior to MRI in predicting a poor surgical outcome (P = 0.01).

All totaled, patients having surgery on positive SNRB levels were 9.1 times more likely to have good outcomes than those who had surgery on negative SNRB levels (P = 0.01).

A retrospective analysis was done on 100 patients who had received cervical epidural steroid injections for neck pain and cervical radicular pain to identify the predictors of outcome after such treatment [91]. The researchers concluded that patients with radicular rather than axial neck pain benefited the most from CILESIs.

CONCLUSION

Cervical Transforaminal (TFESI) and Interlaminar CILESI are safe and effective, demonstrating both short- and long-term benefits for patients with cervical radicular pain. Further outcome studies are needed for both ILESIs and TFESIs to definitively prove their utility. Predictive outcome studies involving MRI diagnostic imaging, SNRB diagnostic injections, and clinical predictors are accumulating evidence to improve patient outcomes by selecting patients most likely to improve with a given treatment. In addition, many techniques have been developed to increase the accuracy of needle placement and to improve the procedural safety profile including the use of particle-free steroids for TFESIs, the routine use of fluoroscopy, epidurograms, and DSA.

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42 Percutaneous Cervical Discectomy

Kenneth M. Alò, Erich O. Richter, and Jayme Trahan

INTRODUCTION

Over the past two decades, minimally invasive spine surgery for the treatment of cervical pathology has evolved rapidly. Through the development of new instrument technologies and subsequent modification of surgical technique, spine surgeons now have the ability to accomplish the same operative goals through smaller incisions and limited corridors, resulting in less tissue trauma. The goal of these techniques is a decrease in postoperative pain, shorter hospital stays, and quicker recovery periods.

Tajima et al. [1] first described percutaneous endoscopic cervical discectomies for the treatment of cervical radiculopathy in 1989. Since this initial description, multiple modalities have been developed to perform cervical disc decompression to treat cervical radiculopathy. The basis of these modalities revolves around two fundamental techniques—percutaneous puncture techniques and percutaneous endoscopic techniques [2].

Percutaneous puncture techniques use the basic approach described in the following section to access the intervertebral disc space. Once accessed, modalities of treatment include chemonucleolysis [3], nucleoplasty using direct radiofrequency ablation [4], and annuloplasty using direct thermoablation [5]. Because annuloplasty is primarily used for the treatment of discogenic cervicalgia and headache rather than radiculopathy [5], it is outside the scope of this chapter and will only be briefly mentioned here.

Percutaneous endoscopic discectomies use the same percutaneous approach; however, through the insertion of an endoscope, the surgeon can obtain direct visualization of the intervertebral disc space to perform a discectomy. Treatment modalities using the endoscope include direct manual removal via forceps [6] and thermodiscoplasty using laser ablation [5–10].

TECHNOLOGY

Chemonucleolysis

Chemonucleolysis is based on the digestive properties of the enzyme chymopapain. It is believed that chymopapain (Discase, Travenol) degrades the proteoglycan content of the intervertebral disc, resulting in loss of water and glycosoaminoglycan. The result is loss of disc [11,12]. Computed tomography (CT) studies at 6 months show a reduction in the disc protrusion by 3 mm and an increase of thecal cross sectional area between 9 and 40 mm^2 (mean: 23 mm^2) [11].

Nucleoplasty

Nucleoplasty procedures are based on the technology of plasma radiofrequency ablation through the use of bipolar electrodes. These electrodes, on the distal end of the device, produce a strong electric field when current is passed through them. This field disrupts the molecular bonds in adjacent tissue resulting in conversion of the nucleus pulposus into liquid and gaseous products, which are resorbed from the target site or escape via the introducer needle, respectively [13].

Endoscope and Laser Technology

With the addition of a working port to the endoscope, surgeons were given the ability to use microforceps in conjunction with laser technology to enhance their ability to perform adequate discectomy to treat cervical radiculopathy [14]. Other notable improvements included a laterally directed port that allow the surgeon to safely work in the epidural space to remove foraminal disc fragments.

Different laser technologies exist to give the surgeon options when performing percutaneous cervical discectomies. The holmium/yttrium-aluminum-garnet (Ho:YAG) laser penetrates the tissue less than 0.5 mm, whereas the neodymium-doped yttrium aluminium garnet (Nd:YAG) laser penetrates to a depth of 2 mm. Therefore, some consider the Ho:YAG laser safer than Nd:YAG laser [5]. Recently, a 2.0 μm thulium-doped laser has been introduced (Figure 42.1) [15]. This near infrared laser system has several advantages, including minimal tissue penetration (500 μm), very focused tissue disruption due to a continuous emission mode, and because of these features, delivery of the energy only to a small volume in front of the fiber tip. Little clinical data is yet reported for this device, but based on previous experience with the earlier generation lasers, these features are expected to lead to increased efficacy and decreased complications.

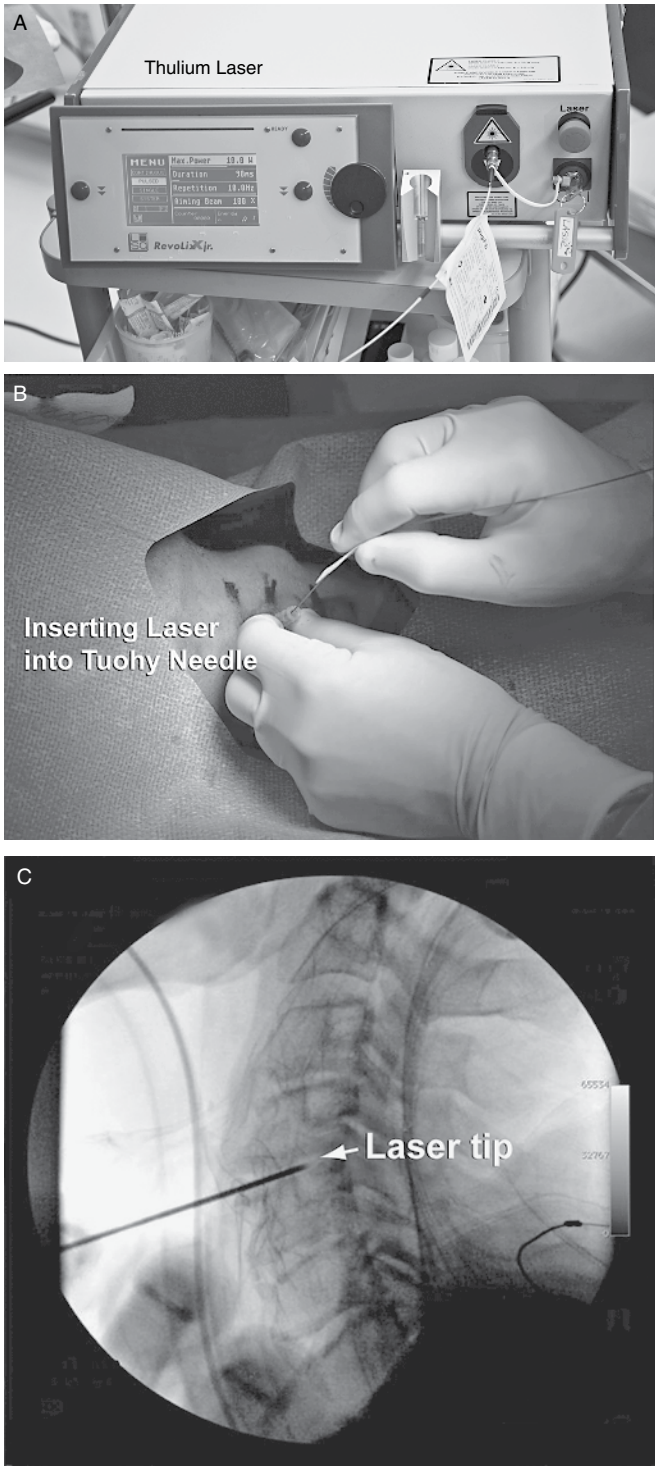

Figure 42.1 **(A)** Thulium laser. **(B)** Fiber optic for laser energy delivery introduced into the disc space through the Tuohy needle. **(C)** Intraoperative x-ray. The laser tip is demonstrated in the disc space by fluoroscopy.

PREOPERATIVE EVALUATION

Before undergoing consideration for operative decompression, extensive conservative management should be carried out including at least 6 weeks of physical therapy and medical treatment with anti-inflammatory medications and muscle relaxants. Radiographic structural workup should include both CT and magnetic resonance imaging (MRI) modalities. Diagnostic blockade may also add significant functional information regarding pain symptoms [16]. If a patient fails to improve with adequate conservative medical management, they should be evaluated for the various operative interventions that are appropriate.

Percutaneous Puncture Discectomy

Indications

Criteria included a radiographically confirmed contained cervical disc herniation with functional radicular complaints and no improvement with conservative management [4].

Contraindications

Relative exclusion criteria include an extruded disc fragment into the epidural space, hemorrhagic diathesis, spondylolisthesis, ossification of the posterior longitudinal ligament, previous surgery at the identified level, severe central canal stenosis, and the presence of myelopathy on examination [4].

Percutaneous Endoscopic Discectomy

Indications

Indications for cervical decompression through a percutaneous endoscopic approach include the presence of soft disc herniation not contained by the posterior longitudinal ligament, either central, lateral, or foraminal in location, and the presence of radicular complaints that did not improve with conservative management [5].

Contraindications

Endoscopic percutaneous discectomy procedures are contraindicated in patients with severe neurologic deficits, signs of myelopathy on exam, structural instability at that joint segment, or etiologies of neural compression other than chronic degeneration. Anatomical contraindications include migrated or calcified discs, ossification of the posterior longitudinal ligament, severe central canal stenosis, and severe disc space narrowing of 3 mm or less [5].

Endoscopic and percutaneous approaches appear to be appealing because they involve less tissue trauma and lower postoperative morbidity. Although the choice of surgical approach is based on surgeon's preference, cases with significant nerve root compression may require "standard" open approach in order to create more space for fine manipulation of nerve root and epidural vessels.

PROCEDURAL TECHNIQUE

Access to the Anterior Cervical Spine

After informed consent is obtained, the patient is brought to the operating room, placed supine on the operative table, and appropriately padded with special attention to place a

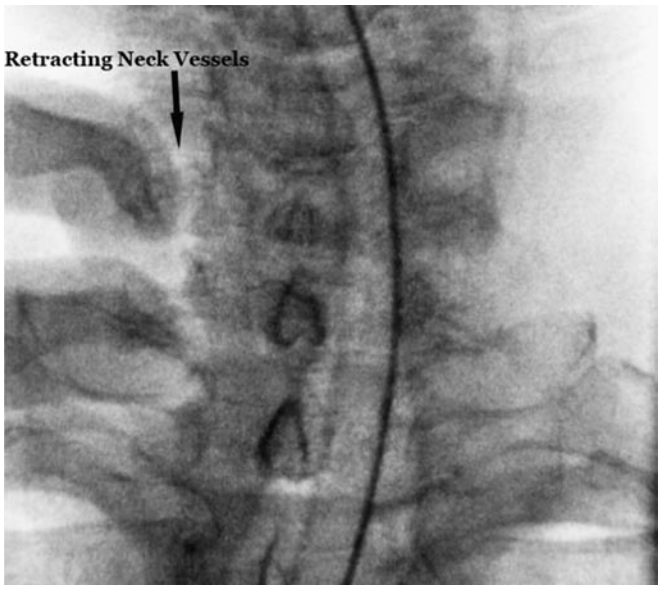

Figure 42.2 Retraction of the neck vessels during the anterior cervical approach.

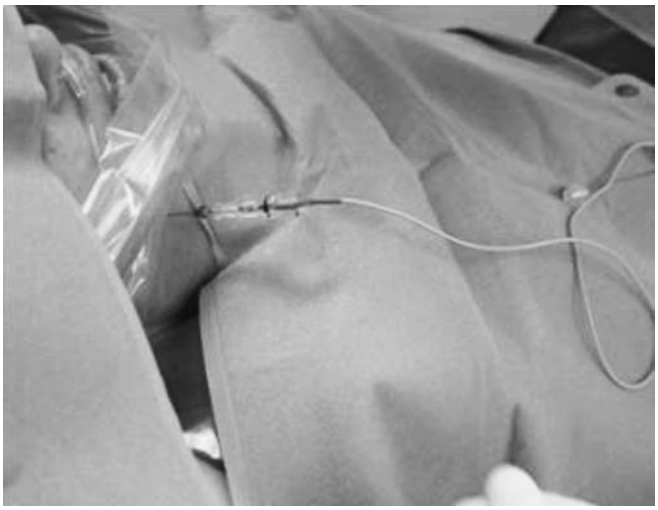

Figure 42.3 Percutaneous approach. Under fluoroscopic guidance, a needle is inserted into the disc space along the medial border of the sternocleidomastoid muscle.

shoulder roll to allow for slight extension of the neck. Also, a soft strap may be used over the forehead for stabilization. Fluoroscopy is used to identify the operative level in the anteroposterior and lateral planes. A right-sided approach is preferred for an intracanalicular disc and a paramedian approach is used for a contralateral foraminal disc herniation [5]. Ahn et al. choose contralateral side in case of a lateral disc herniation, which offers a better visualization of the foraminal portion and allows for easier removal of fragments [14]. Local anesthetic is used at the point of entry, which is usually the medial border of the sternocleidomastoid (SCM).

To begin the procedure, firm pressure is applied to the medial border of the SCM and the lateral border of the trachea in the direction of the anterior vertebral column. At this point, the trachea and esophagus are displaced medially and the carotid, laterally (Figure 42.2). The anterior cervical spine should be palpable. Under fluoroscopic guidance, an 18-gauge spinal needle is inserted through the skin along the medial border of the SCM between the airway and the pulsation of the carotid artery with a trajectory aiming at the center of the disc space [5] (Figure 42.3). Once the disc space is entered and confirmed by fluoroscopy, intraoperative discography can be performed to visualize the presence of annular tears by observing leakage of contrast into the epidural space (Figure 42.4).

Percutaneous Puncture Discectomy

Chemonucleolysis

As described by Hoogland et al. [3], once the desired disc space is entered and confirmed using fluoroscopy, 500 IU chymopapain is slowly injected into the disc space and allowed to sit for 10 minutes. A fine Kirschner wire is then introduced through the spinal needle, and the spinal needle is subsequently withdrawn. Next, a 2-mm nucleotome is introduced over the Kirschner wire, into the disc space. Once the Kirschner wire is withdrawn, the nucleotome can be used to remove the digested nucleus pulposus. Once complete, instrumentation is removed.

Nucleoplasty

Once the desired disc space is entered and confirmed using fluoroscopy, the radiofrequency ablation Perc-D spine wand (Arthrocare, Sunnyvale, CA) is inserted through the 18-gauge needle into the disc space (Figure 42.5). The ablation device is then connected to the Arthrocare power generator and the ablation process is begun. Various generator settings are described in the literature [4,17]. The basic technique involves 360° rotation of the wand in two planes within the disc space; the chosen planes are dictated by the topographic location of the herniation. Small amounts of nucleus pulposus are removed creating two channels of decompression [17]. When the discectomy is complete, the instrumentation is removed from the disc space and the site is bandaged.

Percutaneous Endoscopic Discectomy

Once the desired disc space is entered and confirmed using fluoroscopy, a guide wire is passed into the disc space followed by removal of the 18-gauge needle over the guide wire. Sequential dilators are then passed over the guide wire to create a working channel. Once the working channel is in place and the dilators have been withdrawn, an endoscope may be inserted into the disc space for direct visualization. Next, microforceps can be inserted to remove any disc fragments identified under direct endoscopic visualization. Following discectomy, various authors have described using laser technology to ablate remaining fragments. Anh et al. [6] described using a Ho:YAG side-firing laser to vaporize residual fragments and annular tears. This laser can also be used

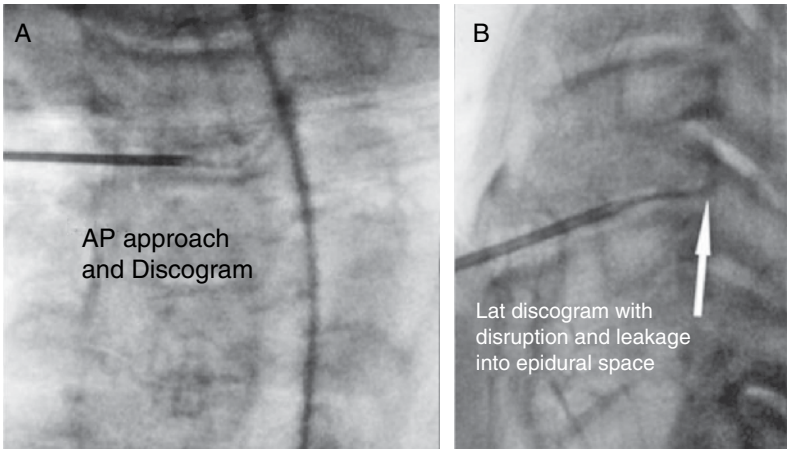

Figure 42.4 Intraoperative discography. In this discogram, contrast material leaks into the epidural space indicating an annular tear. **(A)** AP view. **(B)** Lateral view.

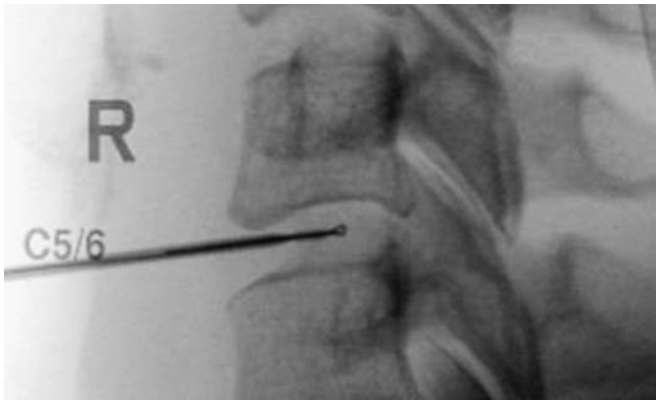

Figure 42.5 Intraoperative x-ray of radiofrequency ablation. The Perc-D spine wand is inserted into the disc space through the 18-gauge needle.

to vaporize posterior osteophytes or any disc fragments within the adjacent foramen decompressing the exiting nerve root. The endoscope can then be used to verify adequate decompression of the foramen. The endoscope and working channel are then withdrawn from the neck for conclusion of the procedure. Gentle pressure is held to the operative site to stop any bleeding.

OUTCOMES

Although trends in spinal surgery have become centered on philosophies of minimization, many surgeons have been weary to adopt this approach when addressing pathology of the anterior cervical spine, due largely in part to the proven success rates of anterior cervical discectomy and fusion (ACDF) procedures.

Percutaneous Puncture Techniques

Chemonucleolysis

Since its first description in 1963 by Smith et al. [18], enzyme dissolution of the nucleus pulposus has been primarily used to treat lumbar radiculopathy. Gomez-Castresana first reported its use in the cervical spine in 1992 [19]. Within this series of 50 patients, initial success rates reached 90% with this new treatment modality. Hoogland et al. [3] shortly later modified this approach by performing chemonucleolysis with percutaneous nucleotomy on 22 patients. Eighty-six percent of patients reported good to excellent scores on MacNab scale (Table 42.1) at 2.5-year follow-up.

Nucleoplasty

Multiple trials have looked at percutaneous cervical nucleoplasty for treatment of cervical radiculopathy. Overall, patients have rated their postoperative pain as good-to-excellent using the MacNab criteria 78% to 85% at their follow-up appointment [4,17,20,21]. A notable study by Li et al. [4] looked at 126 patients undergoing percutaneous cervical nucleoplasty for treatment of cervical radiculopathy caused by disc herniation. Postoperatively, 84% of patients rated their outcome as excellent to good on the MacNab grading system. Visual Analogue Scores (VAS) decreased from an average of 7.2 preoperatively to a postoperative score of 2.4.

Percutaneous Endoscopic Techniques

As previously noted, there are two types of lasers used to perform percutaneous discectomies, each with similar patient outcomes. Within the studies in which the surgeon used the Ho:YAG laser, 86% to 95% of patients rated their postoperative pain scores as good-to-excellent [6,7], with preoperative pain scores on average of 7.9 decreasing to a postoperative average of 2.6 on the VAS [5]. Authors using the Nd:YAG laser have produced similar outcomes within their patient populations. 75% to 90% of patients undergoing discectomies with this laser achieved resolution of their preoperative radiculopathy [8,9]. With the thulium laser, the therapeutic goals were achieved in 93% after initial operation and in 98% after a second neuroendoscopic operation [15].

Although percutaneous discectomies offer a minimally invasive alternative to treating radicular pain caused by disc herniation, there is an inherent risk of inadequate

Table 42.1 Clinical Outcome According to Modified MacNab Criteria

Result	Criteria
Excellent	No pain; no restriction of mobility; return to normal work and level of activity
Good	Occasional nonradicular pain; relief of presenting symptoms; return to modified work
Fair	Some improved functional capacity; still disabled and unemployed
Poor	Continued objective symptoms of root involvement; additional operative intervention needed index level irrespective of length of postoperative follow-up

decompression resulting in continued symptomotology and possibly requiring additional open cervical discectomy and fusion. Various studies have used the MacNab grading scale to access postoperative outcomes. 5% to 14% of patients rated their postoperative outcome as fair, having improved functional capacity despite persistent pain. Approximately 10% of patients undergoing percutaneous cervical discectomies rate their outcome as poor, with continued symptomatology and additional intervention needed at the index level [2,5].

Predictive Factors for Positive Outcome

Previous authors have evaluated patient characteristics that offer a predictive value for long-term excellent results following percutaneous endoscopic cervical discectomy. What was concluded was that the preoperative factors that showed positive correlation with a successful outcome were radiating arm pain and the presence of a lateral disc herniation into the foramen [5,6,14]. These factors are inherent to the underlying pathology and symptomatology, and removal of subsequent soft disc should produce symptomatic relief. Conversely, patients who have complaints of axial pain and vague numbness have not been found to have positive outcomes with percutaneous discectomies because the underlying pathology is usually disc degeneration and hard compression [5,6,14].

Complications

Although percutaneous discectomy procedures aim to avoid the morbidities associated with standard open ACDFs, such as postoperative dysphasia, epidural bleeding, graft-related problems, and hoarseness, there is still inherent risk for complication related to this procedure [5,14,22–24]. In a direct comparison of these two treatment modalities used to alleviate cervical radicular pain, complication rates are both similar and low. When accessing the risk of ACDF procedures, it is generally accepted that the risk of complication is at 3% [25]. Within the literature accessing the complication rate of percutaneous discectomy procedures, complication rates have been comparable ranging from 1% to 3% in the larger series [4,9,21,26].

Potential risks associated with this procedure can be categorized as injury to surrounding non-neural structures including carotid vessels, trachea, hypopharynx, and esophagus, negative impact on biomechanical stability, and injury to underlying neural structures. The potential also exists for postoperative infection, and hematoma formation [5,6,22,24,27].

Within this literature review, one neck hematoma was documented during a percutaneous discectomy via the use of a nucleotome [26]. Evaluation noted injury to the inferior thyroid artery. Other complications noted involved the breakage of the distal component of the Perc-D Wand within the disc space. The component was unable to be retrieved in both instances; however, no clinical consequence was noted in either case. [4,17]. Of note, neural structure injury did not occur in any series within the literature reviewed on this topic.

Complication Avoidance

In an effort to define the safety zone when performing percutaneous cervical approaches, Lee et al. [28] studied the CT scans of 30 patients when manipulating the SCM, trachea, and esophagus in the same technique described earlier. What they found was that the safety zone for anterior approaches is much narrower in the upper cervical spine compared to that of the lower cervical spine. This is due in part to more medial trajectory of the carotid vessels higher in the cervical spine with the vessel lying along the medial border of the SCM, where as conversely, the carotid is well underneath the SCM more inferiorly. Also noted in this study was that the superior thyroid artery is found in the safe zone at the C3-4 level in 86.7% of patients, which increases the risk of bleeding when approaching this level for discectomy. To lessen the risk associated with this level, one could use intraoperative ultrasound to avoid injury to the superior thyroid artery (STA). Also noted in this study was the potential for injury to the thyroid gland. It was found to be present in the safe zone at the level of C5-6 and C6-7 in 76.6% and 90%, respectively. However, at this time there is no clinical evidence to support that injury to the thyroid gland would have clinical significance.

Percutaneous removal of disc material, without intervertebral graft placement and anterior instrumentation, potentially raises concern over altered spine biomechanics. Li et al. [29] looked at the influence of surgical treatment for disc degeneration specifically at the C5-6 levels as it relates to biomechanical parameters. Multiple treatment modalities were studied including, but not limited to, ACDF procedures and percutaneous nucleotomy procedures performed on cadaveric specimens with a control of a nonoperative cadaveric specimen. Various physiologic loads were applied to each spine during which biomechanical properties were measured. What was found was that markedly fewer changes were observed in the spine that underwent percutaneous nucleotomy when compared to the baseline intact spine. This study supported the trend of evolving clinical practice of using minimally invasive decompressions to treat soft cervical disc herniations.

CONCLUSION

Because of the recent advancements in surgical technology, instrumentation, and modification of technique, the

treatment of cervical radiculopathy secondary to a herniated disc can now be accomplished through a minimally invasive percutaneous approach. This allows a surgeon to safely and effectively treat the underlying disorder, while causing minimal postoperative morbidity compared to standard open procedures.

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43 Neuromodulation for Cervical Radicular Pain

Timothy Deer, Jonathan Carlson, and Patrick W. Hogan

CLINICAL PRESENTATION

Cervical radiculopathy is defined by an objective loss of sensory and/or motor function as a result of conduction block or axon loss in a spinal nerve or its roots. Symptoms include numbness and weakness in the distribution of the affected nerve. Neurologic examination and diagnostic tests such as electromyography can help confirm the neurologic abnormality. Magnetic resonance imaging or computed tomography with myelogram should also be ordered to assess pathology and anatomic variations. It is important to underscore that radicular pain and radiculopathy are not synonymous. The former is caused by ectopic impulse generation of nociceptive afferent fibers, whereas the latter relates to a conduction block, which is manifested by actual physical examination signs [1]. Cervical radiculopathy has neurologic components of paresthesias, numbness, hyporeflexia, and weakness [2]. Patient description is usually sharp, shooting, lacinating or shock-like pain that may affect the neck, shoulder girdle, anterior chest wall, arm, forearm, and hand. The pain can be exacerbated by coughing, sneezing, neck extension, and a Spurling test [3]. Common pain and neurologic presentations of cervical radiculopathy with the respective affected nerve root include; C5 with sensory deficits / areas of numbness and parathesia in the deltoid, weakness in the deltoid and biceps, and pain in the neck, shoulder and anterloateral arm; C6 with sensory deficits / areas of numbness and parathesia in the thumb and lateral hand, weakness in the biceps and wrist extensors, and pain in the neck shoulder and lateral arm; C7 with sensory deficits / areas of numbness and parathesia in the index and middle finger and dorsum of the hand, weakness in the triceps, wrist flexors and finger extensors, and pain in the neck, shoulders, lateral arm, and dorsum of the forearm; C8 with sensory deficits / areas of numbness and parathesia in the ring and fifth finger, medial hand and forearm with weakness in the finger flexors and interossei muscles, with pain in the shoulder, lateral arm and hand [4,5]. The most common spine-related etiologies for cervical radiculopathy are herniation of the nucleus pulposus, central spinal and foraminal stenosis, and cervical spondylosis with associated osteophyte formation [6,7].

TREATMENT

After gathering sufficient clinical data to exclude the presence of malignancy, infectious, or congenital pathology, the provider must then determine if the radiculopathy has associated myelopathy which may necessitate surgical referral.

If the patient's pathology does not require immediate surgical correction, then conservative treatment measures are typically implemented to manage the symptoms. If the patient's main symptom is pain or numbness with no neurologic deficits, many spine surgeons will recommend conservative measures prior to surgical intervention. A multidisciplinary approach for conservative treatment has been shown to have the best results when treating radicular pain [8]. Treatment modalities such as physical therapy, pharmacologic (nonsteroidal anti-inflammatory drugs, neuropathic pain medication, analgesic medications), chiropractic care, and epidural steroid injections/selective nerve root blocks are usually implemented. If these treatments fail, then frequently the patient faces a decision between surgery or continued conservative management. However, a growing trend is emerging wherein some clinicians are recommending their patients try neuromodulation as a less-invasive and possibly more cost-effective alternative to conventional spine surgery [9]. This option may be particularly valuable in those patients who do not meet the criteria for good outcomes with spine surgery.

Occasionally, those patients who fail conservative management and undergo traditional spine surgery will continue to have pain symptoms postoperatively. These patients are often diagnosed with "failed neck surgery syndrome" or cervical postlaminectomy syndrome. Failed neck surgery syndrome is the most common indication for cervical spinal cord stimulation (SCS) in the United States.

BRIEF TIMELINE OF NEUROMODULATION

Norman Shealey performed the first SCS implant in 1967. During the next decade, the procedure was performed mostly by neurosurgeons who implanted subdural electrodes directly onto the spinal cord. Secondary to

significant complications such as spinal cord injury and cerebrospinal fluid leak, the trend progressed toward epidural placement of the electrodes. These operations were typically "open" procedures which required laminotomy or laminectomy to place the electrodes into the epidural space.

In 1974, Dr Dooley implanted the first *percutaneous* SCS electrodes using an epidural introducer needle. SCS for treatment of cervical neuropathic pain began shortly thereafter using both surgically and percutaneously placed leads. Hosobuchi reported the use of cervical lead placement to improve blood flow to the cerebral vessels [10]. The next area of documented benefit was placing leads in the lower cervical spine to improve complex angina that had failed other treatments [11]. Work in the cervical spine to treat cervical radiculopathy was noted in studies that included an eclectic number of patients by Kumar, North, and Meglio [12–14]. Over time, the improvements in technology, patient selection, programming, and more efficient rechargeable batteries led to more candidates for cervical SCS.

MECHANISM OF ACTION

The gate control theory of pain, published in 1965 by Ronald Melzack, a pain psychologist, and Patrick Wall, a neurophysiologist, provided a scientific basis for the use of electrical stimulation to treat pain by proposing that a "gate" regulates transmission of pain sensations from the dorsal horn in the spinal cord to the brain. This gate opens when small-fiber (C fibers and A-delta) afferents are unusually active and closes when large-fiber (A-beta) activity is dominant [15]. This theory can explain how rubbing injured skin may reduce pain, or how a transcutaneous electrical nerve stimulator unit can reduce pain by distributing pleasant electrical sensations through electrodes on the surface of the skin. Selective depolarization via neuromodulation of large-fiber afferents in the dorsal columns of the spinal cord theoretically "closes the gate" for pain transmission without causing undesired motor stimulation. This model may be an oversimplification of the true mechanism, and other mechanisms of action have been proposed. In reality, the precise mechanism of action remains undefined.

INDICATIONS

An implantable neuromodulation system is indicated for SCS as an aid in the management of chronic, intractable pain of the trunk and/or limbs. Cervical radiculopathy pain can be unilateral or bilateral and can be associated with the following conditions:

- Failed neck surgery syndrome
- Radicular pain syndrome or radiculopathies resulting in pain secondary to failed neck surgery syndrome or herniated disk
- Cervical postlaminectomy pain
- Multiple back operations
- Unsuccessful disk surgery
- Degenerative disk disease/herniated disk pain refractory to conservative and surgical interventions
- Epidural fibrosis
- Inoperable stenosis

CONTRAINDICATIONS

- Uncorrected coagulopathies
- Current sepsis/infection with fever
- Implantable cardiac defibrillator
- Inability to control device or lack of patient cooperation
- Cervical syrinx

Relative Contraindications

- Cervical stenosis [if < 10 mm for a percutaneous lead [16]]
- Patients who may require serial magnetic resonance imaging evaluations (e.g., multiple sclerosis)
- Demand cardiac pacemaker

PATIENT SELECTION

In the early development of SCS, patient selection was less than ideal. Because of the relatively new nature of the technology, there was limited evidence in the literature from which to base selection criteria. However, in the past 20 years, there have been improvements in defining proper selection to improve outcomes.

The decision to proceed with SCS typically occurs after failure of more conservative therapies and involves consideration of both patient-related factors and the characteristics of the pain itself. The practitioner should ensure that reasonable and less-invasive treatment modalities (injections, medication management, physical therapy, etc.) have failed. It is also important to consider available surgical options or that the patient has declined available surgical intervention. To maintain this modality as an option for future patients, maximizing cost-effectiveness by following a conservative approach may be prudent.

ETIOLOGY OF THE PAIN AND THE LIKELIHOOD OF PAIN REDUCTION

Regarding the characteristics of the pain, the best outcomes have been observed in patients with steady, lancinating, and burning neuropathic pain. Further, patients with unilateral radicular pain in an extremity have demonstrated improved outcomes. However, with the advent of new technology such as dual and tripolar lead configurations, there have been great strides in outcomes for patients with axial pain, bilateral radicular pain, and even pain relief from secondary to wider variety of pain syndromes.

A recent article by Deer and Masone discusses those disease characteristics that predict good outcomes in SCS patients. Those patients with a *high probability* of successful pain reduction include [16] the following:

- Chronic cervical or lumbar radicular pain syndromes
- Complex regional pain syndrome, types 1 and 2

- Painful peripheral mononeuropathies
- Angina pectoris refractory to conventional surgical bypass and medical management
- Painful ischemic vascular disease refractory to medical management or surgical intervention

There is *moderate possibility* of successful pain reduction in patients with the following:

- Axial pain of the cervical or lumbar region (these indications may require more complex multicolumn paddle leads or the use of combined epidural and peripheral nerve leads)
- Pelvic pain
- Visceral pain syndromes of the abdomen
- Postherpetic neuralgia

There is *low probability* of successful pain reduction in patients with the following:

- Neuropathic pain following spinal cord or brain injuries, nerve root avulsions
- Iatrogenic nerve root destruction
- Phantom limb pain

In the United States, the most common patient population who experience benefit from SCS are patients with "failed back surgery syndrome." These are patients who underwent attempted surgical correction for a disorder of the spine and suffer from persistent pain in an extremity, their axial spine, or both.

Assuming that the patient's pain complaints fit into a category wherein there is likelihood of successful pain reduction, the next step in decision-making is to consider factors unique to the patient.

PATIENT FACTORS

The patient's overall mental and physical health are important considerations in successful outcomes with SCS.

Regarding the patient's physical health, it is prudent to consider their preoperative risk for general medical complications, infection (diabetics, immunocompromised patients, patients with systemic or local infections), bleeding (genetic coagulation abnormalities, chronic oral anticoagulant therapy), and also to assess whether they have a prohibitive degree of spinal stenosis or a syrinx. Many practitioners avoid introducing SCS leads into the epidural space of patients who suffer from a significant degree of spinal stenosis. However, if there is severe spinal stenosis, a surgical paddle-type lead could be placed following a decompressive laminectomy.

Assuming the patient's physical health is conducive to SCS, the practitioner must then consider the mental health of his or her patient.

First, the practitioner must assess the patient's overall baseline cognitive function. Patients with a low level of cognitive function may be unable to comprehend aspects of the technology such as recharging their device generator, and using their handheld programmer.

After ensuring adequate physical health and cognitive ability, the patient is usually sent for a psychology screening examination. The psychology screening evaluation is required by a majority of third-party payers in the United States, and the goal is to identify psychiatric abnormalities that would predict a poor outcome.

A typical psychology screening involves assessment by a licensed psychologist including psychological interview (pain history, medication review, pain descriptors, psychosocial history, behavioral observations, and mental status examination), possible interview of a family member, spouse, or close friend, and key psychological screening tests (Minnesota Multiphasic Personality Inventory or MMPI, McGill Pain Questionnaire, etc.).

Patient expectations should also be addressed prior to undertaking the SCS trial. The goal of the trial and implant is for pain reduction (typically at least 50%) not necessarily "curing" their pain condition. If patients are prepared adequately for what to expect in the trial, and what constitutes a successful trial, there may be improved outcomes and patient satisfaction.

Psychiatric comorbidities (acute psychosis, personality disorders, severe depression/anxiety), significant drug or alcohol addiction, and issues of secondary gain should be addressed. The patient's baseline beliefs, attitudes, and expectations for the modality will likely play a role in the success of SCS.

Poor treatment outcomes have been correlated with the most psychologically and physically distressed patients, as well as those with depression and catastrophizing attitudes [17].

Patients with severe underlying psychiatric disorders may not be able to differentiate their pain from their anxiety, depression, or other pathology. A study by Burchiel et al. demonstrated that measures of depression (via the MMPI), perception of pain intensity (McGill Pain Questionnaire), and advanced age predicted the patient's pain status 3 months following SCS implant [18].

A study by North demonstrated that low scores on anxiety and high organic symptoms scores (per Derogatis Affects Balance Scale and Wiggins scales of the MMPI) predicted success of an SCS trial leading to permanent implant. These same predictors for pain relief from the SCS trial did not hold true at 3 months after SCS implant. The author acknowledged that no psychological predictors were identified for long-term success with SCS implant; however, additional studies were underway [19].

After establishing appropriate indication and patient selection, it is reasonable to proceed with a SCS trial. In the authors' opinion, one of the key benefits to SCS in the treatment of chronic pain is the temporary test trial. The test trial affords the patient the opportunity to experience the stimulation over the course of 3 to 7 days, wherein they determine if their pain is alleviated by 50%, and if so the option for permanent implant is available.

The typical outpatient SCS trial involves percutaneous placement, using a 14-gauge epidural introducer needle, of one or two SCS leads into the desired position in the epidural space. During the procedure, a test stimulation and programming is performed to confirm "coverage" of the patient's painful areas.

During the procedure, the patient is typically given a light intravenous anesthetic so that they are comfortable, but able to communicate adequately during the intraoperative programming phase.

After adequate coverage of their painful areas is established, the introducer needle is carefully removed and the leads are anchored with tape and/or suture material. Finally, prior to discharge home, the patient is then transported to a postprocedure recovery area and another programming session is undertaken to confirm adequate coverage of the painful areas.

The patient is then discharged home, often with oral antibiotic prophylaxis, and returns in 3 to 5 days for removal of the leads. The authors try to avoid making any changes in the patients' medication regimens during the time immediately preceding the trial and during the trial period itself to minimize any confusion.

PROCEDURAL TECHNIQUES FOR PERCUTANEOUS SCS LEAD PLACEMENT

First, written and verbal informed consent must be obtained following discussion of the risk, alternatives, and benefits of the procedure. Preoperative antibiotics, 30 minutes prior to incision, is recommended prior to the SCS trial, and is considered standard of care prior to the permanent implantation [20].

Next, careful attention to patient positioning is key. One must ensure proper padding of the patient's extremities to avoid iatrogenic nerve injury, and the arms should be at the patients' side. Failure to place the patients' arms at their sides may lead to difficulty seeing the electrode on lateral view. The patients should also have adequate padding beneath their upper chest to facilitate forward flexion of the patient's neck. The forehead should be stabilized to avoid intraoperative movement, and proper oxygen delivery should be ensured. The positioning should allow for optimal opening of the lower cervical/upper thoracic interspaces to facilitate entering the epidural space, and should approve patient satisfaction. Visualization of the desired interspace can be further enhanced by a slight caudal tilt of the C-arm.

When considering the type and level of anesthesia to administer, it is important to note that for the SCS trial and permanent implant, a minimal amount of anesthesia should be administered until the leads are properly placed. The patient must have an adequate level of consciousness to indicate whether the intraoperative neuromodulation paresthesias "cover" their painful areas, and it not, the leads must be repositioned. The authors recommend that the sedation be minimized to the point that the patient is able to engage in meaningful conversation with the proceduralist.

After proper positioning, meticulous sterile technique must be used. Some providers suggest that their patients undergo a chlorhexidine shower at home, the evening before surgery. At a minimum, the patient should be prepared and draped in the usual sterile fashion with povidone-iodine or chlorhexidine (some providers also advocate iodophor impregnated adhesive drapes). Many providers also employ the use of a surgical mask, cap, and gown.

Fluoroscopic guidance is utilized throughout the procedure, particularly during lead placement. The typical fluoroscopy views are anteroposterior and lateral views. Some experts advocate entering the epidural space below the T1-T2 interspace. Lirk et al. demonstrated in 52 cadavers that there was failure in ligamentum flavum fusion that varied based on the spinal level. The author noted that at the C7-T1 level, and higher, the failure rate was 51% to 74%, but the T1-T2 level only had a 21% failure rate. The significance of this failure of fusion is that the loss of resistance (LOR) which occurs, as the ligament is traversed, is an important safety margin for needle depth and avoidance of spinal cord injury [21].

Once the entry level is determined, the skin is typically anesthetized with local anesthetic, and the authors use 1% lidocaine mixed with sodium bicarbonate and epinephrine. The sodium bicarbonate that is added should be approximately 10% of the total volume (i.e., 1 mL of sodium bicarbonate in with 9 mL of lidocaine). The sodium bicarbonate hastens the onset of topical analgesia and decreases the burning sensation of the local anesthetic. The use of epinephrine optimizes vasoconstriction, thus theoretically decreasing the risk of bleeding and hematoma formation. A 22-guage 3.5-inch spinal needle can be used to anesthetize the deeper tissue to the laminae, thus decreasing the risk of bleeding and improving patient comfort. Some specialists, however, do not advocate this because of the risk of intrathecal injection.

A 14-gauge modified-Tuohy or RX-Coude needle can be used to enter the epidural space with a LOR technique. The question of whether air or preservative-free normal saline should be used to confirm entrance into the epidural space remains a decision based on clinical judgment. The current literature has shown either no significant difference or slightly improved safety with saline in numerous literature reviews for labor epidurals [22,23]. The superior LOR technique has not been extensively studied in accessing the epidural space for SCS lead placement; however, some experts in pain medicine feel that the LOR with air is superior because there is less risk of current disbursement in the epidural space, thus theoretically improving the probability of a successful trial. The presence of saline or air in the cervical epidural space has never been shown to make a clinical difference in any significant clinical setting.

The needle entry point at the skin is usually entered one and one-half to two interspaces below the epidural entry interspace, medial to the pedicle (Figure 43.1). The needle angle should not be greater than 30º from the skin. While using fluoroscopy in anteroposterior and lateral views, it is safest to contact laminae to gauge the needle depth prior to accessing the epidural space with the LOR technique. Some experts advocate entering the epidural space with the needle bevel down, thus decreasing the risk of dural puncture and then turning the bevel up once the epidural space is entered. This has not been extensively studied, and these suggestions are anecdotal in origin (Figures 43.2 and 43.3).

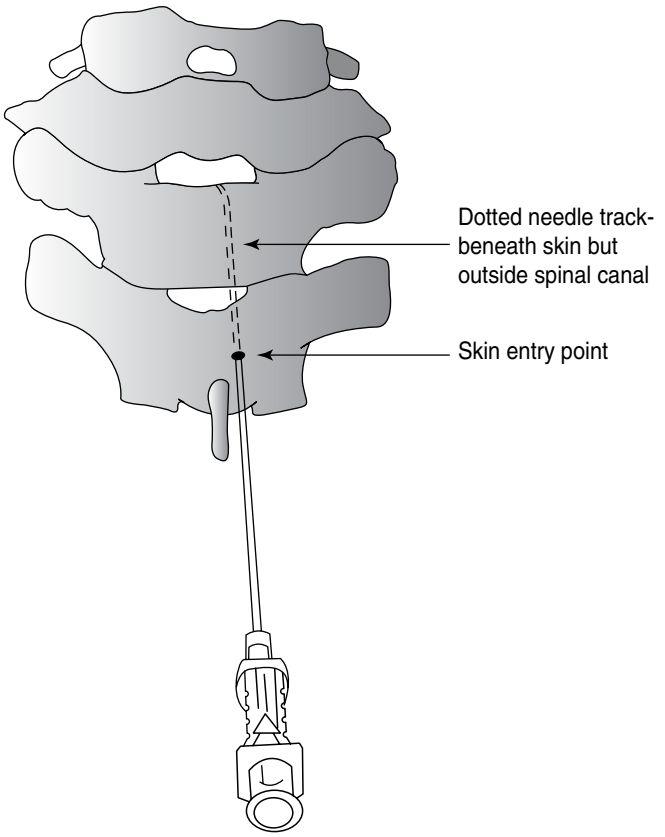

Figure 43.1 Needle entry point one and half interspace levels below the entry interspace. Courtesy of Epimed International. With permission from G. Racz.

Placement of the leads should be done with fluoroscopic guidance (Figures 43.4, 43.5). Neuromodulation should then be implemented and the leads placed in accordance with the patient's painful areas. The use of a single-lead versus a dual-lead array is at the discretion of the practitioner. A single-lead array was shown to help both radicular pain and axial complaints in a few studies [24]. However, the technological advancements involving the use of dual, four-contact and dual, eight-contact leads have demonstrated better efficacy in relieving significant portions of the neuropathic axial component. Most of the work on axial pain has been reported in the lumbar and thoracic regions, but the chance of success in the cervical axial region can be enhanced by newer leads and by considering combined techniques of epidural and peripheral nerve leads. More studies are needed to clarify the best route to cover neck and limb pain [25]. If the decision is made to use dual leads, entry of the second needle on the contralateral versus ipsilateral side is at the discretion of the specialist, as the superiority of either technique has not been established. A study by Barolat et al., Table 43.1, has mapped out sensory responses based on the level stimulation of the dorsal column via the epidural space [26].

The discussion of detailed neuromodulation programming technique extends beyond the scope of this chapter; however, it is recommended that lead location and programming be optimized at the time of the procedure. Once this is achieved, the leads should be securely anchored to prevent migration. If the percutaneous leads are placed for a SCS trial, the trial should not extend beyond 3 to 7 days to minimize the risk of infection and epidural adhesion formation. Last, fluoroscopic images with multiple planes should be stored to best duplicate lead placement for a permanent implant if the trial is considered clinical successful.

COMPLICATIONS

All invasive surgical and interventional pain management techniques involve risks and potential complications which must always be weighed against the potential benefits of the treatment. Regarding SCS trial and implantation, the complications can include infection, bleeding, spinal cord injury, nerve injury, lead fracture, and lead migration.

The most common complication in the literature is lead migration, which can result in a change in the stimulation pattern and decreased analgesia. In a literature review by Monroe et al., the overall incidence of lead migration was 13.5%. Their analysis included 67 articles published since 1981 that reported on 4634 patients. Lead breakage, which occurred in 7.6% of the patients, was the second most common complication. This analysis did not break down the numbers based on lead position. In theory, cervical has a higher migration risk, but this has never been shown in any prospective comparative studies [27].

Internal manufacturers data has shown results of a meta-analysis indicating that lead migration occurred in 5.7% of 300 patients who received the company's non-reprogrammable internal programmable generators. They also noted that the incidence of lead migration may be decreasing over time secondary to improved anchoring devices and techniques. These complications can be minimized by improved techniques in anchoring and tunneling. First, some experts advocate firmly securing the lead to the paraspinous fascia, whereas others anchor the lead in the subcutaneous tissue. It is also important to leave a tension loop in the anchoring site and generator pocket to allow for decreased tension on the lead during natural patient movement. Lead migration can also be reduced by educating the patient on the postprocedure activity restrictions such as prohibiting patients from bending and twisting for 4 weeks, and considering the use of soft cervical collars until the leads heal into place. The incidence of lead fracture can also be reduced by avoidance of the midline both for entrance into the epidural space and during tunneling of the lead. The fracture usually occurs as a result of lead compression between bony surfaces. The symptoms of lead fracture include sudden loss of stimulation, and quick diagnosis can be made by verifying impedance data.

Next, another frequent complication of SCS is inadvertent dural puncture. The rate of this complication even with experienced specialists is usually estimated at

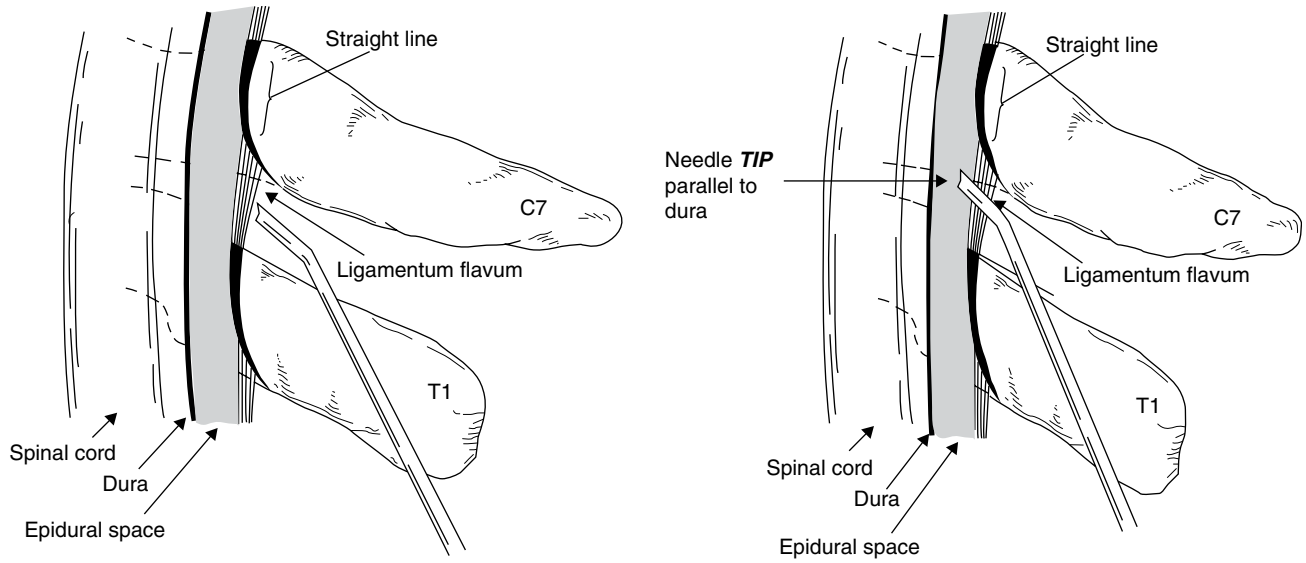

Figure 43.2 Demonstration using an RX-Coude needle to enter the epidural space. Courtesy of Epimed International. With permission from G. Racz.

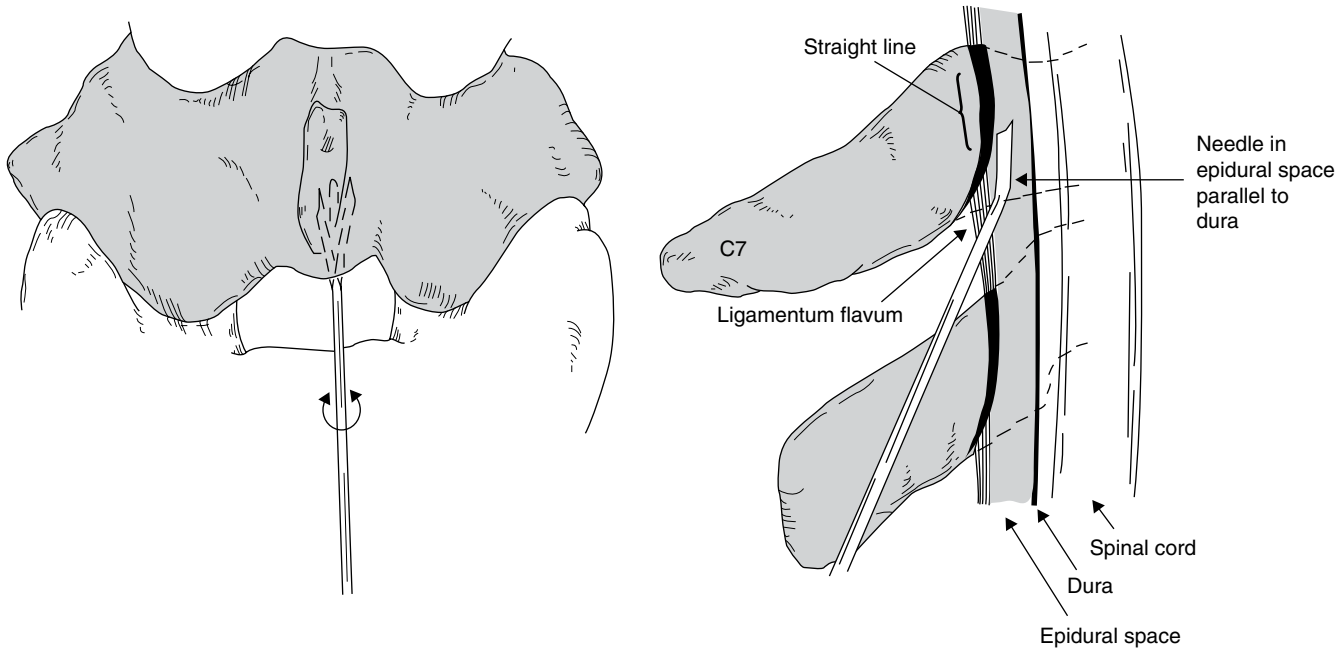

Figure 43.3 Demonstration using a RX-Coude needle to enter the epidural space. Notice that the bevel is down upon entering the space and then carefully turned up, thus theoretically decreasing the risk of dural puncture. Courtesy of Epimed International. With permission from G. Racz.

1%. Dural puncture can occur with the introducer needle itself, or it can occur with advancement of the SCS lead through the introducer needle. The tip of the SCS leads are somewhat firm and can easily puncture the dura. The practitioner should have a preformulated plan for how to proceed if this complication does occur. There is insufficient evidence-based data in the literature to establish universal algorithm for management of this complication. The decision is based upon the practitioner's clinical judgment and includes either abandonment of the procedure and rescheduling for 2 to 3 weeks later, or continuing the procedure at an alternate spinal level. The disadvantage of continuing the procedure includes the potential onset in the patient of a postdural puncture headache, which may impact the value of the trial or implant. Frequently, especially in younger patients, the headache will require epidural blood patch which some experts argue may also increase the chance of infection during the trial or implant.

Table 43.1 Sensory Mapping in Spinal Cord Stimulation

Location of Pain	Approximate Spinal Cord Stimulator Lead Placement
Neck	Usually at C1-C3 levels No major variation between midline and lateral placement
Shoulder	Usually C2-C4, satisfactory coverage inconsistent
Hand, arms, fingers	Usually C4-C6

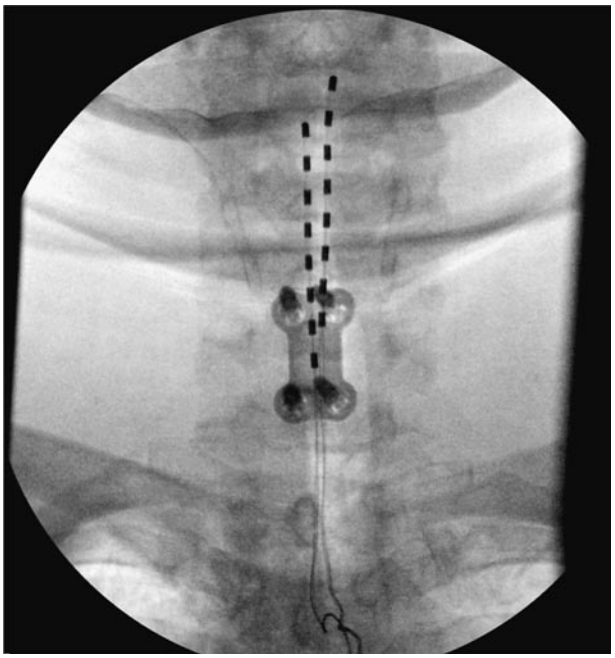

Figure 43.4 Anteroposterior fluoroscopic view. Dual eight-contact percutaneous leads placed in a patient with cervical postlaminectomy syndrome with symptoms of right-sided neck, arm, and hand pain.

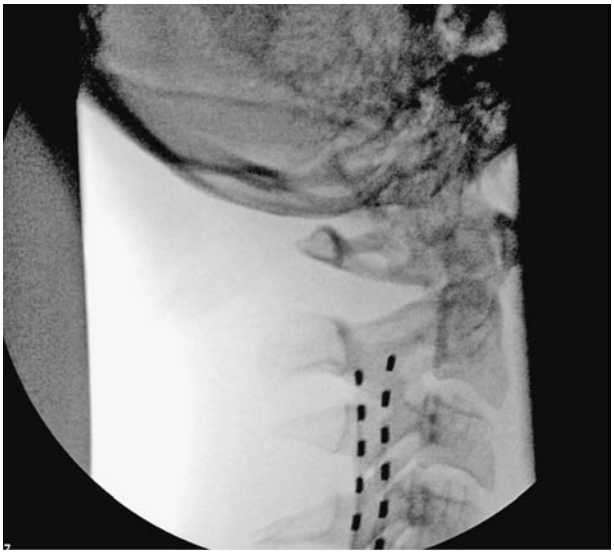

Figure 43.5 Lateral fluoroscopic view. Dual eight-contact percutaneous leads placed in a patient with symptoms of left-sided neck, shoulder, and arm pain. Notice that one lead is placed in the left lateral recess to achieve better coverage of the left shoulder.

However, abandoning the procedure is not without serious implications as well. For example, some patients may be forced to obtain a second authorization for the procedure from their insurance carrier, and therefore risk denial of access to the treatment.

Another potential complication of SCS is infection. It is important to identify and optimize patients who are at increased risk for infection such as diabetics with poor glycemic control, patients on immunosuppressive medications, immunocompromised patients, patients with systemic infections, and patients with local infections near the procedure site. Further, many practitioners advocate pre- and postoperative antibiotics in addition to strict sterile technique. Early identification and aggressive treatment of superficial wound infections may prevent more extensive infection and avoidance of explantation of the device. Regarding permanent implants, the infection usually presents in the first 10 to 14 days following the procedure.

Bleeding at the generator pocket site and in the epidural space (epidural hematoma) are also serious potential complications. To minimize the risk of bleeding, it is prudent to identify those patients who are on anticoagulants and to closely follow the accepted guidelines for perioperative management of anticoagulants during neuraxial procedures. The American Society of Regional Anesthesia published the most frequently followed guidelines for those interventional pain physicians whose primary background is anesthesiology [28].

Next, there is a risk of spinal cord and/or nerve injury during placement of the large-bore epidural introducer needle and during advancement of the SCS lead(s). It is possible to reduce this risk by ensuring an "awake" patient during the key portions of needle and lead placement (light anesthesia is acceptable and common) so that they can alert you should they experience paresthesias or unexpected pain. The use of fluoroscopy with frequent lateral images during needle advancement can help minimize inadvertent dural puncture and spinal cord injury. The typical LOR upon entrance into the epidural space is much less pronounced with the introducer needle than with the typical Tuohy needle commonly used for epidural injections. Epidural hematoma is also a risk given the large-bore needle used, and practitioners should be well aware of the clinical signs and symptoms of this complication.

The generator pocket site is the source of several potential complications. First, of these is seroma, which is a gathering of sterile fluid in the wound pocket. The incidence of seroma formation can be reduced by using a blunt dissection technique to minimize tissue damage, creating an appropriately sized device pocket (avoid excessively large pockets), and ensuring that at-risk patients have adequate serum albumin levels. The treatment of seromas can include the use of abdominal binders, and although controversial, some experts suggest sterile needle aspiration.

The depth of implantation of the generator is another important and occasionally overlooked consideration. The first rechargeable generator was introduced in 2003, and

to be able to charge these generators, the depth of subcutaneous implantation cannot exceed the manufacturer's recommendations. Practitioners should be aware of the manufacturer's criteria regarding generator depth.

Finally, surgical wound dehiscence is a more remote possible complication and unfortunately typically involves removal of the implanted hardware, and reimplantation after adequate time for tissue healing.

OUTCOMES AND COST-EFFECTIVENESS

In a meta-analysis of 49 studies in which SCS systems were implanted for chronic pain patients who had a greater than 50% pain relief or a statistically significant reduction in visual analogue scale, investigators showed a long-term (>6 months) success rate of greater than 67% of the patients [29].

The medical costs of SCS compared with an alternative regimen of surgeries and other treatments in patients who respond well to SCS showed a payback period of 2.1 years or less. This includes factors such as the high initial cost of the system, periodic generator replacement, and revision [30]. A prospective study in 219 patients by Burchiel et al. indicated that patients with SCS, with whom a 1-year follow up was available, reported a significant long-term improvement in pain and quality of life. These patients had SCS placed for chronic back and lower extremity pain. These patients also showed marked improvements in pain intensity, social interactions, sleep, mobility, depression, and most aspects of daily living. These data, in combination with a low complication rate, demonstrated that SCS represents a relatively safe and effective approach in long-term pain management [31].

CONCLUSION

SCS has been shown to be a valuable option for those suffering with cervical radiculopathy. The physician should understand the indications, lead targets, techniques, and complications management to successfully treat this patient group. Future advances will continue to expand the candidates for this therapy over the next decade.

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44 Functional Rehabilitation of Painful Cervical Spinal Disorders

David J. Kennedy, Christopher J. Visco, and Joel Press

INTRODUCTION

Cervical spine pain is exceedingly common, with the lifetime prevalence being between 67% and 71%, and the 1-year incidence between 16% and 18%, slightly more common in females [1,2]. Degenerative changes increase with age, and by age 70 they are ubiquitous on magnetic resonance imaging, although not necessarily indicative of pain [3]. The majority of cervical spine pain cases is self-limiting and responds well to treatment, particularly with early intervention. The patient with severe or chronic injury may require a more extensive evaluation and management. The development of prolonged injury and pain is multifactorial in nature and can be due to pain avoidance patterns leading to weakness of surrounding musculature, postural changes, loss of spine flexibility, and altered biomechanics of the spine. This can significantly delay healing or even predispose to secondary injuries. The purpose of this chapter is to review current concepts regarding the functional rehabilitation of painful cervical spine disorders through rehabilitation techniques.

SOURCES OF CERVICAL SPINE PAIN

The etiology of spine-related pain can be divided into local and referred causes. Similar to lumbar pain, local causes of cervical spine pain include those structures that are implicated in degeneration or damage such as zygapophyseal joints (Z-joints), intervertebral discs, ligaments, muscles, or nerves. Referred pain can occur from many of the same structures, in particular Z-joints, and ventral rami have specific referral patterns. In addition to local and referred causes of spine pain, there are multiple pain syndromes clustering a constellation of symptoms such as whiplash and cervicogenic headaches.

The term cervical strain is nonspecific nomenclature used to refer to any pain originating from a muscle or tendon about the neck. The underlying pain generator should be correctly identified to help guide treatment. This can be challenging given the large potential variety of etiologies including the following: muscles, ligament or tendon injury, herniated nucleus pulposus, discogenic, Z-joint, cervicogenic headache, whiplash, rheumatologic disease, and neuritis, to name a few.

One of the most common causes of cervical pain is the arthrodial joints [4]. They are composed of articular cartilage surrounded by synovium and a complete capsule, and oriented in a coronal plane in the cervical spine. The innervation originates from medial braches off the dorsal rami at the vertebral level above and below the joint. Any structure about the joint can be a pain generator. The referral pattern of pain originating from the cervical Z-joint can be in the periscapular area, posterior and lateral shoulder [5–8]. Zygapophyseal pain should be considered in patients with complaints of pain in these areas. Degeneration at the C4–5 joints is most common, with the prevalence as high as 30% in older populations [9]. Z-joints should be distinguished from uncovertebral joints, which are not true joints, but an articulation of adjacent vertebral bodies. Another common cause of cervical pain is discogenic pain [4]. Degenerative disc changes can include loss of disc height, desiccation, annular tears, and herniated nucleus pulposus.

These various potential pain generators all have different mechanisms of injury and subsequent pain patterns, and it is the duty of a spine specialist to be familiar with the presenting symptoms and natural history to properly diagnose and treat.

NATURAL HISTORY OF CERVICAL SPINE PAIN

Although cervical spine dysfunction is extremely prevalent, it results in far less work absenteeism compared with lumbar spine pain and only accounts for 2% of workplace injuries [10]. It is however a major source of functional loss and pain following whiplash-type injuries from motor vehicle accidents [11].

Despite its high prevalence, the natural history of cervical spine pain is favorable. Although it varies somewhat between underlying etiologies, 80% to 90% of patients have pain resolution within 8 weeks [12,13]. Nonoperative treatment for cervical radiculopathy has also been shown to have good to excellent outcome in most cases [14]. However, in some patients, neck pain can be protracted, recurrent, and episodic. In two long-term studies, nearly 60% of subjects reported recurrent problems [15,16]. In another 12-year follow-up study, only 4% of those initially sick listed were neck pain free and 44% the same or worse [17]. In yet another study of 800 individuals, 48% of subjects had symptoms 1 year later [18]. Taken together, the

literature demonstrates at least an overall 40% relapse rate [2]. So despite the initially good improvement regardless of intervention, these patients will often have recurrent symptoms. Given this high relapse rate, it is likely that some underlying mechanism predisposes the injured neck to re-injury.

It is known that the spine obtains it stability primarily from muscles, and that the denuded spine buckles with minimal pressure [19]. There is also a multitude of literature on cervical spine musculature being abnormal with decreased cross-sectional area, fatty infiltration, type 2 muscle atrophy, abnormal timing of activation, and overall decreased activation [20], following pain or injury of any type to the cervical spine [21].

Muscle function in static and dynamic positioning is needed for normal cervical spine function and also likely predisposes the spine to re-injury when imbalanced or abnormal. Literature assessing functional rehabilitation for lumbar spine pain shows decrease recurrence rates [22]. Functional rehabilitation programs for the cervical spine are based on these data. It is felt that a functional rehabilitation program will not only enhance recovery but may also decrease recurrence rates. The rest of this chapter goes through a functional restoration algorithm to reduce pain, restore function, and decrease recurrence of pain, with an emphasis on the kinetic chain and understanding common maladaptive patterns and ways to treat them.

Table 44.1 Stages of Functional Spine Rehabilitation

I. Initial phase
Pain control medications (anti-inflammatory, etc.)
Physical modalities
Peripheral or axial injections
Activity modification
II. Restorative phase
Correcting local flexibility and strength deficits
Mobilization of hypomobile tissues
Stretching exercises to improve trunk and extremity flexibility
Strengthening exercises to improve cervical or lumbar stability
Local flexibility and strength deficits correcting kinetic chain
Abnormalities that affect the cervical spine
Restoration of normal posture and positioning
Must include kinetic chain analysis for deficits predisposing to mechanical abnormalities
III. Functional strengthening
Normalization of spine mechanics
Following a continuum from static to dynamic functional strengthening
IV. Final phase
Pain free
Preinjury range of motion and strength

FUNCTIONAL REHABILITATION

Functional rehabilitation typically follows a standard logical progression through interconnected stages. See (Table 44.1). The main goal of the first stage is to adequately control pain. Mechanisms to accomplish this include physical therapy modalities such as exercise and manipulation, acupuncture, medications, and injections. Once pain is adequately controlled, the second stage proceeds with a focus on restoration of normal spine movement patterns. It is imperative to regain normal movement patterns prior to addressing strength. An emphasis on restoration of normal posture and positioning defines the third stage. This includes a full kinetic chain analysis for any potential abnormalities that predispose to abnormal, painful cervical mechanics including evaluating the linkage of the upper and lower quadrants through both hip and pelvic girdle strength and scapulothoracic stability. Once proper joint movement and posture are obtained, strengthening follows in the fourth stage with a continuum from static to dynamic functional strengthening. The rest of this chapter will outline a progression though these four stages.

Stage One—Pain Control

It is imperative to achieve adequate pain control to facilitate maximal functional recovery. There are a wide variety of agents that are currently utilized for functional pain control. The majority of these agents are covered elsewhere in this text and are beyond the scope of this chapter. There are, however, several key points demonstrated by the literature regarding therapeutic agents for pain control. First, although some degree of activity modification is prudent in most acute pain syndromes, aggressive controlled physical activity (i.e., bed rest) should be avoided. It is known that the cervical spine obtains its stability primarily from muscles [23]. In fact, it has been shown that the denuded lumbar spine buckles with a minimal pressure of 20 pounds [19]. Because bed rest results in rapid and substantial strength loss [24,25] above and beyond that already seen in painful spine disorders, it is counter-productive to a functional rehabilitation program to weaken these muscles and possibly cause a protracted course with delayed return to normal activity level and increased relapse frequency.

Hard cervical collars are frequently utilized for obvious bone or ligamentous injury that may result in instability, but are rarely used for pain control. On the other hand, soft cervical collars are frequently utilized for painful cervical conditions, although in most cervical spine disorders, they should have a limited role [26]. Although as many as 76% of patients report decreased pain with use of a soft collar [27], most studies do not demonstrate a benefit to their use in painful cervical disorders [28]. Also with prolonged use, there is a concern for atrophy-related secondary damage [29], although this fear is based on studies of immobilization with a closed plaster cast and animal data [30]. Regardless, if a soft collar is used for acute cervical pain control, it should be used for as short a time as possible (i.e., a few days) to minimize the potential deleterious side effects.

Another frequently utilized modality to help acutely control pain is cervical traction. Although the exact mechanism remains unclear, investigators have attributed the therapeutic effects of traction to a multitude of

Table 44.2 Cervical Traction—Contraindications

1. Ligamentous instability
2. Osteomyelitis
3. Diskitis
4. Primary or metastatic tumor
5. Spinal cord tumor
6. Severe osteoporosis
7. Clinical signs of myelopathy
8. Severe anxiety
9. Untreated hypertension

possible mechanisms including the following: unloading the components of the spine through stretching muscles, ligaments and functional units [14], decreasing adhesions within the dural sleeve [14], increasing joint mobility [31], decreasing intervertebral disc pressure [32], and relieving tonic muscle contractions [33]. Although there are limited prospective randomized studies regarding its use, overall when taken as a whole, the literature does reveal moderate evidence that the use of intermitted cervical traction has benefit, whereas the use of continuous traction has moderate evidence of no benefit [34].

Although a wide variety of traction forces have been utilized in the literature overall, most agree that a force of 25 pounds is adequate to off load the weight of the head and possibly produce therapeutic effects. Maximal distraction generally occurs between 20° and 30° of flexion without rotation or side bending. The use of intermittent cervical traction does have several contraindications that the practitioner must be aware of before its use. See (Table 44.2). In addition to contraindications, caution should be used in those patients with acute radiculitis or inflammation about the nerve root as cervical traction can exacerbate symptoms. It is prudent to trial manual traction to evaluate for exacerbation of symptoms and also to see if some relief, even temporary, is possible prior to instituting treatment with a home unit.

Stage Two—Restoration of Proper Joint Mechanics

Once pain has been adequately controlled with these and other techniques, the practitioner should attempt to restore proper joint mechanics. It is imperative to restore proper mechanics prior to a functional strengthening program to avoid maladaptive and compensatory muscle activation strategies. Frequently, the restoration of proper joint mechanics occurs simultaneously with the previous stage as it is often felt to help decrease pain. It is often difficult to put good functional strength on bad movement patterns and limited ranges of motion.

To fully restore proper joint mechanics, joint kinematics must be assessed with a focus on mobility. However, the literature shows a wide variability in the interexaminer reliability of tests assessing both the segmental mobility of the cervical spine and passive intervertebral motion with kappa values ranging from 0.01 to 0.8 [35,36]. There is a substantial heterogeneity of the clinical tests studied in these trials, thus making it difficult to draw a definite conclusion about their reliability [37]. The literature does, however, support the ability to generally demarcate hypomobile joints versus hypermobility versus normal mobility; and this can be useful in guiding treatment [38,39]. A recent Cochrane review also demonstrated some evidence of cervical stretching and range of motion in some cervical spine disorders, but only when combined with stretching and strengthening of the shoulder and thoracic region [40].

This demarcation is important as it is conventionally felt that the hypomobile joint responds to mobilization that can occur through a variety of techniques. The large variety of functional techniques available to the practitioner to restore motion to the hypomobile joint include the following: manual manipulation with high velocity low amplitude trusts, soft tissue mobilization, strain/counterstrain, muscle energy, neural mobilization, manual techniques, Z-joint segmental mobilization manipulation, etc. There are a large variety of studies on these various techniques; however, most studies are small, assess a heterogeneous group of underlying pathology, and utilize multiple different techniques. Because of the great variation in the exact technique and treatment protocols used in these studies, it is difficult to draw definite conclusions as to the most effective technique to treat each pathology. However, when taken as a whole, the results from clinical studies published to date indicate that these techniques may be effective at reducing spinal pain and restoring appropriate joint kinematics [40].

Stage Three—Kinetic Chain

Although progressing through a functional rehabilitation program, it is useful for the practitioner to review total body mechanics with a thorough evaluation of the entire kinetic chain. To adequately design a treatment algorithm that restores proper mechanics to the cervical spine, the evaluating clinician must possess a full understanding of the kinetic chain and its effects on the cervical spine. The exercises utilized in the first two stages of functional rehabilitation can be augmented by treatments aimed at correction of underlying biomechanical abnormalities described later.

There are multiple factors that affect the posture and mechanical movement of the cervical spine. In addition to the direct anatomical structures of the spine, extrinsic postural abnormalities exert forces on the cervical spine through the kinetic chain. Proper positioning must involve all aspects of the kinetic chain including linkage of the upper and lower quadrants and a focus on achieving a neutral spine. For instance, the seated position can contribute to abnormal cervical spine mechanics through the kinetic chain with multiple potential originators. Shortened hamstrings can accentuate the normal posterior tilting of the pelvis during sitting. This tilting further accentuates the normal loss of normal lumbar lordosis that occurs with seating. The loss of normal lordosis results in increased thoracic kyphosis. The protracted shoulder

girdle and increased glenohumeral internal rotation serves merely to further accentuate this maladaptive posture. Scalene hyperactivity results in a fixed and elevated first rib. To keep looking forward, the neck has a resultant hyperflexion of the lower cervical spine and upper thoracic spine with an unlocking of mid cervical Z-joints and decreased size of intervertebral foramen in mid cervical spine. This overall posture combines with hyperextension at the suboccipital and upper cervical segments and leads to mechanical abnormalities, segmental dysfunction, and resultant pain. These forces functionally push the head and neck forward [2].

All of these biomechanical alterations can result in a head-forward posture. The multiple segments involved are interconnected, and dysfunction at any segment can cause compensatory changes distally and proximally through the kinetic chain. The goal of the rehabilitation professional is to correctly identify the underlying pathology and treat it, even if the segmental dysfunction is removed from the primary pain generator. Thus, the neutral spine should be optimized with a focus on lumbar lordosis, retracted shoulder, and chin parallel to the floor. This position allows for optimal mobility of the cervical spine. The neutral spine position helps to minimize any potential abnormal segmental forces so that individual structures do not experience undue strain. This is of extreme importance during activities of daily living. The authors believe that excessive sitting and computer work help contribute to poor posture. The kinetic chain should be emphasized with upper and lower quadrant linkage. Scapulothoracic position is imperative and has a dominant effect over the cervical spine and direct linkage to the lower quadrant through muscles such as the latissimus dorsi [41].

Figure 44.1 Cervical traction unit with force applied at 25° flexion.

Figure 44.2 Head-forward posture, with a protracted shoulder girdle resulting in hyperflexion of the lower cervical spine and hyperextension of the suboccipital and upper cervical segments.

Stage Four—Functional Strengthening

Functional strengthening should commence only after appropriate joint kinematics have been restored. Muscle dysfunction has been repeatedly shown in patients with neck pain [42]. Abnormal firing patterns and muscle activation of the upper trapezius has been documented with surface electromyography. Also, cervical multifidus muscles have been shown to have a smaller cross-sectional area in those with chronic neck pain, as demonstrated with ultrasound [21].

In light of this research, it is no surprise that therapeutic exercise has repeatedly demonstrated efficacy in reducing pain and perceived disability in people with neck pain disorders [43–45]. In addition to a change in symptoms, therapeutic exercise has been shown to improve cervical muscle function [43,45–47] although changes in muscle activation patterns may not occur [48].

Treatment algorithms have varied greatly in the published literature and have included the following: high load strength training of the neck flexors [49], Feldenkrais and home exercise program [50], endurance training of the neck flexors and upper extremities [51], craniocervical flexion exercises, cervicoscapular and postural exercises [46], shoulder strengthening [52], proprioceptive rehabilitation program with eye-neck coordination [53], group exercise and Pilates [54], eye fixation and cervicothoracic endurance exercises [55], and active and passive repeated movement such as McKenzie and Maitland [56]. To date, the literature has failed to conclusively prove the superiority of one specific type of exercise regimen over another. Given the heterogeneity of pathology in cervical spine disorders, it is likely that different techniques may be better suited to

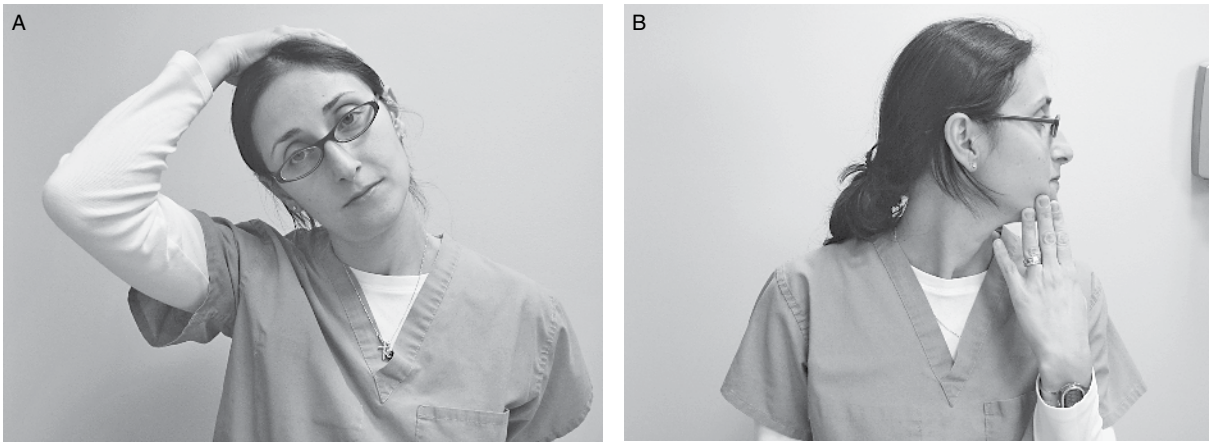

Figure 44.3 Cervical spine range of motion exercise. (**A**) Lateral bending. (**B**) Rotation.

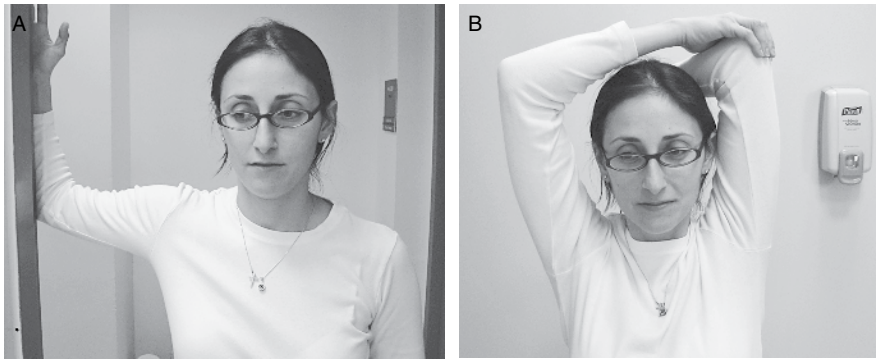

Figure 44.4 (**A**) Wall stretch for pectoral flexibility to help counteract protracted shoulder girdle. (**B**) Arm stretch to decrease excessive internal rotation associate that is frequently associated with a protracted shoulder girdle.

different pathologies although the literature to date is lacking in this area.

Despite this wide variety of treatment options available, overall the literature does demonstrate efficacy of exercise for painful cervical spine disorders. However, the literature has failed to establish which specific exercise or routines are most effective in the treatment of painful cervical disorders. Exercise regimes targeting cervical sensorimotor functions and eye-head-neck coordination have been shown to reduce pain and improve kinesthetic sense [44,55,57].

Regardless of exact exercise program utilized, the regimen should follow a natural progression starting with static exercises and progressing through to dynamic activities. For instance, cervicothoracic stabilization should be incorporated early into the exercise program for optimal outcomes to establish postural changes prior to strengthening. Please see Figures 44.1 to 44.7 for a sample cervical spine exercise program. Realize that exercises should always be tailored to the individual and their deficits.

SUMMARY

Functional rehabilitation of painful cervical disorders must be integrated into the rehabilitation program to decrease both pain and the likelihood of recurrence. After addressing proper pain control, progressing through flexibility, posture, and strengthening can increase the chances of a good result. Because there is not convincing evidence on the exact exercises to use, the focus should be on improving flexibility in the hypomobile areas and stabilizing the areas that are weak. The importance of a thorough evaluation identifying these deficits cannot be overemphasized.

Figure 44.5 Chin tuck exercise helps give the patient proprioceptive feedback to correct position of neutral cervical spine.

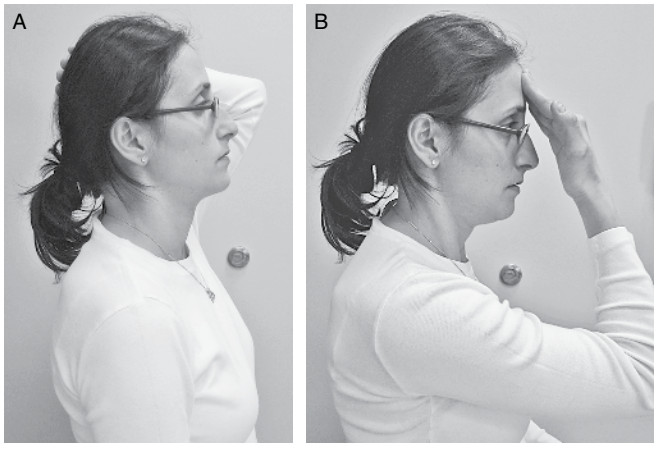

Figure 44.6 Cervical spine (**A**) extension strengthening and (**B**) flexion strengthening, can be done with either isometric or isotonic resistance.

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Figure 44.7 "Elbows in back pocket"—a simple home exercise that helps accentuate shoulder retraction, and potentially helps restore the proper cervical spine kinematics.

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Part III
Osteoporotic Spine and Pelvis

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45 Biomechanics and Pathophysiology of the Osteoporotic Spine and Pelvis

Mark Makumbi Kayanja

ANATOMICAL OVERVIEW

The spine is composed of 7 cervical, 12 thoracic, 5 lumbar, and 5 fused sacral vertebrae. The spine normally has a cervical lordosis, a thoracic kyphosis, and a lumbar lordosis. Volume of the vertebral body increases in a cranial to caudal direction (Figure 45.1). A lumbar vertebra is composed of both anterior and posterior elements (Figure 45.2). The vertebral body lies anterior and is connected to the posterior elements through pedicles. The posterior vertebral arch is composed of laminae that coalesce into a spinous process. The superior articular facet of the vertebra articulates with the inferior articular facet of the superior adjacent vertebra, and the pars lies at the lateral margin of the laminae joining the pedicle to the inferior articular facet.

Figures 45.3 and 45.4 show a three-level thoracic vertebral segment (T10-12) with intervening discs. The ligaments joining the vertebrae include the anterior longitudinal ligament (ALL), the posterior longitudinal ligament (PLL), the ligamentum flavum (LF), the interspinous ligament (IL), and the supraspinous ligament (SL).

Figure 45.5 shows a sagittal cross section of a thoracic vertebral segment with vertebral bodies and intervening discs, the ALL, PLL, LF, and IL. The intervening disc is composed of an outer annulus fibrosus and an inner nucleus pulposus as shown in Figure 45.6.

Load is transferred through the spinal column anteriorly through the vertebral body and disc, and posteriorly through the posterior elements. There is a sexual difference in the size of vertebrae with the cross-sectional area of female vertebrae 25% smaller with 30% to 40% greater stress in women than in men for equivalent loads, which may explain the higher number of VCF in elderly women [1].

OSTEOPOROSIS

Osteoporosis is defined by the World Health Organization Working Group based upon the measurement of bone mineral density (BMD) using dual-energy X-ray absorptiometry. Osteoporosis is BMD < 2.5 standard deviations below peak value for young adults (T score) [2]. Osteoporotic vertebral compression fractures (VCF) are an important public health concern, leading to significant morbidity, mortality, and economic burden, with the incidence of osteoporotic VCF increasing with advancing age. Peak bone mass and menopause determine the magnitude of bone loss while physical activity and peak bone mass are important predictors of bone density. Weight-bearing exercise stimulates the maintenance and improvement of bone health [3–6].

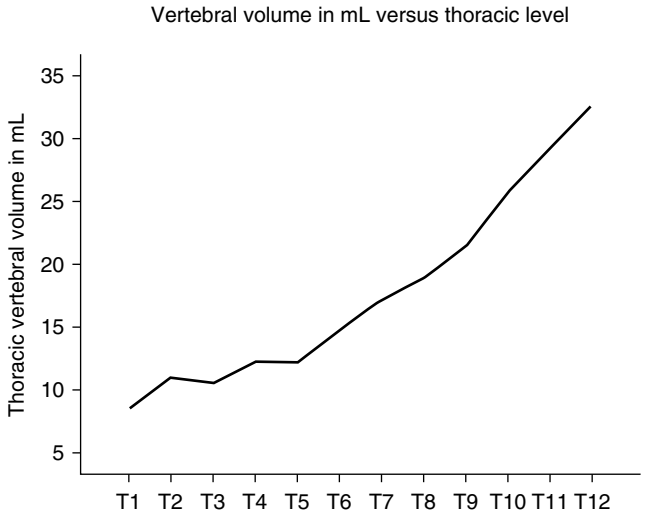

Figure 45.1 Volume of the vertebral body versus cranio-caudal level.

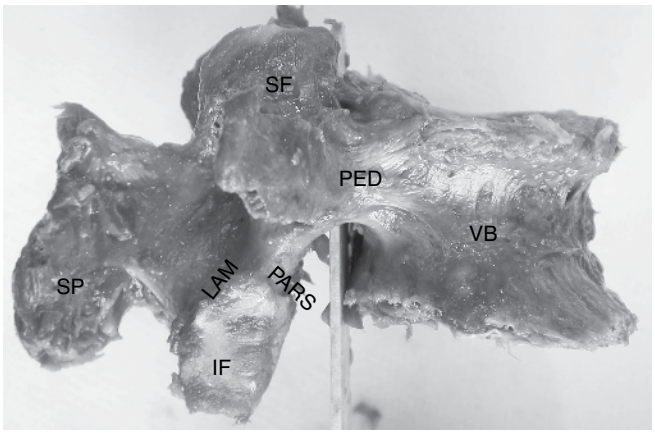

Figure 45.2 Lumbar vertebra. SP, spinous process; SF, superior articular facet; LAM, lamina; IF, inferior articular process; PED, pedicle; VB, vertebral body.

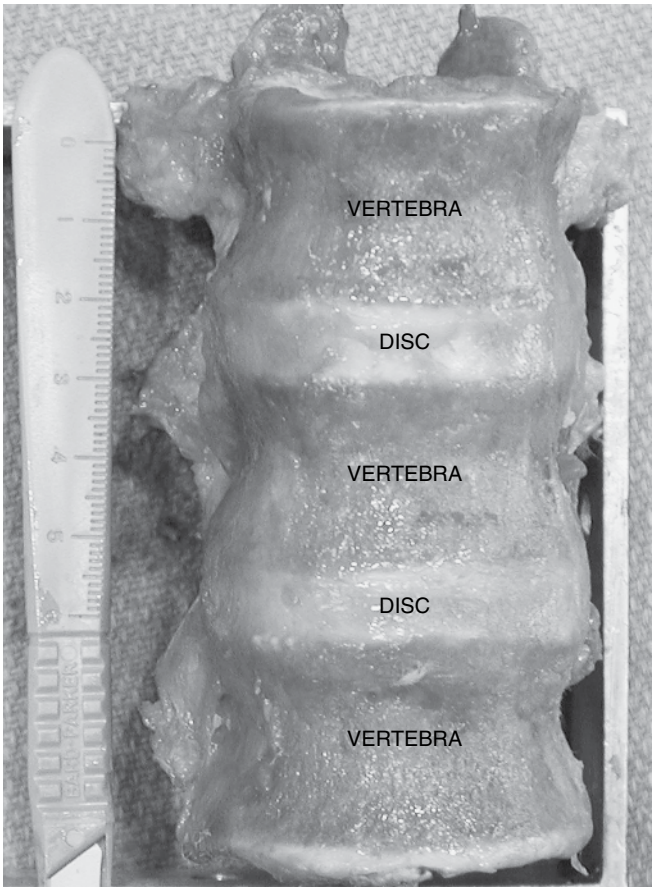

Figure 45.3 Anterior view of a 3-level thoracic vertebral segment (T10-12) with intervening discs.

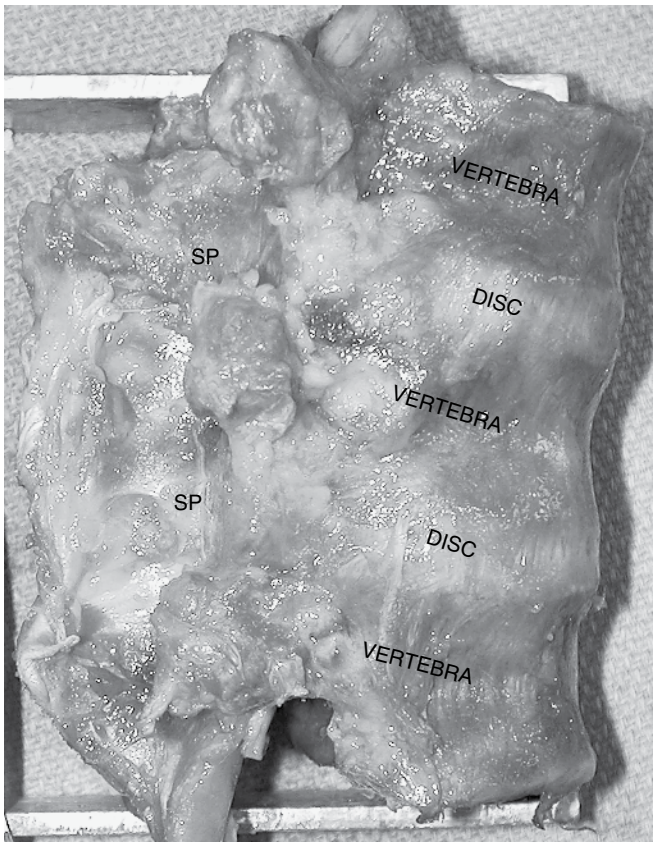

Figure 45.4 Lateral view of a 3-level thoracic vertebral segment (T10-12) with intervening discs.

Calcium supplementation helps achieve and maintain bone mass while smoking and alcohol reduce bone mass. Individual genetic factors predict the peak bone mass [7], which is the point from which decline occurs with advancing age. Lower peak bone mass will result in a greater likelihood of bone mass decline resulting in osteoporosis.

VCF are a common clinical problem and may follow trauma or be pathological. Osteoporosis increases susceptibility to fracture by reducing bone mass and weakening bone architecture. Approximately 2.5 million osteoporotic fractures occur worldwide annually, usually involving the vertebrae, wrist, and hip. In the United States 700,000 VCF occur annually, and an initial VCF often leads to subsequent VCF [8].

Pathophysiology

Osteoporosis may be primary (idiopathic), which occurs with age or following menopause. Secondary osteoporosis may occur from an underlying disorder. Risk factors for the development of primary osteoporosis include female sex, smoking, estrogen or testosterone deficiencies, and sedentary lifestyle. Secondary osteoporosis may result from endocrine diseases like Cushing's syndrome, hyperthyroidism, primary hyperparathyroidism, use of drugs

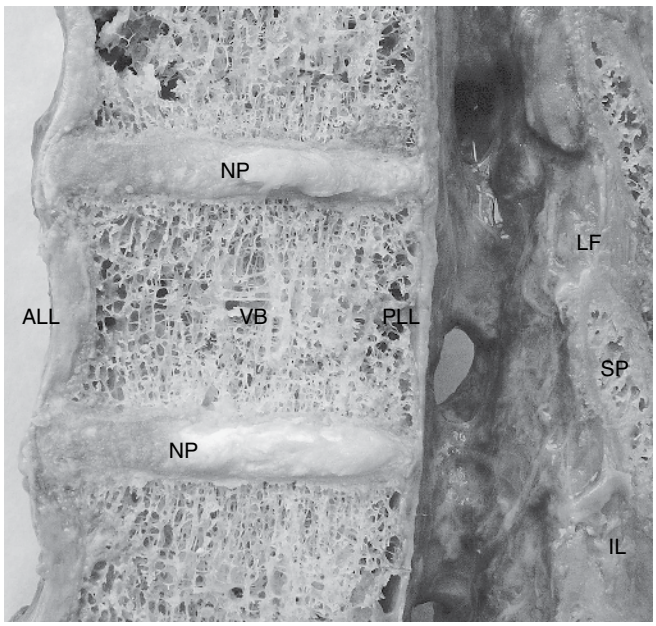

Figure 45.5 Sagittal cross section of a thoracic segment. ALL, anterior longitudinal ligament; NP, nucleus pulposus; PLL, posterior longitudinal ligament; VB, vertebral body.

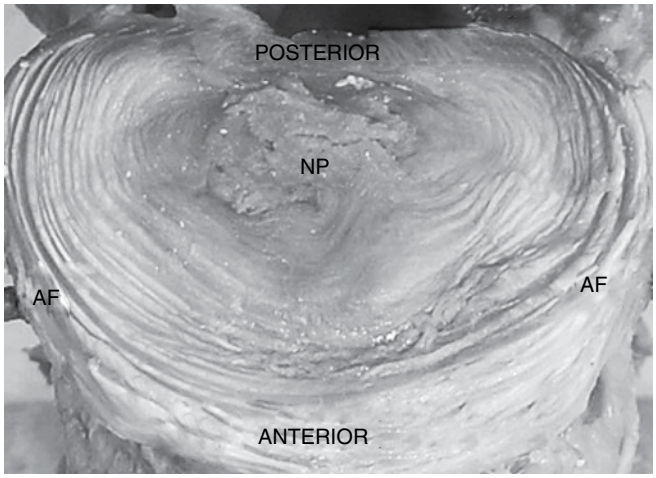

Figure 45.6 Axial section of the intervertebral disc. AF, annulus fibrosus; NP, nucleus pulposus.

such as glucocorticoids, heparin, and anticonvulsants, malignancy like multiple myeloma, tumors, chronic metabolic alkalosis as may occur in COPD, absence of gravity, and sarcoidosis.

Bone formation by osteoblasts and resorption by osteoclasts are coupled and regulated by parathyroid hormone, calcitonin, estrogen, vitamin D, cytokines (interleukin-1, tumor necrosis factor-α, granulocyte-macrophage colony-stimulating factor, interleukin-6), and local factors. Peak bone mass occurs by mid-20s after which there is a decline. The reduction in bone mass may then occur from increased osteoclastic activity affecting predominantly trabecular bone in postmenopausal osteoporosis, or from reduced osteoblastic activity. The resultant effect is a reduction in bone mass and alteration in microarchitecture that leads to increased fragility and VCF [9].

Bone Strength

Bone strength is not only determined by BMD but also by bone architecture. Osteoporotic VCF occur from a combination of a reduction in bone mass and alteration in microarchitecture. The occurrence of osteoporotic VCF is inversely related to BMD [10] and with loads encountered during normal activities [11]. The age-related reduction in bone mass occurs concurrently with trabecular thinning, increased trabecular spacing, and loss of trabecular contiguity [12,13]. Age alone is responsible for 70% to 80% decline in bone mass [14]. Bone architecture is characterized by trabecular number and morphology, trabecular inter connectivity, and the volume of the marrow space. Osteoporosis leads to a reduction in trabecular thickness and number, an increase in connectivity and marrow space volume [15]. These changes are illustrated in Figure 45.7A showing normal vertebral body architecture and Figure 45.7B showing osteoporotic vertebral body architecture.

Mechanical Vertebral Dysfunction

The mechanical function of the vertebral body depends upon trabecular architecture [16]. Loss of trabecular connectivity results in strut buckling, which when coupled with a reduction in trabecular remodeling thus prolonged trabecular microdamage [17], culminates in VCF; this is seen in Figure 45.8. Fracture models used to predict VCF risk are more accurate when both BMD and microstructure are taken into account [18]; however, the inclusion of deformity and morphometry does not significantly improve modeling [19]. Vertebral BMD is determined by both cortical and trabecular components; yet, inclusion of these individual components in VCF risk prediction modeling does not significantly improve fracture risk estimation [20,21]. Compressive strength is therefore dependent primarily on BMD [22,23].

Osteoporotic vertebrae have yield strength significantly lower than normal so when subjected to loads from everyday activities, they develop cumulative microdamage [24]. This microdamage occurs at strains below that encountered in normal vertebrae and is concentrated in certain regions leading to VCF [25]. Physiologic load measurement in osteoporotic VCF showed significantly greater compression, shear force, and flexion moments than in non-osteoporotic vertebrae [26]. In the non-osteoporotic spine, stress is concentrated in the nucleus pulposus, the central endplate, and the middle of the posterior vertebral cortical wall. Osteoporosis and disc degeneration concentrate stresses peripherally within the annulus and anterolateral portions of the vertebral body, which results in the fracture patterns seen in VCF [27,28]. Figure 45.9 shows a wedge compression fracture at T12 and L3, and biconcave fracture at L2. Eighty-three percent of VCF are due to mild to moderate trauma, and a specific traumatic event is absent in 59% of cases [29]. VCF have also been associated with activities such as golf [30] and have been associated with malalignment above a lumbar kyphoscoliosis [31].

VERTEBRAL COMPRESSION FRACTURE

VCF Morphology

VCF may appear as wedge compression fractures where the anterior height of the vertebra is reduced, biconcave VCF where the middle height of the vertebra is reduced, and compression VCF where the anterior, middle, and posterior heights are all reduced [32] as shown in Figure 45.9 at L1. In the latter, the vertebra may appear as an area of increased density on radiography and further investigation with bone scans may be necessary to determine the etiology [33]. In contrast to Figure 45.9, Figure 45.10 is a normal L1-sacral segment with no VCF. An anatomical biomechanical model to predict the effect of vertebral bone loss and disc degeneration on the elderly spine showed that gravity and postural stresses caused anterior wedge-like fracture deformities at T7 and T8. The decreased spinal height and anterior translation of the spine created sagittal imbalance that resulted in increased compressive load in the thoracolumbar region of the spine [34]. VCF alter spinal posture by causing kyphosis from the reduction in the anterior vertebral height and preservation of the posterior column height. This kyphosis can be seen in Figure 45.9; kyphosis alters load transmission by concentrating stress in the anterior vertebral cortex. The ratio of the load on the

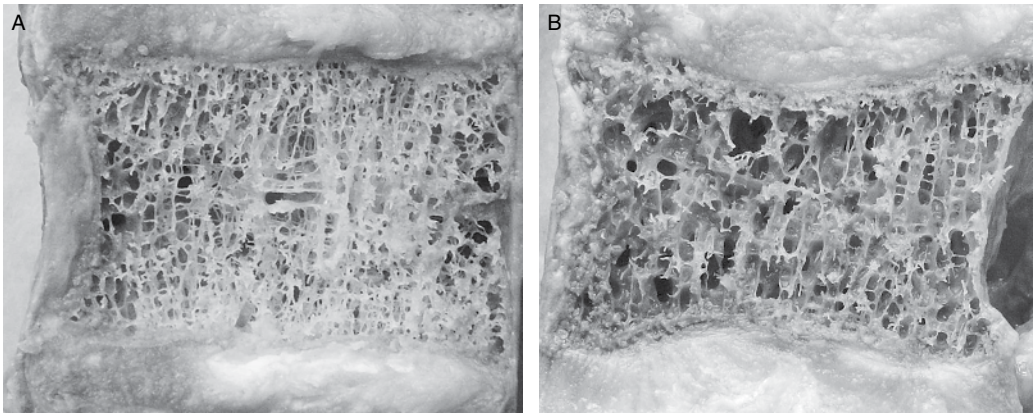

Figure 45.7 **(A)** Normal vertebral body architecture. **(B)** Osteoporotic vertebral body architecture.

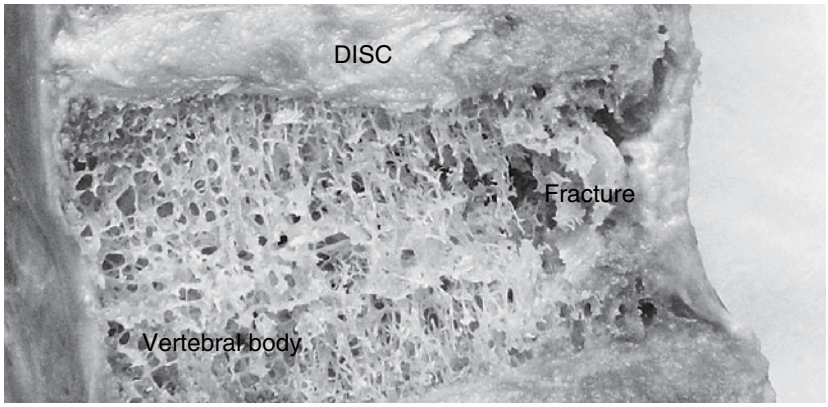

Figure 45.8 Morphology of a vertebral compression fracture.

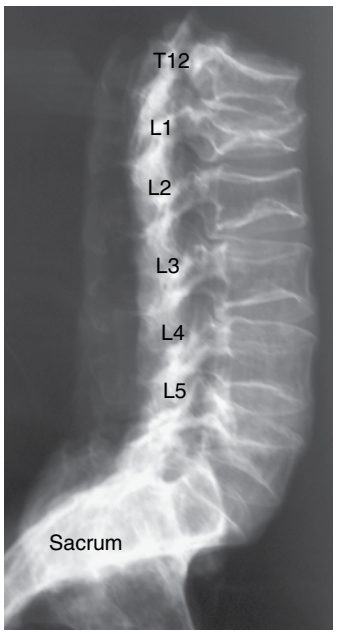

Figure 45.9 Vertebral compression fractures at T12, L1, L2 and L3.

spine to the failure load is called the factor of risk, a value that indicates whether a VCF is likely during a given activity. Failure load depends on density and architecture of the trabecular bone. Bending and lifting activities (compression and flexion loading of the spine) generate loads on the spine that exceed the failure load of vertebrae with very low BMD and may lead to VCF. VCF prevention involves reduction or limitation of these activities [35].

Spinal Load Transmission

The vertebral body is composed of a trabecular core surrounded by a cortical shell. Axial load is transmitted primarily through two main mechanisms: centrally through the vertebral body trabecular core to the adjacent disc nucleus and peripherally through the annulus and the vertebral cortex. Load is shared between these two components as a function of osteoporosis and disc degeneration, and the relative position of the adjacent end plates [36]. Figure 45.11 is a representative diagram of the motion segment in the neutral position. The spine undergoes movement in three dimensions; during axial compression the intervertebral disc is compressed causing an endplate deflection in the adjacent vertebral bodies and an annular bulge. The region of the spine that does not undergo displacement

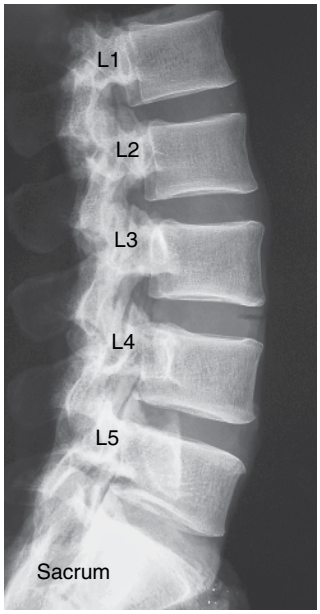

Figure 45.10 Normal L1-sacral segment with no VCF.

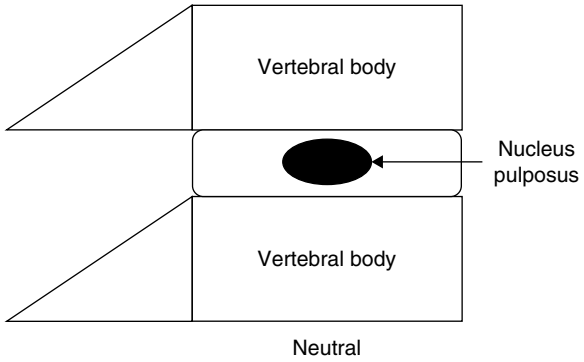

Figure 45.11 Representative diagram of the motion segment in the neutral position.

is the instantaneous axis of rotation; all points ventral to this come closer together in flexion and all parts dorsal to this move further apart. The opposite occurs in extension. During flexion, tension develops in the posterior elements; the nucleus is displaced posteriorly toward the PLL; this is shown in Figures 45.12A and 12B. In extension, tension develops in the anterior annulus; the nucleus is displaced anteriorly and the posterior elements are in compression; this is shown in Figures 45.13A and 13B.

Osteoporosis affects the centrum to a much greater degree than the cortical shell, leading to a greater degree of peripheral load transfer. In young healthy discs, the majority of the load transmission is evenly distributed throughout the central trabecular core to the adjacent disc nucleus. As the intervertebral disc degenerates, increased load is transmitted more peripherally within the vertebral body. The bulge of the vertebral body endplate and intradiscal pressure depends on the grade of osteoporosis in the vertebral body. A wedge-shaped vertebral body fracture causes an anterior shift of the upper body centre of gravity and a resultant increase in intradiscal pressure [37]. The compressive excursion of the motion segment is equivalent to the endplate displacement into the trabecular centrum; this displacement is in turn dependent upon the trabecular BMD [38]. In compression, predominantly tensile stress develops in the endplate as it is displaced into the vertebral centrum. As compression becomes flexion, the load is transferred from the trabecular centrum to the anterior cortical shell, and once fracture threshold is exceeded a VCF occurs [39,40]. Disc degeneration results in peripheral distribution of load and in compression this predominantly is transferred to the posterior cortex. Conversely in flexion anterior load transfer is greatly increased [41] increasing the risk of VCF. Figure 45.14 shows load distribution through a normal disc/normal bone and load distribution through degenerate disc/osteoporotic bone.

The major load-bearing pathway in the spine in compression is the vertebral centrum and in flexion is the anterior cortex. Strain is concentrated at the anterior cortex in flexion and when this strain is above the fracture threshold a VCF occurs. The strain previously concentrated at the VCF site is transmitted to the adjacent levels, and the predisposition to a subsequent VCF occurs from the altered load pathway [8]. This subsequent VCF becomes manifest with continued loading of the compromised spine in

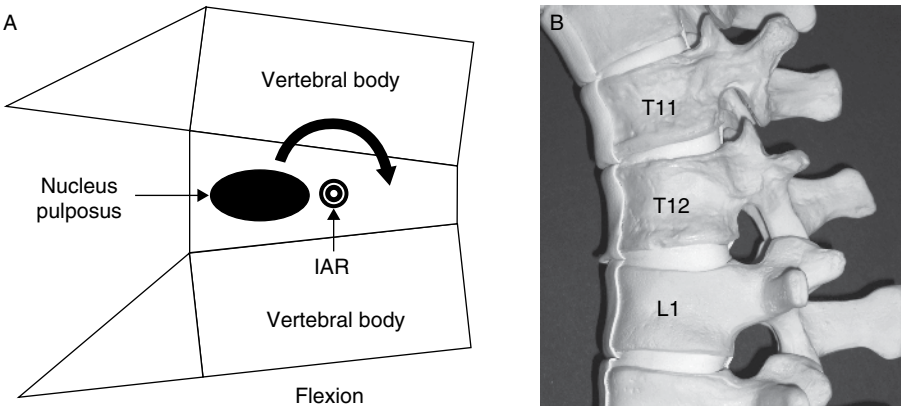

Figure 45.12 (A,B) Flexion of the motion segment.

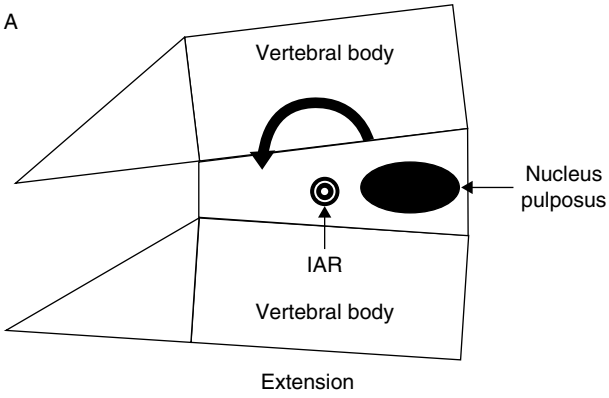

Figure 45.13 (A,B) Extension of the motion segment.

flexion with an anteriorly shifted load-bearing pathway. The strain at subsequent VCF is greater than that at initial VCF. Subsequent VCF have been observed to occur at the superior adjacent vertebra [42,43]. Cement augmentation of the VCF by kyphoplasty may lead to partial height restoration, but more importantly opens up an alternative pathway. This results in load transfer to adjacent vertebrae both centrally through the cement core and peripherally through the anterior cortex probably alleviating adjacent level fractures [42]. Figure 45.15A shows the central load pathway through the cement core and Figure 45.15B shows a sagittal section of a vertebra with augmentation. Natural history suggests that there is a 19% incidence of subsequent fracture in the year following a VCF when no surgical intervention is performed. These subsequent VCF are more likely to occur at the level above but also occur below and remotely [44]. VCF risk is greatly increased by flexion compared with compression while the augmentation of a fractured vertebral segment redistributes loads away from the superior adjacent vertebra to the inferior adjacent vertebra sparing it from VCF. Index fractures in osteoporotic vertebrae therefore may occur from everyday actions like forward flexion or loads from body weight [45].

There is a segmental increase in BMD from the thoracic to the lumbar spine with vertebral surface area and strength increasing from the thoracic to the lumbar spine. As adjacent vertebrae have similar BMD and morphometry, the location of VCF may then depend on local curvature [19,42,46]. VCF cause an anterior shift of the compressive load path in adjacent vertebrae and can induce additional flexion moments. This eccentric loading may then contribute to the increased risk of new VCF in adjacent osteoporotic vertebrae [47]. Intuitively, restoring alignment is a key issue in restoring load transfer in the spine.

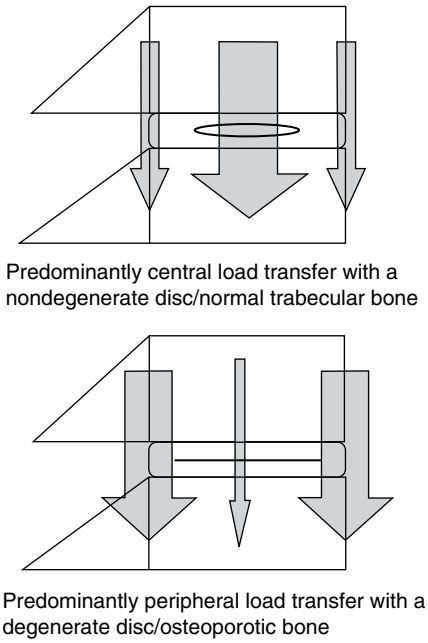

Figure 45.14 Load transfer in spinal segment influenced by disc degeneration and osteoporosis.

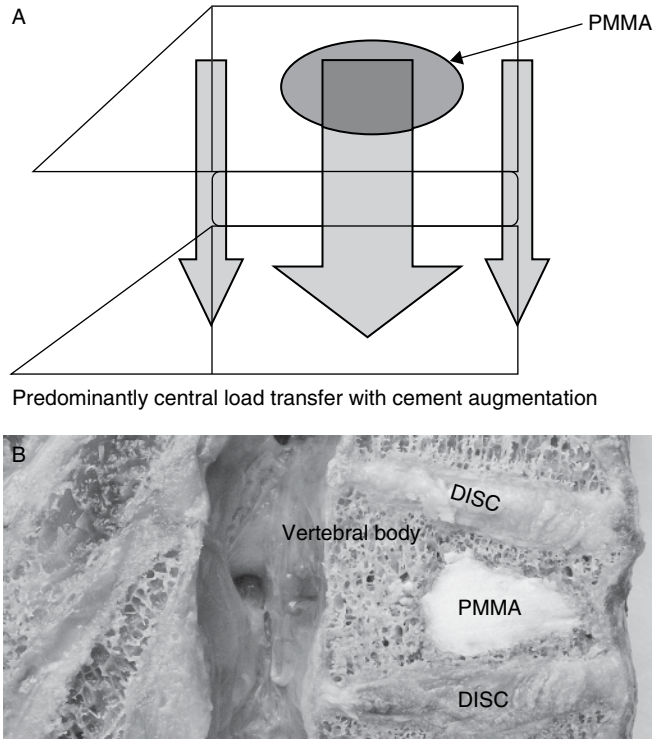

Figure 45.15 (A) Load transfer following cement augmentation. **(B)** Augmented vertebral compression fracture

SACRAL INSUFFICIENCY FRACTURES

The sacrum is the keystone of the spine and serves as the structure that transmits load from the lumbar spine to both lower limbs. The predominant weight-bearing portion of the sacrum is the sacral ala that forms the junction between the sacrum and the iliac wing. Figure 45.16 shows a pelvis indicating the regions commonly affected by sacral insufficiency fractures (SIF). Osteoporosis affects the sacrum in a manner similar to the rest of the spine. SIF are characterized by low back pain and sacral tenderness [48]. They were first described by Lourie in 1982 [49] that plain radiography may be unrewarding and imaging by bone scan, CT, or MRI may be necessary to adequately define these fractures [50]. Repetitive loading of the sacral alae result in cumulative microdamage that is eventually manifested as SIF. SIF cause low back pain and have characteristic patterns extending vertically in the sacral alae, parallel to the sacroiliac joints. These SIF are located just lateral to the margins of the lumbar spine and could be caused by load transmission through the spine [51]. Fractures biomechanically produced with vertical compression forces appear similar to SIF [52], and the consistent location of SIF has been associated with the stress from walking [53]. Treatment may be through rest and pain medication [54] or by cement augmentation. This cement augmentation may be performed under fluoroscopic or CT guidance [55–57], through either long-axis percutaneous sacroplasty [58]—cement injected along the SIF, or through short-axis sacroplasty—cement injected across the SIF [59]. The goal of augmentation is to inject cement into the alar void in the sacrum, which abuts the predominant weight-bearing region of the anterior sacral cortex in zone 2. Anatomic studies have demonstrated the predominant weight-bearing region and therefore the strongest is the anterior cortex of S1 in zone 2; this is followed by zone 3 then zone 1. The weakest area is zone 1 at the junction of S2 and S3 [60]. The internal architecture of the sacrum has trabeculae whose density is lost in a systematic manner with the creation of an alar void in the region of S1 [61]. It is through this region that SIF occur, which is the target of the augmentation. SIF have been biomechanically created with vertical forces approximately 4.5 times body weight in zone 1 of the sacrum [52]. Treatment of SIF through cement injection has been shown to provide pain relief [55–57]; however, biomechanical testing has shown that neither stiffness nor strength is restored from the augmentation [62,63], which may be a contributory factor to refractory pain. Pain at the SIF may result from micro motion, and with the reduced healing potential from the osteoporosis delayed and nonunion may result. Even though cement augmentation is used, lower stiffness allowing painful motion may result in delayed pain relief and the development of chronic pain.

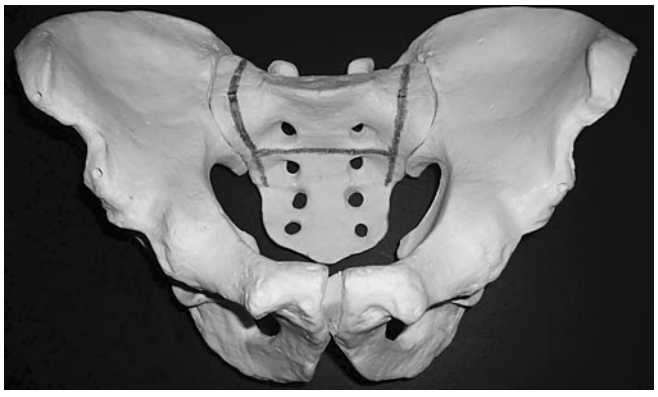

Figure 45.16 AP view of the pelvis showing the areas commonly involved in sacral insufficiency fractures

CONCLUSIONS

Osteoporosis is BMD < 2.5 standard deviations below peak value for young adults (T score); this is the most important determinant of vertebral strength. Osteoporosis occurs when there is either an increase in osteoclastic or a reduction in osteoblastic bone activity. Load is transmitted in the spine through both the trabecular core of the vertebra and the peripheral vertebral cortex. Osteoporosis predominantly affects the trabecular centrum while disc degeneration causes peripheral load transfer. The combination of these two effects concentrates anterior load transfer in flexion and may result in VCF when vertebral strength is exceeded. At the time of the initial VCF anterior load pathway shift, increased flexion moment and strain concentration may lead to subsequent VCF. SIF result from repetitive loading of compromised sacral alae leading to back and leg pain, which may be treated by sacroplasty.

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46 Percutaneous Vertebroplasty

Mary E. Jensen

INTRODUCTION

Percutaneous vertebral augmentation was conceived in 1984 when Deramond injected polymethylmethacrylate (PMMA) into a painful vertebral body hemangioma under fluoroscopic control [1]. "Vertebroplasty," as the procedure came to be known, is an image-guided therapeutic procedure involving the placement of a trocar into a vertebral body fracture or neoplasm, followed by the application of an acrylic polymer to provide improved compressive strength to the vertebral body. A similar procedure called kyphoplasty, also known as "balloon-assisted vertebroplasty" was developed shortly after the emergence of vertebroplasty and also gained popularity as a way of enhancing the strength of a compromised vertebra. Any percutaneous technique that attempts to achieve internal vertebral body stabilization through the introduction of a permanent implant should be considered "vertebral augmentation."

The evolution of vertebral augmentation procedures has dominated the growth of minimally invasive spine surgery for the past 10 years, resulting in improved devices and new techniques such as deployable grafting systems (OptiMesh, Spineology, St. Paul, MN) and permanent structural implants (StaXx Fracture Repair System, Spine Wave, Inc., Shelton, CT). In some ways, the technology has outpaced the science because prospective, randomized controlled trials comparing vertebral augmentation to best medical therapy have been published only recently (FREE trial, VERTOS II) [2,3]. At the same time, studies comparing vertebroplasty to a sham procedure [4] or control intervention [5] concluded that the pain relief achieved with vertebroplasty was no better than that achieved with the comparison procedure. This conundrum has yet to be explained, and is the focal point of the current debate surrounding the efficacy of vertebral augmentation procedures.

This chapter will describe the clinical and technical aspects of vertebral augmentation, with emphasis on the most commonly performed procedure—vertebroplasty—as it is used in the osteoporotic patient. This chapter also will summarize the current body of literature as it pertains to augmentation techniques, including the current controversies that surround them.

HISTORY OF VERTEBRAL BODY AUGMENTATION

In 1987, Deramond and Galibert [1] described the percutaneous application of acrylic polymer to vertebral body defects associated with painful hemangiomas, with resultant good control of pain. Small series followed, emphasizing the effects of vertebroplasty on hemangiomas or metastases [6–8]. The first report of its use in osteoporosis appeared in 1991, [9] where five patients suffering from painful osteoporotic vertebral compression fractures (VCFs) demonstrated complete, immediate relief of pain after vertebroplasty.

Vertebroplasty was virtually unknown in North America until 1993 when the concept was introduced at a lecture given at the American Society of Neuroradiology annual meeting. Dion and Jensen successfully treated the first patient in the United States at the University of Virginia later that year. The first paper focusing on the technical aspects of vertebroplasty was published in 1997 [10], and included 29 patients with 47 osteoporotic VCFs of whom 90% demonstrated pain relief as evidenced by their verbal expression of perceived pain and analgesic use. Deramond and colleagues [11] reported similar results in 80 patients treated for osteoporotic fractures, with rapid and complete relief of pain in more than 90% of cases. Follow-up of 1 month to 10 years showed prolonged analgesic effect and only a single complication was reported. The first open prospective study was not published until 1999 [12]; no control group was used and the follow-up period ended at 6 months.

Vertebroplasty was enthusiastically embraced by interventional radiologists and the elderly population that it served. Kyphoplasty was introduced in 2001 [13] as an alternative approach. Based on the positive outcomes seen with vertebroplasty, kyphoplasty was readily accepted as an alternative augmentation procedure, although embraced primarily by the surgical community. Despite the popularity of augmentation procedures, the reason for their efficacy remains obscure. The answer may be found in the effect that PMMA has upon the vertebral body, both mechanically and physiologically.

BIOMECHANICS OF VERTEBRAL BODY AUGMENTATION

The loss of substantive bone tissue from primary or secondary osteoporosis, tumor erosion, or osteonecrosis may lead to vertebral collapse when the axial load is more than the involved vertebral body can withstand. PMMA, an acrylic polymer noted for its excellent compressive strength, has long been used by spine surgeons for vertebral packing following tumor debulking [14–16]. During the PMMA preparation phase, liquid and powdered acrylic components are mixed together to create a "dough," which is then used to fill the surgically created void. The material cures in a matter of minutes to form a dense "cement."

Extensive research on PMMA as a suitable material for vertebroplasty was published early after initial clinical descriptions of its use. Biomechanical testing of PMMA injected into osteoporotic vertebral bodies demonstrated an increase by almost 200% in the force required to compress treated vertebrae when compared to an untreated control group [17]. Even when altered by the addition of opacification agents or antibiotic powders [18], or by changing the monomer to polymer ratio [19], the compressive strength easily surpassed that of an unadulterated osteoporotic vertebral body. When vertebrae are compressed past the point of initial failure, injected specimens are more likely to resist continued deformation than native vertebrae [20], thereby maintaining spinal axis alignment.

As to the mechanism of pain relief associated with vertebral augmentation, many theories have been proposed. The PMMA curing process is an exothermic reaction that generates significant heat, although thermal injury to adjacent neural, bony, and disc tissue has not been seen in animal models [21,22]. However, when PMMA is applied directly to tumor tissue, the acrylic causes necrosis at the PMMA-tumor interface, possibly from direct cytotoxic effects and/or heat-induced tissue coagulation [23,24]. Mechanical compression of the adjacent tissues by the PMMA or inflation of the bone tamponade used in kyphoplasty may disrupt nerve fibers running in the trabecular space. Stabilization of microfractures and decreased mechanical stresses placed upon the affected vertebrae may also play a role [25]. However, if this vertebral strengthening effect is the cause of the therapeutic response, one would expect to find the degree of pain relief to be proportional to the total amount of injected acrylic and the extent of vertebral filling. To date, there has been no correlation between pain relief and the volume of PMMA used [25,26], and the physiological basis for the analgesic effect seen with vertebroplasty continues to defy explanation.

CLINICAL ASPECTS OF VERTEBROPLASTY

In 1995, an estimated 700,000 VCFs occurred in elderly individuals, affecting 25% of postmenopausal women [27]. Because there is no universally accepted definition of a compression fracture, the prevalence of fractures could vary by up to threefold [28]. Its prevalence steadily increases with increasing age in both sexes, and the life-time risk of a clinically diagnosed fracture is 16% in white women, as compared to 5% in white men [27]. Individuals with fractures have a higher mortality rate than their nonaffected counterparts, particularly in the first 5 years following fracture, with men more likely to die than women [29]. VCFs lead to a substantial decline in the individual's quality of life (QOL), increase their risk for subsequent vertebral and nonvertebral fractures, and often lead to further disability. Clearly, osteoporosis of the spine and its clinical consequences are important health care issues that deserve attention.

Clinical Picture

Osteoporotic VCFs are most likely to occur in postmenopausal Caucasian and Asian women [27]. Age-related bone loss is the most common cause and each standard deviation decrease in lumbar spine bone mineral density is associated with a two-fold risk of fracture [28]. A personal history of fracture is one of the strongest clinical predictors of further occurrences, increasing the likelihood of new fractures by five-fold [31]. Certain conditions are associated with the development of osteoporosis in 20% of women and 40% of men presenting with vertebral or hip fractures including steroid therapy, early oophorectomy, hypogonadism in men, hyperparathyroidism, chronic obstructive pulmonary disease, immobility, anticonvulsant use, smoking, and alcohol consumption [30].

A vertebral fracture may be defined as reduction in vertebral height by 15% or more [32], or classified by degree and type of deformity (wedge, biconcavity, or compression) [33]. The lower thoracic and lumbar vertebrae are most commonly involved, especially T8, where the physiologic thoracic kyphosis places the greatest axial load, and the thoracolumbar spinal junction, where mobility changes between the relatively restricted thoracic spine and the more freely moving lumbar vertebrae [34,35].

Most fractures occur spontaneously or are associated with routine, everyday activities; only 25% are associated with falls [28]. Although many fractures are asymptomatic, 84% of patients with VCFs have some degree of pain [32]. Pain is often described as intense and deep, localized to the level of the involved vertebra, and exacerbated by palpation or percussion over the affected spinous process [32,35]. Pain may radiate to the flanks or anteriorly along the ribs, and may be referred as far away as four levels from the fracture site [35]. Pain is usually position-dependent and often exacerbated with weight-bearing or lifting and relieved with lying supine. Radicular pain involving the legs is uncommon [35].

Affected patients often experience a substantial decline in their functional status and QOL after a fracture. Elderly women with symptomatic VCFs demonstrate significant performance impairment in physical, functional, and psychosocial testing when compared to a control group with no fractures [36]. Complications associated with VCF and the immobilization used in its treatment include loss of bone and muscle mass; diminished cardiac output; atelectasis and pneumonia; constipation and early satiety; deep venous thrombosis and pulmonary embolism; pressure sores; urinary tract infections and renal calculus formation; low self-esteem, depression, and loss

of independence; and increased admissions to nursing homes and mortality [37–39].

Pain associated with VCFs is usually self-limiting, lasting from 2 weeks to 3 months. For this reason, treatment of acute fractures has been largely conservative, with current therapy emphasizing pain control with maximal conservative therapy [40]. Narcotic and/or anti-inflammatory medications, prolonged bedrest, trigger point injections, heating pads, ice packs, or massage therapy or trigger point injections may be useful. Other therapies such as back bracing, physical therapy, and low-intensity exercise are introduced once the patient is capable of bearing weight. Preventative medical therapy (bisphosphonates, calcitonin and in some instances, hormonal replacement therapy [HRT]) is encouraged to prevent new fractures. *Teriparatide*, a recombinant form of parathyroid hormone, has been shown to reduce the risk of new or worsening back pain in patients randomized to placebo, HRT, or alendronate [41]. Calcium, vitamin D, and vitamin D analogues have shown no statistically significant effect on vertebral fractures or deformity [40]. Traditional open surgery with internal fixation is rarely indicated and reserved for the patient with gross deformity, instability, or neurologic deficits [42].

Patient Selection Criteria

Given the significant and potentially fatal sequelae of prolonged immobilization, it is appropriate to consider vertebral augmentation when a patient is not responsive to maximum medical management. Most patients show significant progress or make a full recovery within 6 to 12 weeks after the incident event [40]; therefore, augmentation is directed toward affected patients who have tried a reasonable time course of conservative therapy and are still plagued by pain or are incapable of ambulation.

The American College of Radiology (ACR) has written practice guidelines for the performance of vertebral augmentation, most recently updated in 2010, and selection criteria are outlined in detail [43]. In short, appropriate candidates include patients with symptomatic osteoporotic VCF or a vertebral body weakened by neoplastic disease, where pain is refractory to medical therapy. "Failure of treatment" is defined as "minimal or no pain relief with the administration of prescribed analgesics, or adequate pain relief with narcotic dosages that produce undesirable side effects (excessive and intolerable sedation, confusion, or constipation)."

Absolute contraindications are few and include asymptomatic VCFs, osteomyelitis of the target vertebra, uncorrected coagulopathy, or allergic reaction to acrylic compounds or opacification agents. There is no evidence to support prophylactic vertebroplasty in osteopenic patients with no acute fracture. Relative contraindications are less well-defined and often operator-specific. Patients with significant spinal canal compromise from retropulsed fragments or epidural tumor extension, or radiculopathy caused by a compressive lesion unrelated to the fracture; patients improving on medical therapy may not be candidates for augmentation. Patients with systemic infections should be approached with caution, and use of prophylactic antibiotic therapy is prudent.

Patient Screening and Evaluation

Potential candidates for treatment should fulfill appropriate clinical and radiologic criteria before vertebral augmentation is offered, and much of this information can be obtained prior to the clinic visit. Requiring a referral from an individual's primary care physician helps eliminate inappropriate patients who attempt to self-refer. A clinical coordinator, such as a nurse or experienced assistant, can collect the patient's "pain" history, other relevant medical conditions or previous surgeries, current analgesic use, and radiologic studies, before scheduling an appointment. In many cases, non-candidates are discovered early on and can be redirected.

History of Present Illness

A detailed history is obtained, focusing on the location, duration, quality, and degree of back pain; current mobility; analgesic dose and schedule, and overall medical condition. Presenting symptoms, indications for the procedure, pertinent medical and surgical history, a list of current medications (including steroids and osteoporosis medications), history of allergies, and evidence of failed medical therapy is documented. Use of Visual Analog Scales (VAS) and/or standardized pain questionnaires for determining pain level and character, and dermatome drawings for pain localization help with data collection and make comparison with the treatment results more exact.

Patients with atypical back pain should be evaluated for a concomitant disease process such as systemic infection, gastric ulcer, or other diseases that may present as back pain. Any condition that results in bacteremia, for example, urinary tract infection, may seed the spinal column resulting in discitis or epidural abscess. A high level of suspicion for infection is required because elderly patients may not mount a vigorous immune response, resulting in an afebrile individual with nonspecific findings and a normal or mildly elevated blood cell count and sedimentation rate [44].

Neurological and Physical Examination

On physical examination, the affected vertebra may be sensitive to pressure over the spinous process, but a lack of focal tenderness does not preclude clinical success of vertebroplasty [45,46]. Patients with diffuse or nonfocal pain, low back pain that radiates to the hip or iliac crest, or lumbar radiculopathy may suffer from facet arthropathy, or a radicular or spinal cord compressive syndrome, which should first be excluded by neurological examination and imaging. Note should be made of medical conditions that may influence how the procedure is performed. For example, patients with pulmonary compromise may find lying prone results in severe respiratory distress, necessitating the use of anesthesia or vertebral access with the patient in the decubitus position.

Radiologic Evaluation

Osteoporotic postmenopausal women with a documented new or subacute fracture on a conventional radiograph

and who meet the clinical criteria for VCF can proceed to vertebral augmentation without further imaging. However, adjunctive studies, particularly magnetic resonance imaging (MRI), are much more sensitive in detecting acute fractures and marrow replacement diseases, and often identify other vertebral fractures that do not exhibit loss of height on the plain film [47].

MRI (Figure 46.1) and bone scintigraphy (Figure 46.2C–D) can identify active fractures [47,48] and predict treatment outcome [49–51]. A limited MR study consisting of T1 and STIR (short-tau inversion recovery) sagittal images may be the only study needed to spot vertebral body edema. Although MRI is sensitive for the detection of acute compression fractures, the duration of vertebral body edema with respect to the presence of pain is unknown. Tanagawa et al. [49] demonstrated a significantly greater clinical response to vertebroplasty in patients with extensive bone marrow edema than in those without edema.

In patients suspected of having active VCFs with no obvious acute fracture on MR, bone scintigraphy may be positive. Selection of treatment sites by preoperative bone scintigraphy has been shown to be an excellent predictor of clinic response in both vertebroplasty and kyphoplasty [50,51]. One pitfall of bone scanning is that activity in facet arthropathy may be confused with activity in a severely collapsed vertebral body on a routine scan. SPECT scanning (Figure 46.2D) localizes the tracer uptake within the vertebral body or the adjacent facet joints.

Computed tomography (CT) plays a role in patients with complex or severe fractures, or marrow replacement disease. CT excels at bone imaging and is used to evaluate the integrity of the posterior wall of the vertebral body, locate fracture lines involving the vertebral body and pedicles, and assess posterior displacement of fragments (Figure 46.2B).

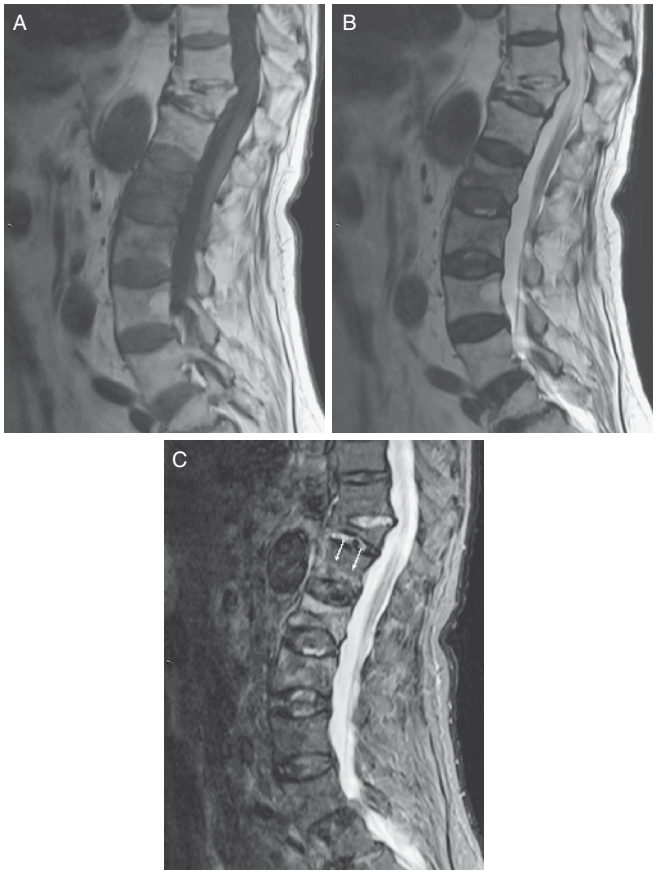

Figure 46.1 T1-weighted sagittal image (**A**) shows low signal intensity involving the L1 and L2 vertebral bodies, in addition to the inferior endplate of T12. The corresponding areas on the T2-weighted image (**B**) show mild hyperintensity, which is inhomogeneous. STIR sequence (**C**) clearly identifies edema at all three levels, especially at T12 (arrows). Also noted are old compression fractures of T11 and L5, and a hemangioma in the posterior aspect of L3.

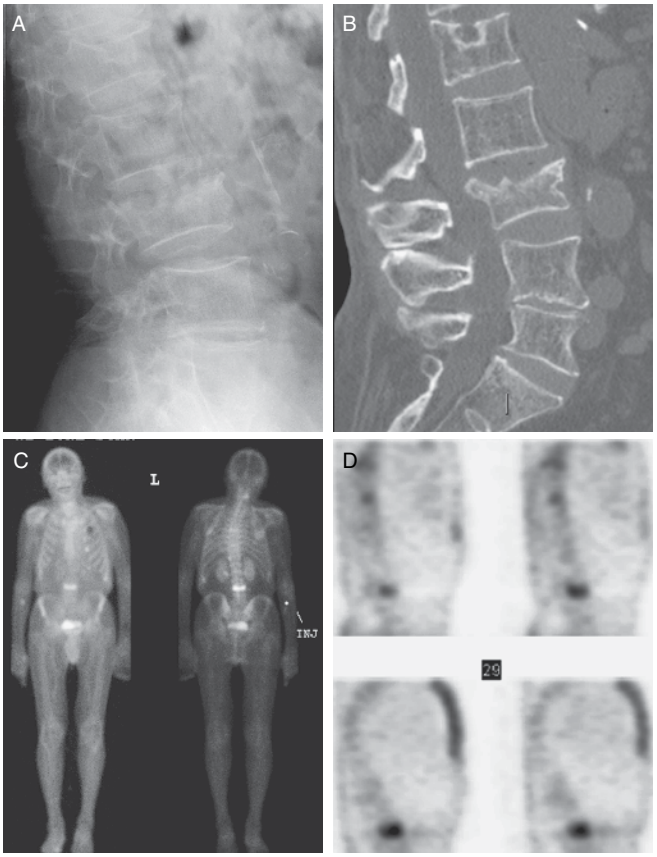

Figure 46.2 This elderly woman presented with sudden onset of new back pain and was noted to have multiple lumbar vertebral body deformities on plain film (**A**). CT scan **(B)** shows an L1 Schmorl's node, a superior endplate compression fracture of L3 with retropulsed fragment, and irregularity of the L4 inferior endplate. Bone scan images (**C**) show increased tracer activity throughout the whole vertebral body at L3, in addition to mild uptake in several thoracic vertebrae, and a right midcervical circular focus consistent with degenerative facet disease. Sagittal SPECT scan (**D**) shows the L3 uptake confined to the vertebral body, with variable degrees of uptake at two thoracic vertebral bodies.

In ambiguous cases, adjuvant imaging may discover another cause of back pain such as disc herniation or spinal stenosis. Facet arthropathy is a common cause of low back pain that radiates to the hip. When the clinical examination and imaging suggest facet disease as the most likely pain generator, a diagnostic facet injection can be performed first as part of the screening process rather than proceeding directly to augmentation.

Preprocedure Preparation and Counseling

Vertebral augmentation, and in particular vertebroplasty, is usually performed on an outpatient basis. Preprocedure instructions should be clearly outlined at the clinic visit. Patients are asked to remain NPO after midnight, avoid taking their morning analgesics, and have a designated driver available to transport them home. Informed consent is obtained at the clinic visit or on the day of the procedure. Risks cited should include infection, bleeding, fracture of the pedicle or vertebral body, extravasation of acrylic into the surrounding epidural or paravertebral veins resulting in worsening pain or paralysis, pulmonary compromise, and death. The potential need for immediate surgical intervention should be discussed.

Elderly patients often have chronic conditions that require special consideration. When indicated, preprocedure laboratory testing is done and often includes hemoglobin, hematocrit, electrolytes, coagulation parameters, complete blood count (CBC) with differential, and sedimentation rate. Vertebral augmentation should be avoided in patients with known infections, fevers, or elevated white blood count (unless due to steroid use) until the source has been discovered and treated [52]. Individuals taking coumadin can be converted to subcutaneous enoxaparin (Lovenox) on an outpatient basis, and restarted on coumadin after the procedure. This process eliminates the need for a lengthy hospitalization but requires coordination with the patient's primary care physician.

In most patients, sedation can be achieved with fentanyl and midazolam. Uncooperative patients, patients in extreme pain, or individuals with chronic respiratory disease who may have difficulty ventilating while lying prone, may require deep or general anesthesia. If anesthesia services are anticipated, the patient should have a preanesthesia evaluation at the time of the clinic visit.

TECHNICAL ASPECTS OF VERTEBRAL AUGMENTATION

Different vertebroplasty techniques have evolved based on the predominant European [11,53,54] and North American [10,55–57] experiences. Descriptions of the procedure are plentiful, with variations related to the operators' training and personal preference, or the availability of the products and equipment used. However, there is no substitute for a "hands-on" experience, and interested operators are strongly encouraged to take advantage of the multiple courses that offer laboratory training in addition to didactic teaching.

Equipment Requirements and Operator Skills

Vertebral augmentation has been described using standard fluoroscopy [10,11,53], CT guidance [6,58,59] or CT fluoroscopy [60] for trocar positioning, followed by acrylic injection under fluoroscopic observation, or a combination of lateral fluoroscopy and CT fluoroscopy [61]. Operators should strive to use the highest quality fluoroscopy available. A biplane digital angiography unit is ideal as procedural time is substantially reduced through the simultaneous projection of orthogonal views during trocar placement and acrylic injection. However, a high quality single plane unit alone will suffice, but low quality portable units should be avoided as the image quality is usually too poor for adequate visualization of bony landmarks and acrylic flow.

In addition to a high quality imaging chain, the operator should possess appropriate cognitive and technical skills to ensure quality and safety of the study. These skills include, but are not limited to, knowledge of the radiographic anatomy of the spine and associated structures on both CT and fluoroscopy; appropriate operation of fluoroscopic equipment; formal training in the principles of radiation safety including techniques to minimize exposure to self and patient; skill in CT or fluoroscopic-guided biopsy procedures of the spine, including radiographic triangulation; and experience with embolization techniques.

Patient Preparation and Monitoring

Vertebral augmentation procedures require a team of individuals committed to the care of the patient. A dedicated nurse or other trained professional whose primary responsibility is to establish and maintain venous access, administer conscious sedation, monitor the patient's physiologic status, and maintain the medical record should be present throughout the procedure. Patients with respiratory compromise may require supplemental oxygen or anesthesia support during the procedure. Equipment and medications for emergency resuscitation should be immediately available. Secondly, a radiologic technologist who is responsible for patient positioning and preparation; management of the equipment, devices, and implants used in the procedure; and proper recordation of the procedure through spot imaging or fluoroscopy loops should be in the room or immediately available. Lastly, the physician operator oversees the actions of all team members, in addition to performing all key elements of the procedure.

The patient is placed prone on the angiography table, and physiological monitors including electrocardiogram (EKG) leads, pulse oximeter, and blood pressure cuff are attached, in addition to oxygen via nasal cannula. For anxious patients concerned about pain when moving onto the table, 25 to 50 μg of fentanyl (Sublimaze, Abbott Labs, North Chicago, IL) can be administered 5 minutes before positioning. Additional conscious sedation is given as needed, using small increments of fentanyl and midazolam (Versed, Roche Pharma, Manati, PR).

The procedure is performed under strict sterile conditions with all persons in the room wearing surgical caps and masks, in addition to sterile gowns and gloves for the

operators and assistants setting up the tray. The level to be treated is identified and marked under fluoroscopy and the overlying skin surface is sterilely prepped and draped. The image intensifiers are also covered with sterile bags to prevent contamination of the operator and the devices. Although there is no clear consensus on prophylactic antibiotic therapy in vertebral augmentation, many authors advocate its use particularly in immunocompromised patients to prevent osteomyelitis [62]. Antibiotics either given intravenously and/or mixed with the acrylic polymer, have been advocated [10,11,52,55–57].

Pedicle Targeting

The pedicle to be punctured is isolated under anteroposterior (AP) fluoroscopy. In the simple "bulls-eye" approach to the pedicle, the fluoroscopic tube is either in a straight AP position, or slightly obliqued (Figure 46.3). In this approach, the largest surface area of the pedicle is presented for targeting and its entire cortical circumference is easily seen. Advancement of the trocar positions the tip in the midportion of the ipsilateral vertebral hemisphere. If holovertebral filling is desired, a contralateral puncture will usually be necessary unless a trocar with a curved inner cannula is utilized (Figure 46.4).

Puncture of the pedicle using the more oblique, "scotty-dog" view (approximately 20° to 30° of ipsilateral angulation), will result in a steeper lateral-to-medial trocar track, with the final trocar position near the midline of the vertebral body (Figure 46.5). From this location, it is more likely that a single transpediculate injection will fill the majority of the vertebral body. This approach is more challenging technically, particularly in the thoracic spine where the pediculate surface area is smaller and its cortex is not as visible. Trocars positioned too laterally may fracture the transverse process or puncture the lung. For gracile or hourglass-shaped pedicles, a steep oblique approach with the trocar positioned between the pedicle and the rib head may be used. The advantages of the unipediculate approach include shortened procedure time, diminished risk as only one pedicle is punctured, and better visualization as in-dwelling opacified acrylic is not present.

With either approach, the trocar track should avoid the medial and inferior borders of the pedicles, where cortical breaches can result in entry into the spinal canal or neural foramen.

Once the angle of approach is determined, the skin, subcutaneous soft tissues, and pediculate periosteum are anesthetized with 7 to 10 cc of bupivacaine hydrochloride, 0.25%; (Abbott Labs, North Chicago, IL), using a 2-inch, 25-gauge spinal needle. Before removing this needle, AP and lateral fluoroscopy should show the tip of the needle approximating the same location on the pedicle in the superior-inferior plane. If there is a discrepancy between the two, and the patient is in the true lateral position, then the AP tube needs to be adjusted in either the cranial or caudal direction until the needle tip approximates the same location as on the lateral view. A small skin incision is made with a No. 11 scalpel blade to allow easy passage of the vertebroplasty needle.

Positioning of the Trocar

A variety of disposable trocars are available for vertebroplasty use, and there are no studies on comparison of performance among the different products that might guide selection. These devices are generally listed as "bone biopsy" needles, consisting of an outer cannula and an inner stylet. Most vertebroplasty procedures use trocars ranging in size from 10-gauge to 13-gauge, because injection of acrylic is difficult through smaller gauge needles. Important trocar features include the availability of different stylet tip shapes and cannula sizes and lengths, radiolucency of the handle, "locking" of the stylet within the cannula, and compatibility of the cannula Luer-lock hub with various injection devices and injectable implants. Kyphoplasty requires dedicated trocars that will accept the drilling tool and balloon tamp, and all necessary components come prepackaged in one kit. In fact, with the exception of vertebroplasty, augmentation devices are

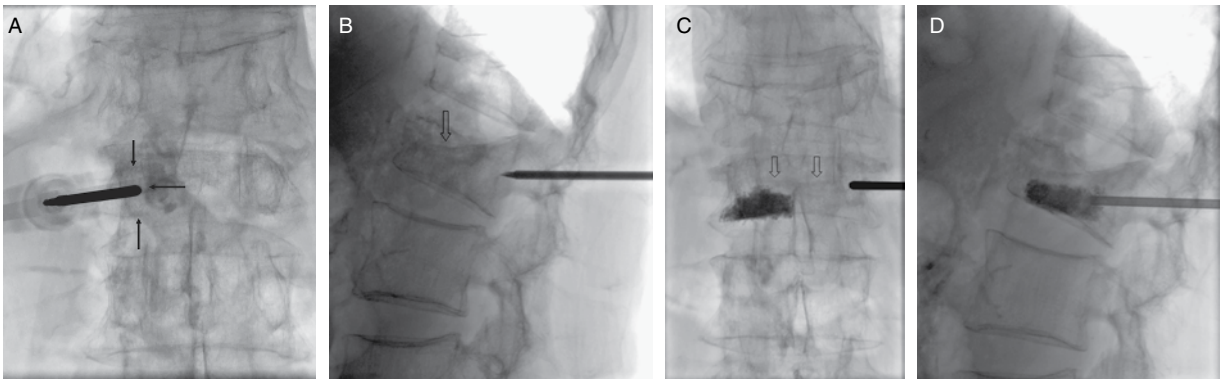

Figure 46.3 The straight or slightly oblique view is often used in thoracic vertebroplasty because of the true posterior orientation of the pedicles. The pedicle outline (**A**, arrows) is difficult to see because of the overlapping trocar handle. On the lateral view, the orientation of the trocar is parallel to the depth of the central superior endplate fracture (**B**,**C**, open arrow) to prevent transgression of the endplate. The PMMA flow was restricted to the left hemivertebra (**C**,**D**), necessitating a contralateral puncture (**C**).

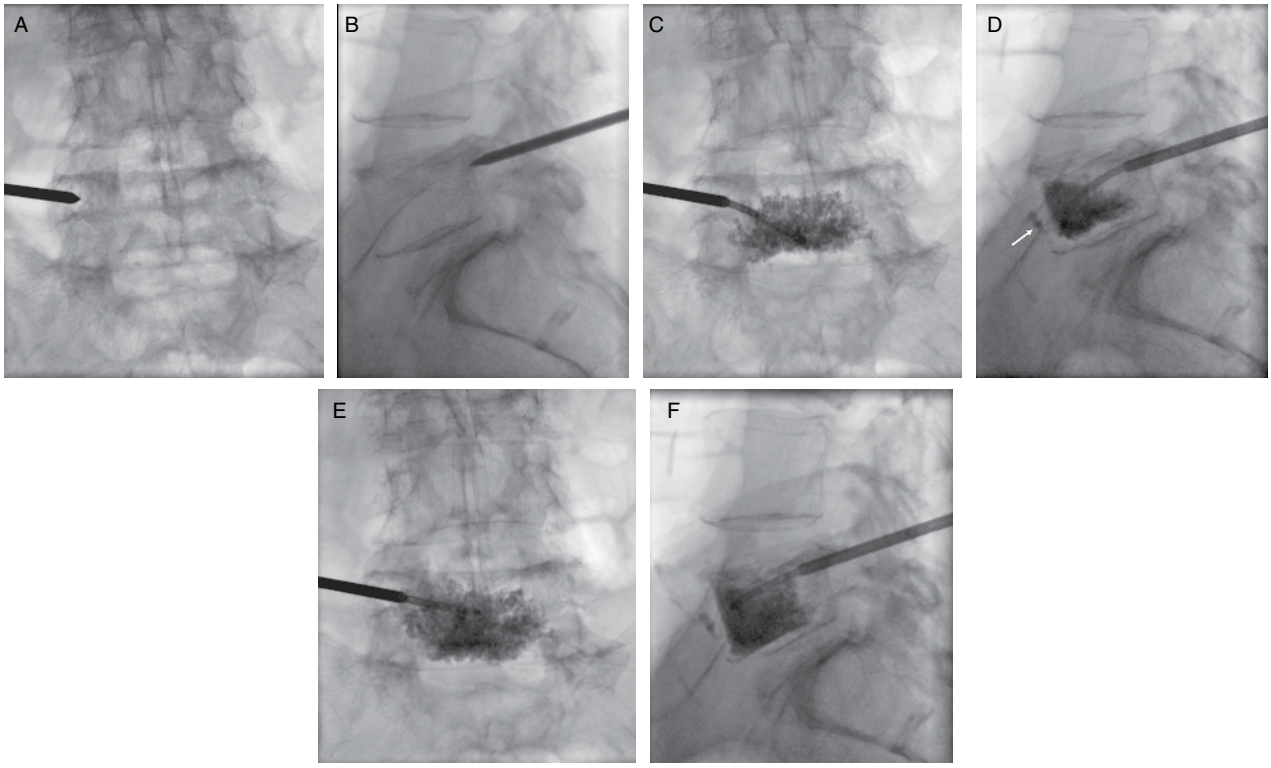

Figure 46.4 Vertebroplasty of the last lumbar vertebra can be difficult because of the oblique angle of the pedicles and the long trajectory required for placement of the trocar in the midline. In this case, the trocar is placed through the pedicle with its tip at the posterior aspect of the vertebral body (**A**,**B**). A curved cannula is advanced across the midline, and the anterior inferior portion of the vertebral body is filled (**C**,**D**). Note the small amount of PMMA in the paravertebral vein anteriorly (**D**, arrow). The cannula is repositioned more superiorly and the remaining trabecular space is injected, with complete filling of the anterior two-thirds of the vertebra **(E,F)**.

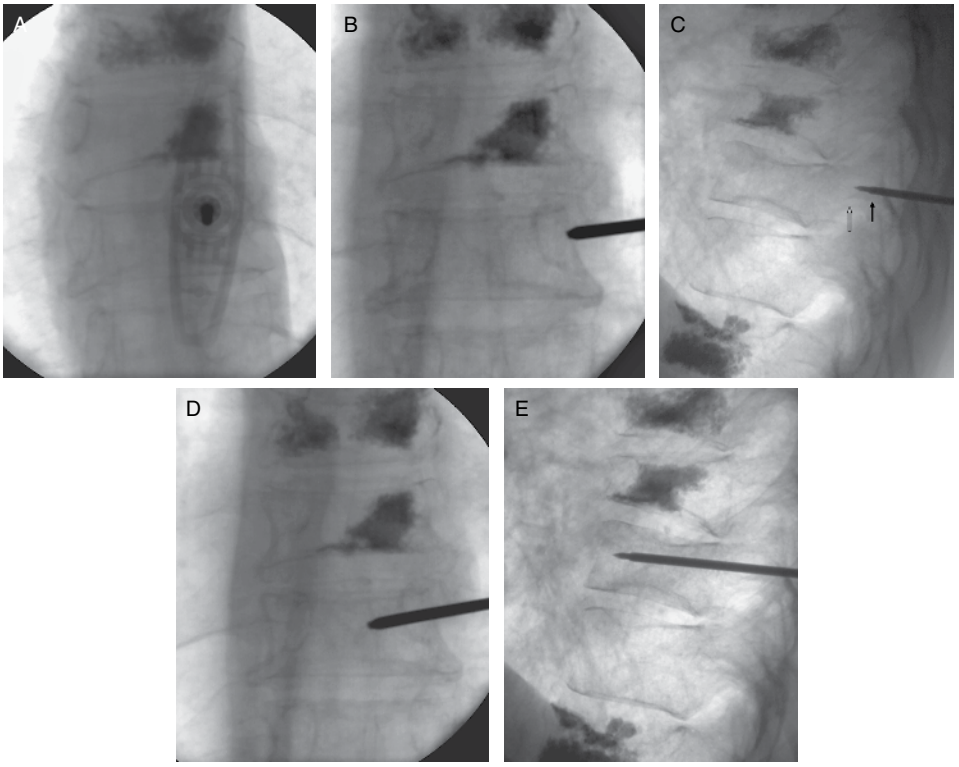

Figure 46.5 With the tube in an obliqued position, the mid to lateral portion of the pedicle is targeted (**A**). When the tube is turned straight AP, the trocar tip located in the lateral aspect of the pedicle will be seen to march across the face of it from lateral to medial, as it is advanced (**B**). On the lateral view (**C**), the trocar is angled to parallel the superior endplate, and enters the pedicle at the level of the undersurface of the rib head (arrow). This shadow is not to be confused with the undersurface of the pedicle (open arrow). The trocar tip ends in the midline (**D**) and anterior third of the vertebral body (**E**), an excellent location for a central fill.

sold as specialty kits with all of the necessary components included, and improvisation is not recommended.

The trocar is advanced until the stylet tip abuts the cortical surface in the superior to midpoint portion of the pedicle. Depending on the shape of the pedicle, the trocar should enter at the widest point, away from the medial and inferior borders. The angle of approach on the lateral view is determined by the degree of endplate compression or anterior wedging (Figures 46.3B and 5C). Often the course of the trocar will parallel that of the superior endplate, in which case the stylet tip position will begin more superiorly on the pedicle. On the AP view, the trocar should traverse the pedicle and vertebral body from lateral to medial (Figure 46.5B,D); otherwise it may abut or exit the lateral wall of the vertebral body. Likewise, the medial pedicle border marks the point of entry into the spinal canal. As the trocar passes this border on the AP view, the stylet tip should be in the vertebral body proper (Figure 46.6). Otherwise the pedicular wall may have been breached and the trocar may have passed through the spinal canal. This position may lead to leakage of PMMA into the canal after removal of the cannula.

The stylet tip of the trocar is positioned precisely before a cortical break is made, because once the track is started repositioning becomes difficult as the stylet has a tendency to slide into the initial divot. Exact positioning is best done with a diamond-point stylet, as beveled stylets have a tendency to slip off the pedicle. A slight back-and-forth twisting motion is used to advance the tip through the cortex, with frequent fluoroscopic checks in both the AP and lateral planes as the trocar traverses the pedicle. Alternatively, a small sterile orthopedic hammer can be used to tap gently on the needle handle, advancing the tip in small increments. Once within the trabecular bone, less pressure is required to advance the trocar and care must be taken not to pierce the endplates or vertebral wall. Use of the single-bevel stylet will allow deflection of the needle tip in the direction opposite to the bevel, allowing minor adjustments in either plane (Figure 46.7). The trocar is advanced using continuous or intermittent lateral fluoroscopy until the stylet tip is placed in the anterior one-third to one-quarter of the vertebral body. The closer the tip is to the midline on the AP view, the further anterior it may be positioned on the lateral view. Because the stylet tip projects beyond the end of the cannula, the final cannula tip position will be slightly more posterior.

When using a trocar system with a curved stylet (Figure 46.4), the cannula tip is placed in the posterior

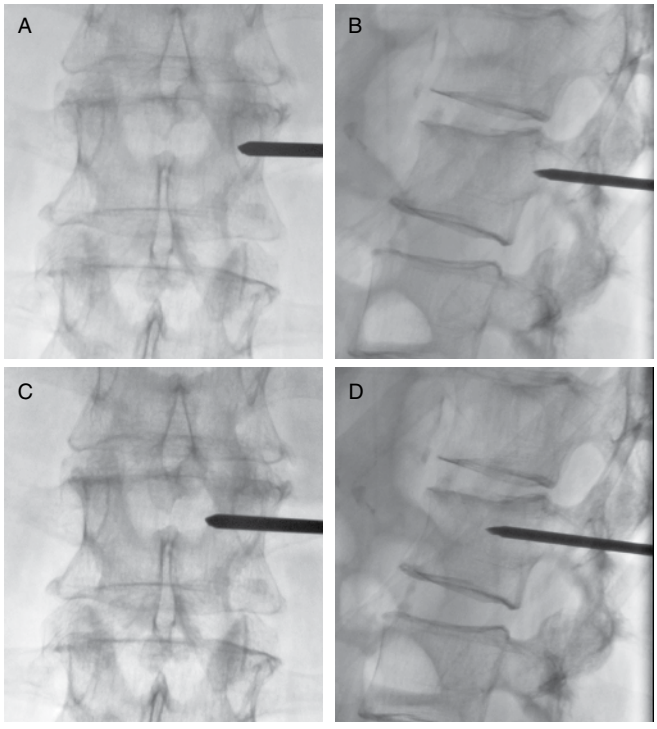

Figure 46.6 As the trocar is advanced through the pedicle, the stylet tip approaches the medial pedicle wall on the AP view (**A**) and the posterior vertebral body wall on the lateral view (**B**). Once the tip has passed across the medial border (**C**), the trocar should be in the vertebral body (**D**). If not, the trocar has passed through the spinal canal, which could result in thecal sac injury or decompression of PMMA into the canal.

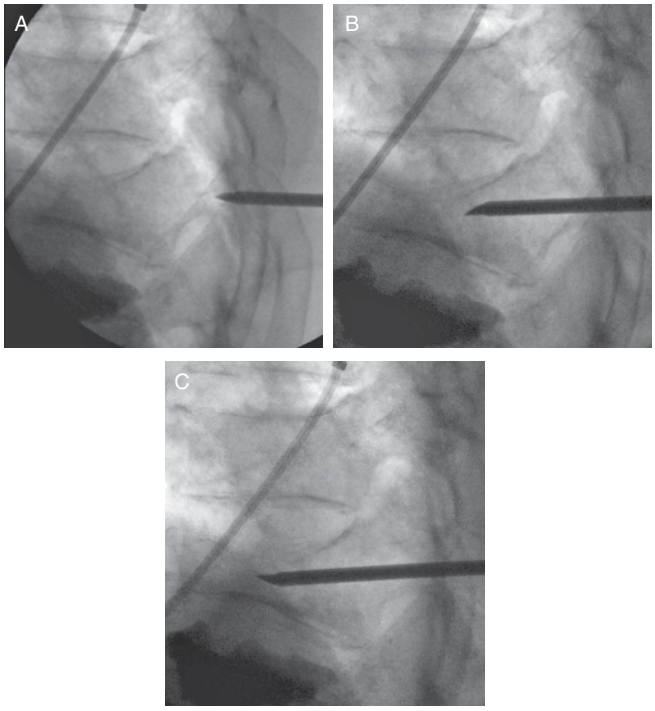

Figure 46.7 The trocar is initially positioned on the posterior pedicle surface using the diamond-tipped stylet (**A**). Once the trocar reaches the posterior wall of the vertebral body, the beveled stylet is inserted and advanced. Initially the beveled surface is pointing superiorly to push the trocar inferiorly around the concave superior endplate (**B**). Once past the depth of the endplate, the trocar is rotated 180° so the beveled surface is pointing inferiorly (**C**), to push the trocar away from the inferior endplate as it is advanced into the anterior aspect of the vertebral body.

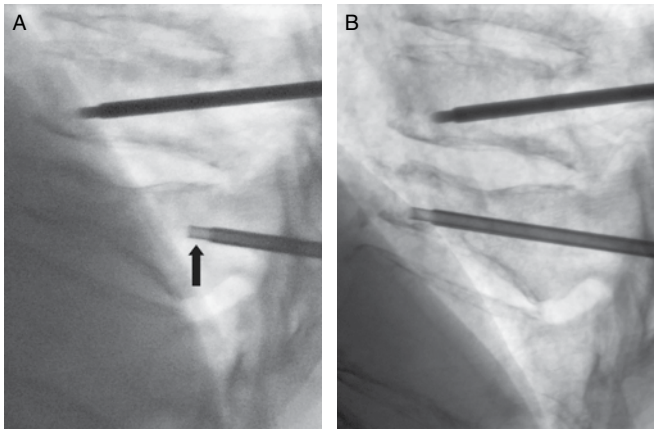

Figure 46.8 In cases of suspected malignancy, a core biopsy can be taken prior to PMMA injection using a special coring cannula (**A**, arrow). The stylet is replaced with the coring cannula after the trocar has been advanced to the posterior wall of the vertebral body. The trocar is then advanced to the anterior third of the vertebral body, **(B)** the coring cannula is removed with the aid of an aspiration syringe, and the tissue core is retrieved.

portion of the vertebral body to allow adequate movement of the curved stylet in all three planes. Positioning the cannula too close to the cortical surface may result in inability to advance the curved stylet, or breaching of the cortical surface. Similarly, if a biopsy of the vertebral body is needed, the cannula is positioned posteriorly and biopsy needles can be placed through the cannula for tissue acquisition. Alternatively, a trocar set with a stylet that is replaced with a coring cannula can be used (Figure 46.8; Core-Assure, Parallax Medical, Inc., Scotts Valley, CA). A core biopsy is taken while advancing the system into position for acrylic injection.

Placement of a Contralateral Trocar

Many experienced practitioners position one trocar in the midportion of the vertebral body and perform only a single injection of acrylic, filling the midportion of the body (Figure 46.9). Some operators prefer to fill the entire vertebra at a single sitting, and will place a second trocar in the contralateral hemivertebra if the initial fill pattern is deemed unsatisfactory or incomplete. Others prefer to place both trocars first so both hemivertebrae can be treated with a single aliquot of acrylic material. In this situation, the contralateral stylet is left in place during the initial ipsilateral acrylic injection; otherwise, the material will track through the trabecular space and egress out the contralateral cannula. The first cannula may be removed prior to injection of the second hemivertebra. Another potential problem is the obscuration of the basivertebral plexus during injection by overlapping trocars, which is solved by placing the second trocar after completion of the first injection. Visualization around the single trocar is easily done by changing the lateral obliquity. Another technical difficulty is observing acrylic flow during contralateral injection because of the presence of PMMA in the ipsilateral hemisphere. Strategies include adding extra barium sulfate to the acrylic mixture used during the contralateral injection so it is seen through the ipsilateral acrylic cast; using final images of the ipsilateral injection as a guide by looking for acrylic extending outside of the existing cast; or injecting under a combination of lateral and AP oblique views. Use of roadmapping technique is not advised as respiratory and bowel gas movement makes precise visualization impossible.

Vertebrography

The initial technical description of vertebroplasty [10] advocated the use of vertebrography prior to acrylic injection to confirm the cannula location within the trabecular space, define the location of the basivertebral plexus and evaluate potential routes of acrylic extravasation. Controversy exists over the need for vertebrography, particularly in the hands of experienced practitioners [63,64] and studies have shown no defined safety benefit [64,65]. Therefore, vertebrography has, for the most part, been abandoned as a recommended part of the procedure.

Acrylic Preparation

The most commonly used material for vertebral augmentation is PMMA. This injectable acrylic is created from two components—a powdered polymer that may or may not contain adequate opacification agent, and a liquid monomer. When the two substances are combined, a chemical reaction begins that leads to progressive polymerization of the mixture to its solid state. There are several commercially available PMMA products, all with different handling characteristics. The injectable products are distinctly different from the "doughy" material used in cranioplasty or joint replacements; therefore, operators should be familiar with the distinct properties of injectable PMMA before treating patients. Bench testing and trial injections in vertebral models or cadavers, either through laboratory testing or a formal course, is the best way to familiarize oneself with the nuances of the material to be used.

The major characteristics of PMMA that impact its use in vertebral augmentation are polymerization time and opacification. The polymerization time, or curing rate, varies among the different products, and the prepared material may be suitable for injection from as little as 5 minutes to close to 20 minutes. The polymerization time of any PMMA can be prolonged by refrigerating its components prior to their use, or by wrapping syringes filled with the acrylic in a sterile glove filled with ice. For acrylics with longer curing times, the powdered polymer component needs to dissolve completely in the liquid monomer before injection. Otherwise, the powder may separate from the monomer in the trocar during the injection and plug the cannula. The addition of a "rest period" of approximately 1 minute after mixing and before injection is recommended by some manufacturers.

The second parameter of great significance is opacification. Because most clinically relevant complications are

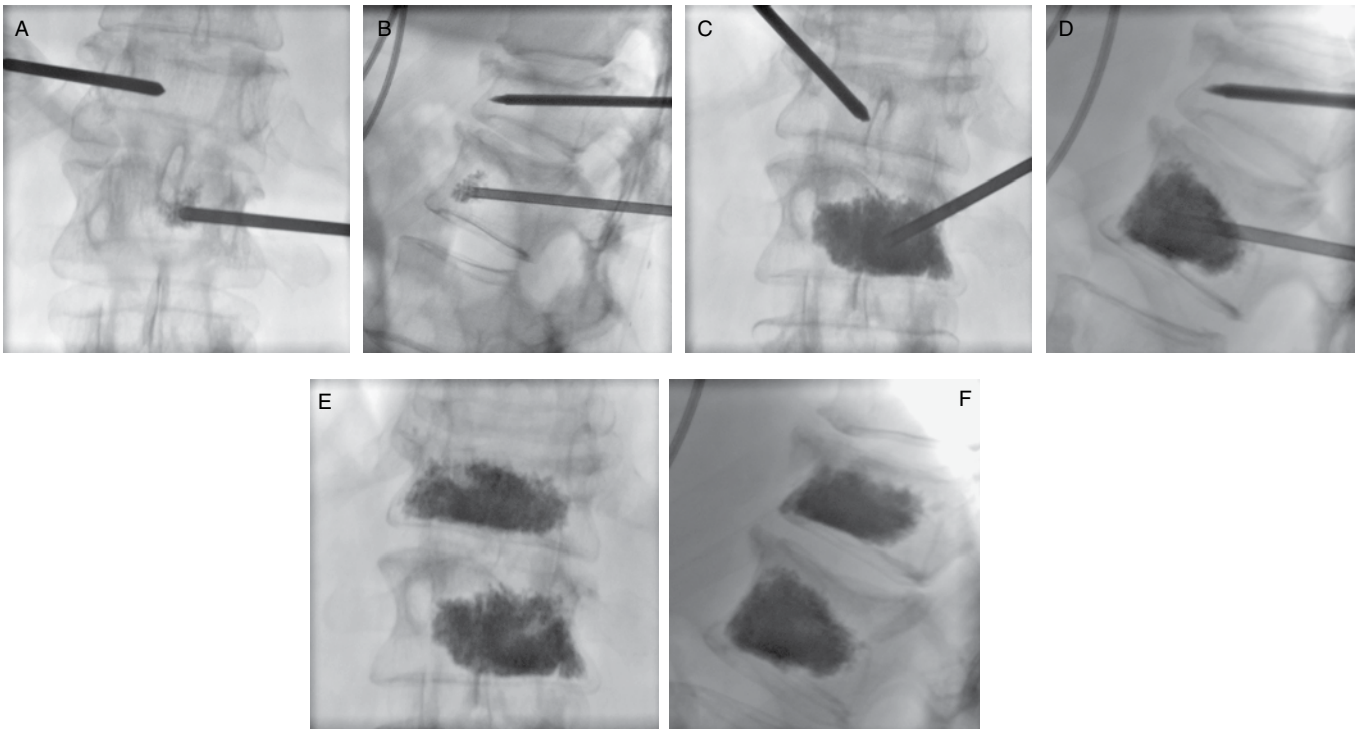

Figure 46.9 Adjacent thoracolumbar vertebral compression fractures were treated at one sitting. Both needles were placed followed by sequential PMMA injection. Note the cloud-like, frothy pattern of trabecular filling (**A**,**B**), which becomes more coalescent as the injection progresses (**C**,**D**). The embolization is terminated (**E**,**F**) when the PMMA reaches the posterior quarter of the vertebral body.

caused by the migration of PMMA into the extraosseous spaces, fluoroscopic visualization of the material during injection is of overriding importance. Visualization is influenced by the amount of barium sulfate within the product, the quality of the imaging chain, the size of the patient, and the location of the target vertebral body. Therefore, all practitioners must be familiar with the opacification traits of their chosen PMMA and should be prepared to supplement their mix with extra barium sulfate if necessary. Sterile barium sulfate is commercially available, and additional material should be thoroughly mixed with the powdered polymer prior to addition of the monomer to guarantee homogenous opacification.

Antibiotic powders for infection prophylaxis, such as tobramycin or vancomycin, also may be added to the powdered polymer. A recent study has shown that the addition of antibiotic powders does not affect the injection characteristics or compressive strength of the material [66]. Readers are advised that any alteration of the manufacturers' product or mixing instructions may change the consistency and/or polymerization time of the material, and the "modified" acrylic is no longer FDA-approved.

Acrylic Injection

Acrylic is delivered either by specially designed delivery systems or with 1-cc syringes. In kyphoplasty, the acrylic is loaded into the delivery cannula and pushed into the created cavity with a tamping device. There are several advantages and disadvantages to each method. Commercially available injection devices are self-contained systems, with a reservoir into which the PMMA is loaded and a twist-type or trigger-activated plunger, which advances the material. The system is attached to the cannula hub via high-pressure tubing. Each turn of the plunger or pull on the trigger delivers a consistent amount of acrylic into the cannula. Injection devices increase the distance between the operator and the x-ray tube, thus minimizing the dose to the hands especially in the AP plane [67]. Only a single connection of the injector tubing to the cannula hub is necessary, resulting in less exposure of the acrylic to the atmosphere and of the connector's Luer-lock threads to the acrylic. With delivery systems, however, the tactile feedback is greatly diminished and the operator has to rely on visual cues, such as crowding of the barium particles in the cannula, to detect compromised acrylic flow. Moreover, pressure build-up in the system resulting in sudden expulsion of acrylic from the cannula tip is more likely with injection devices than 1-mL syringes. The devices are often expensive, and may only be available in a "kit," often significantly increasing the cost of a procedure.

One-cc syringes with Luer-lock hubs are inexpensive, readily available, require minimal storage space, and allow exquisite tactile feedback during injection, which improves acrylic flow control. However, their use places the operator's hands closer to the radiation field than an injection device, increasing exposure. In one prospective study, the radiation dose per minute to the operator's fingers was

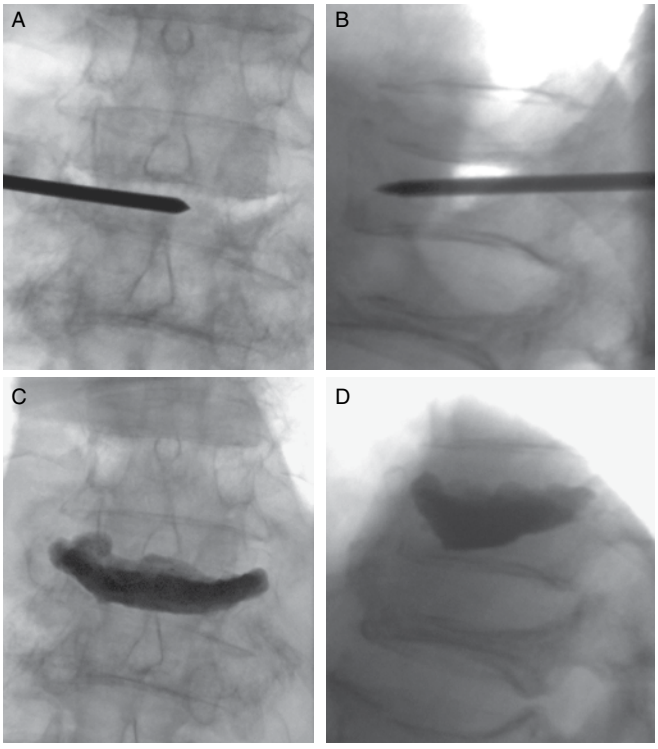

Figure 46.10 Cavity formation within a vertebral body because of osteonecrosis (Kummell's disease) is a common finding, manifesting as a lucent, crescent-shaped area within the bone (**A**,**B**). The cavity fills with PMMA easily and usually does not flow into the adjacent trabecular bone (**C**,**D**). It is possible to overpressurize the cavity during injection with resultant decompression of acrylic along the cannula track when it is removed.

significantly lower when using an injection device versus 1-cc syringes. However, because of the longer duration of the injection with the device, the total dose to the operator was the same in both cases [67]. Regardless of whether a device or syringes are used, operator dose can be minimized by placing the x-ray tube on the side of the patient opposite to the operator and by using radiation protection gloves [68].

Fluoroscopy times are increased with kyphoplasty because of the additional step of placing and inflating the balloons. A recent study [69] ascertained that without the operator wearing eye or hand protection, the total radiation dose to these areas would exceed the occupational exposure limit after 300 cases per year. Another factor that affects exposure dose is lack of experience. In one study, fellows and residents showed a higher operative time per vertebra than staff [69]. Use of intermittent fluoroscopy during K-wire and cannula placement and holding the K-wire with a clamp while positioning the cannula may decrease operator dose but is technically more challenging [70].

The injection of PMMA to the vertebral body is an embolization procedure, and all injections are visualized under fluoroscopic control. Lateral imaging is used primarily to ensure that epidural extravasation of cement does not occur; intermittent AP fluoroscopy monitors any lateral paravertebral extravasation. As the acrylic exits the cannula, it permeates the trabecular space, giving the appearance of a concentrically expanding cloud (Figures 46.9A–D). Alternatively, it may seep along intraosseous cracks, leak through endplate fractures, or fill an internal cavity (Figure 46.10). In some instances, vertebral body expansion with reduction of kyphotic and wedge angulation will occur (Figure 46.11) [71–73]. The cannula is withdrawn slightly whenever injection becomes difficult, creating a space for acrylic flow. Whether using an injection or a syringe, forward pressure is removed before repositioning the cannula to avoid sudden PMMA deposition into a new space. Typically, the injection is terminated when the acrylic reaches the posterior one-quarter of the vertebral body, to avoid embolization of the basivertebral plexus. Overzealous attempts at complete vertebral filling risks complication for little clinical gain as studies have shown that low-volume and hemivertebral fills achieve as good a clinical outcome as patients treated with bipediculate and/or high volume approaches [26,74,75].

Failure of the PMMA to egress from the cannula tip may be due to obstruction from bony trabeculae, or from a blockage within the 1-cc syringe, injector tubing, or cannula. Acrylic compaction occurs when continued injection against a relative obstruction forces the liquid monomer out of the material. The resultant powder plug will obstruct the cannula lumen, compelling its removal. Compaction is best identified by the lack of movement of PMMA into the vertebra, with crowding of the constrained opacification particles within the cannula. If repositioning of the cannula tip slightly posteriorly does not result in acrylic flow, then the syringe or delivery system is disconnected and evaluated for plug formation. If no obstruction is present, the cannula is cleared with the stylet under fluoroscopic observation, and, if successful, injection resumes.

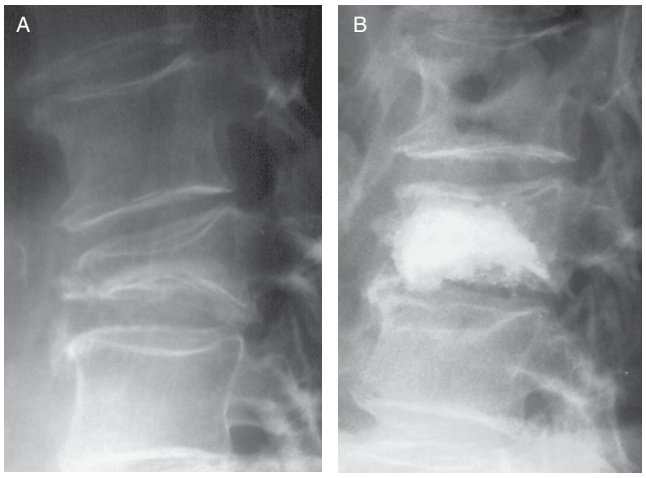

Figure 46.11 A retired physician who developed low back pain was found to have a wedge fracture of the L3 vertebra (**A**). Height restoration and kyphosis correction were noted during injection of PMMA. The patient experienced excellent pain relief. On a follow-up visit, a lateral plain film (**B**) showed good anatomical alignment, but a new superior endplate fracture of the L4 vertebra was noted.

Large amounts of PMMA within the disc space may act as a wedge causing fracture of the adjacent vertebra [76], although smaller volume leaks seem to be tolerated without complication [77]. If the acrylic preferentially flows to a vein, the needle is repositioned more posteriorly and the material is allowed to thicken. Injection is terminated if continued venous or extravertebral filling occurs.

Before its removal, the cannula is rotated 360° in order to separate any stream of acrylic that may remain attached to the material within its lumen. It is withdrawn under fluoroscopy to ensure that acrylic does not extend into or detach within the subcutaneous soft tissues where it may act as a source of pain or infection. In addition, decompression of PMMA along the track has been seen when large intraosseous cavities have been filled and are under pressure, and the cannula should be removed cautiously.

If the vertebral filling volume is deemed inadequate, then the procedure is repeated on the opposite side. Otherwise, the skin is cleaned and dressed with small adhesive bandages, and the patient is transferred to the recovery room for further observation and care.

Postprocedural Care

In the outpatient setting, most postvertebral augmentation patients are observed for 2 hours prior to discharge. Individuals remain supine for 1 hour, and are gradually allowed to sit up and/or stand over the next hour under direct nursing or physician supervision. Once recovered, ambulatory patients are discharged to the care of a responsible adult. It has been common practice to admit kyphoplasty patients for observation overnight. In many cases, the patients had been placed under general anesthesia, requiring a longer recovery period. In reality, admission was done primarily for billing purposes. However, kyphoplasty is reimbursed in the outpatient setting, and the majority of cases can be performed using conscious sedation with same-day discharge.

Patients often experience some immediate pain relief, either from the residual effects of the local anesthetic or from the procedure, or a combination of both. Focal pain at the puncture sites is common and may last up to 48 hours. Pain medication may be taken as needed; however, patients are encouraged to either discontinue their narcotics or substitute nonsteroidal anti-inflammatory agents so that efficacy can be determined. Prior to discharge, patients are evaluated for chest or back pain, new neurological dysfunction, dyspnea, or other potential complications of the procedure. Most significant complications are due to extraosseous acrylic deposition and early recognition is vital so that appropriate treatment can be instituted. Usually, neurological and pulmonary complications are apparent during the observation period and are treated emergently. For this reason, access to CT scanning and surgical back-up is an absolute requirement for any vertebral augmentation service.

Patients are instructed to remove bandages at 24 hours and to keep the skin incisions clean and dry. Follow-up either by direct contact or telephone interview is done within the following week. Patients are to notify the physician of redness or discharge at the operative site, recurrent or new back pain, chest pain or shortness of breath, or unexplained fever or neurological symptoms. Any new symptom requires clinical evaluation and, possibly, imaging. New back pain may indicate recurrent or new fracture, unrecognized facet pain, or epidural abscess. Chest pain may be the result of rib fractures or unsuspected pulmonary embolization of acrylic. All neurological symptoms require immediate CT scanning to search for misplaced PMMA, and suspected osteomyelitis or abscess is best investigated with MRI.

People who have been immobilized for a long period of time should gradually increase their activity, often under the auspices of a physical therapist. Some individuals who feel better immediately try to return to full activity, only to fracture another vertebra, a hip, or a wrist. Patients who are not receiving preventative medical therapy are referred to primary care, endocrinology, or geriatrics for further evaluation and implementation of appropriate medical treatment.

COMPLICATIONS

As the number of vertebral augmentation procedures increase worldwide, it is anticipated that the number of complications will increase, particularly as inexpert operators tackle their first cases. As with any procedure, an individual's complication rate will be highest during the learning phase, and one's confidence and competence will be enhanced by preclinical experience using models or cadavers, and by observation of a seasoned practitioner. Complications are best avoided by a thorough understanding of the factors that contribute to their occurrence. They are most commonly associated with (1) poor visualization owing to inadequate fluoroscopic equipment, poor patient cooperation ("the moving target"), or unsatisfactory acrylic opacification; (2) operator error, such as inappropriate patient selection; lack of knowledge of the radiographic spinal anatomy, particularly bony and venous; poor fluoroscopic-triangulation skills; unfamiliarity with equipment, devices, and PMMA; and poor embolization technique; (3) lack of patient monitoring; and (4) improper aseptic technique. By recognizing and avoiding these potential pitfalls, and thoroughly educating oneself before performing vertebroplasty, operators will markedly decrease their chances of causing a significant complication.

The primary cause of a symptomatic vertebroplasty complication is leakage of PMMA into adjacent structures—through fracture lines or cortical destruction (Figure 46.12), along the needle track, or into the epidural and paravertebral venous complexes [11,25,53,78,79]. Often, it is the overzealous quest for complete vertebral body filling that results in this type of complication, but "more" definitely is not "better" where vertebral augmentation is concerned.

Acrylic material located within the epidural venous plexus (Figure 46.13A) or foraminal veins (Figure 46.13B) may cause spinal cord or nerve root compression, with resultant worsening pain and/or neurological dysfunction (Figure 46.13C). Migration of small amounts of PMMA to the pulmonary vasculature via the epidural or

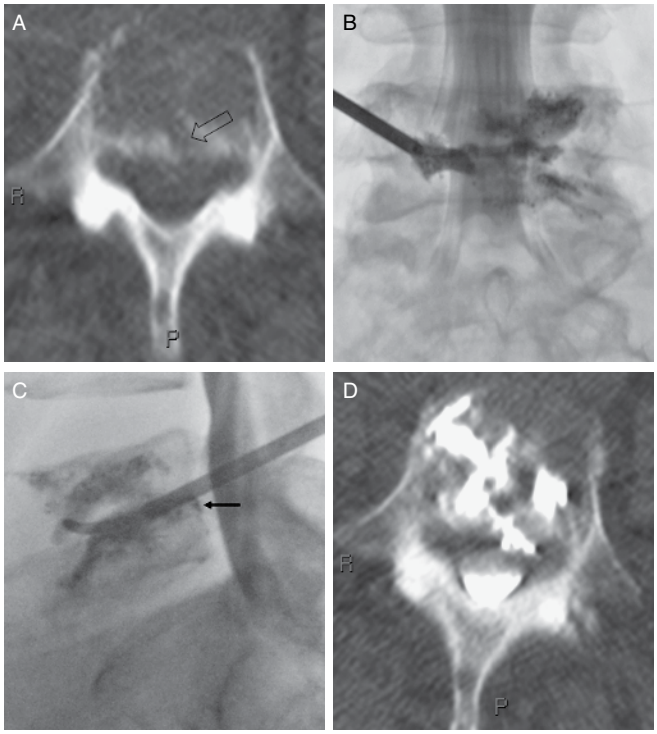

Figure 46.12 This 56-year-old male with multiple myeloma was evaluated for acute back pain, and was found to have multilevel involvement, worst at the L5 vertebra. CT (**A**) confirms the osseous destruction and better demonstrates the cortical erosion of the posterior wall (**A**, open area). Vertebroplasty was performed after instillation of contrast material within the thecal sac to detect any leaks that might compress it. AP (**B**) and lateral (**C**) views of the vertebroplasty show inhomogeneous acrylic spread throughout the vertebral body, with a small amount of material located outside of the posterior wall (**C**, arrow). CT after vertebroplasty (**D**) shows PMMA within the tumor located in the vertebral body with extension through the discontinuous posterior wall and into the canal with no thecal sac compression noted.

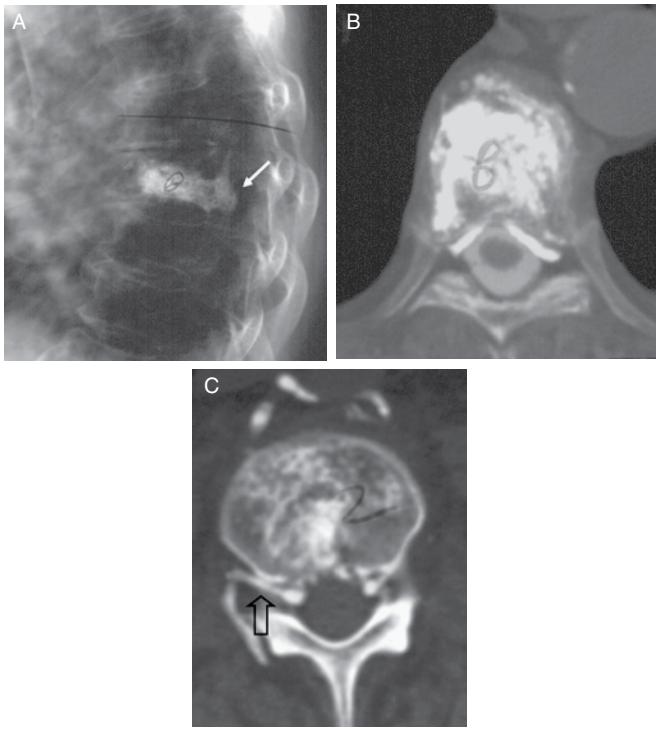

Figure 46.13 This patient had an uneventful postprocedure course following vertebroplasty of the T8 vertebral body. There was acrylic located posterior to the vertebral body on the postprocedure lateral image (**A**). A few months later, she had a cervical myelogram followed by a CT and the T8 vertebra was scanned for interest (**B**). Note the acrylic that has migrated into the epidural venous plexus and is located within the intervertebral veins. In an unrelated case, a patient who underwent L2 vertebroplasty awoke with excruciating back and leg pain due to compression of the right L2 nerve root from PMMA in the foraminal vein (**C**, open arrow). The patient required a nerve root block for pain relief.

paravertebral venous system is usually without clinical significance [80], but symptomatic pulmonary embolus has been reported [81].

Rarely, perivertebral acrylic has resulted in esophageal compression and dysphagia [82]. An increase in the adjacent fracture rate when acrylic migrates into the disc space has been described [76], most likely because of decreased cushioning ability, but this phenomenon may be volume dependent [77]. Rarely, material within the disc space may cause disc extrusion resulting in acute myelopathy following vertebroplasty.

More often than not, PMMA leakage is asymptomatic, even in malignant lesions. Cotten et al. [25] demonstrated venous and cortical acrylic leaks by CT in 29 out of 40 patients with osteolytic metastases or myeloma. Most of these leaks were asymptomatic, but two of eight foraminal leaks produced nerve root compression that required decompressive surgery. In a later series, Cotten et al. [53] reported 1 out of 258 treated patients with spinal cord compression that required surgery and 3 out of 13 patients with radicular pain that required surgical decompression. Most radiculopathies respond well to anti-inflammatory or narcotic analgesics or local anesthetic infiltration. Deramond et al. [11] noted a single transient neurologic complication in 80 patients with osteoporotic fractures. Review of all major vertebroplasty series shows that the complication rate ranges from 1% to 10%; Murphy and Deramond [78] divide it further into 1.3% for osteoporosis, 2.5% for hemangiomas, and 10% for neoplastic disease.

It has been postulated that extravertebral leakage occurs more often in vertebroplasty than kyphoplasty, because the cavity created in kyphoplasty maintains the acrylic within the space. Extraosseous PMMA is most likely to be detected on CT. In a recent retrospective review of vertebroplasty performed under CT guidance [61], 55.4% of the treated levels demonstrated extravertebral material, yet the clinical complication rate was only 2.1%. In a retrospective review of CT performed after vertebroplasty or kyphoplasty [83], extravasation was seen in

89% of vertebroplasty cases, but only 50% of kyphoplasty cases. However, PMMA was more likely to be seen in the perivertebral soft tissues in vertebroplasty cases, whereas it was more likely to be found in the perivertebral veins in kyphoplasty. Although the percentage of patients with acrylic leaks may be higher with vertebroplasty, the percentage of catastrophic complications is possibly higher with kyphoplasty [79], but there is no compelling evidence that one technique is safer than the other.

Significant complications also may occur from inappropriate trocar positioning. Improper placement of the cannula tip within or near the basivertebral plexus places the patient at risk for deposition of PMMA into the epidural venous plexus. Advancement of the trocar through the anterior vertebral body wall could damage the aorta or inferior vena cava. Use of the paravertebral approach may injure the intercostal or lumbar artery resulting in a paravertebral hematoma. Transgression of the dura may lead to a symptomatic cerebrospinal fluid leak or decompression of PMMA into the thecal sac after cannula removal (Figure 46.14). Pneumothorax is a potential complication in thoracic procedures. Fracture of the transverse process or pedicle, epidural abscess, seizure, respiratory arrest, and death have been reported in vertebral augmentation. Severely osteoporotic patients may sustain rib fractures from lying prone on the procedure table [10]. Padding the table, performing the puncture with the patient in the decubitus position, or advancing the needle through the bone with the use of a hammer may help to decrease the chance of a rib fracture.

Transient systemic hypotension during acrylic injection in vertebroplasty has been seen clinically [84], and induced in an experimental sheep model [85]. This phenomenon may be due to toxic effects of the monomer, or from the release of marrow fat into the circulation. Yet, a large retrospective study of the cardiovascular effects of PMMA in vertebroplasty found no generalized association between acrylic injection and systemic cardiovascular derangement [86].

One theoretical complication is thermal injury to adjacent neurological structures during acrylic polymerization. There have been no clinical reports of this phenomenon and its possibility appears unlikely based on in vitro tests, which showed no significant temperature rise in the spinal canal with vertebroplasty [87], and in vivo animal experiments, which showed no injury to adjacent neural, bony, and disc tissue in animal models [21,22].

Injury to the medical staff from PMMA vapor exposure is an important concern. Occupational Safety and Health Administration (OSHA) limits for personnel are set at 100 ppm per 8-hour shift. Cloft et al. [88] have shown exposure of less that 5 ppm to physicians performing vertebroplasty in a standard-ventilation angiography suite. Even though the exposure is negligible, some people may experience an idiosyncratic reaction or asthma exacerbation in response to the pungent smell of the monomer.

The issue of increased risk for fracture after vertebral augmentation is a matter of intense debate. Grados et al. [89] found a slight but statistically significant increased risk of vertebral fracture in the vicinity of a treated vertebra when compared to a vertebral fracture in the vicinity of an untreated fracture. Mudano et al. [90] compared patients treated with vertebroplasty or kyphoplasty to patients with a previous VCF, and found that treated patients had a significantly greater risk of secondary VCFs than the untreated patients for fractures within 90 days of the procedure or comparison group time point. A review of the pertinent literature in 2006 by Trout and Kallmes [91] concluded that it was difficult to make strong conclusions about a causal relationship between vertebroplasty and incident fractures. The review highlighted the necessity of detailed reporting and analysis of incident fracture risk in prospective studies, the requirement of a discussion of the potential "new fracture" risk with patients, and the call for preventative medical management of osteoporotic patients.

In summary, the small number of reported serious complications should not lull operators into a false sense of security. By recognizing and avoiding the potential pitfalls described earlier, and by thoroughly educating oneself before performing vertebral augmentation, operators will markedly decrease their chances of causing a significant complication. If significant neurologic compromise were to occur, surgical colleagues must be available for immediate consultation or intervention. Vertebral augmentation should only be performed at sites where a surgical back-up plan has been established.

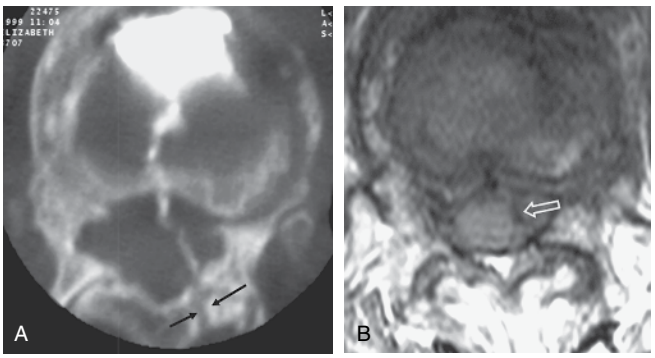

Figure 46.14 This 80-year-old female with a T11 compression fracture underwent vertebroplasty at an outside institution. Following the procedure, she complained of incontinence and leg weakness. Spiral CT with axial reconstruction shows the trocar track through the lamina (**A**, arrows) and decompression of PMMA along through the subarachnoid space. Axial MR image at the same level demonstrated application of the PMMA along the lateral aspect of the conus (**B**, open arrow).

CLINICAL OUTCOMES

The past 20 years have produced a prodigious outpouring of vertebral augmentation literature, of which over 100 studies address the clinical outcomes of patients treated with vertebroplasty (VP) or kyphoplasty (KP). Vertebroplasty has consistently shown immediate and considerable improvement in pain and patient mobility following treatment [9–12,25,26,45–51,53,54,58–60,74,75,82,89,92–96].

Unfortunately, these "clinical outcomes" papers were plagued by methodological limitations including lack of control groups, retrospective assessment of pain and functional ability, and use of either self-developed, nonvalidated outcome measurement instruments or validated instruments that were not specific to VCFs. However, there have been some prospective studies completed in the past 5 years that corroborate what practitioners have believed about the benefits of augmentation.

In a prospective investigation of VP outcomes in 167 patients with 264 compression fractures, Do et al. [97] showed statistically significant clinical benefit in pain, analgesic use, activity level, and SF-36 scales at 1 month following VP. Long-term continued benefit was shown on the SF-36 scale at 6-month to 3-year follow-up. In the largest and longest of the prospective, nonrandomized series [98], 1000 treated patients were studied and the results showed statistically significant immediate and sustained (up to 2 years) good outcomes as measured by the VAS and the Roland-Morris Disability (RMD) score, with a low complication rate composed primarily of rib fractures.

Prospective series comparing vertebroplasty to best medical therapy are small in number, but four have been completed and demonstrated the superiority of vertebroplasty over conservative care. Alvarez et al. [99] and Diamond et al. [100] enrolled patients suffering from acute vertebral fractures (1–6 weeks of pain unrelieved by oral analgesia) and offered them vertebroplasty. The treated patients were compared to a cohort that declined VP and opted for medical management. In the Alvarez study, the patients who elected for VP showed improvement in pain, function, and general health scores from their preoperative mean values ($P < 0.001$) in all postoperative periods (postprocedure, 3 months, 6 months, and 1 year). The treated group also showed statistically significant less pain than the medical cohort at 3 and 6 months, although no difference was seen at the 1-year point. Narcotic use following vertebroplasty was markedly reduced (71%–26%), with a greater reduction in the VP group than the medical group at 3 months. In the Diamond study, the VP-treated group showed improvement in pain and function and a lower rate of hospitalization at 24 hours ($P < 0.001$) when compared to the medical cohort. The pain outcome continues to surpass medical therapy at 6 weeks, although the functional outcomes as measured by the Barthel Index approached the maximal score for both groups at this time point. Three minor complications were noted, and there was no difference in the new fracture rate between the groups.

In a recent randomized, prospective trial of VP (18 patients) versus optimal pain medication (OPM; 16 patients) [101] the VERTOS study intended to follow the cohorts for a year but was altered as almost all patients on OPM crossed over to VP at 2 weeks. Analysis of the data at 2 weeks demonstrated that pain relief and improvement in mobility, function, and stature after VP was significantly better when compared with the medical cohort. Fourteen of the sixteen OPM group crossed over to VP; they experienced similar significant pain relief at 1 day and 2 weeks when compared to the short-term results of medical management.

The results of the VERTOS II trial were published in August, 2010 [3]. This study was a multicenter, randomized, controlled trial with the objective of comparing the cost-effectiveness of vertebroplasty to conservative treatment in terms of pain reduction, QOL, complications, secondary fractures, and mortality [102]. The inclusion criteria were strict—patients had to have an MRI-positive compression fracture measuring of at least 15% of the vertebral body height involving T5 or lower, a pain scale score of 5 or greater on the VAS with point tenderness over the fracture site, a bone density score of less than or equal to –1, and symptom duration of no longer than 6 weeks. Four hundred thirty-one patients were eligible for randomization; 202 patients with persistent pain were randomized with 101 patients enrolled in each group. Patients randomized to the conservative group were treated with an optimized pharmacologic regimen, consisting of analgesics ranging from acetaminophen to oral, and possibly, parenteral narcotics. Physiotherapy was also used. Both groups received osteoporosis medications. Patients were evaluated using the VAS score, QOL Questionnaire of the European Foundation for Osteoporosis (Qualeffo), EQ-5D, and the RMD questionnaire. Data was collected at 1 day before, 1 day after, 1 week, and 1-, 3-month, and 1-year intervals after VP or after the start of conservative care. The primary outcome was pain relief at 1 month and 1 year as measured on the VAS.

This landmark trial showed that patients with severe pain from a recent (less than 6 weeks) vertebral fracture were statistically more likely to experience quicker and greater pain relief with VP than a similar cohort treated with highly tailored drug regimens and physical therapy. Day-to-day, individualized drug therapy was afforded to both groups, thus eliminating variable medication regimens as a confounding factor in the outcomes. Not only did the VP group show improved pain, but they used a lower class of drugs than the control group, or no drugs at all. The increased pain relief after VP remained significant up to the 1-year endpoint, suggesting that patients in the control group may develop chronic back pain in response to nonhealing fractures. The incremental costs of vertebroplasty were primarily procedure-driven, but the difference was insubstantial at the 1-year endpoint. No serious complications occurred, and the incidence of new fracture was comparable between the two groups. Overall, the VERTOS II trial was a resounding success, and justifies the use of vertebroplasty in patients with fractures 6 weeks or less over conservative treatment.

Two recently published double-blind, placebo-controlled, randomized trials, one VP versus sham treatment [4] and the other VP versus control intervention [INVEST] [5] brought into question the efficacy of vertebroplasty. In the Buchbinder trial, VP demonstrated no benefit in pain relief over a sham procedure consisting of tapping on the pedicle with a blunt trocar. In the INVEST trial, patients responded positively to vertebroplasty, but the pain relief was no better than that afforded by instillation of bupivacaine into the pedicle. The implication of these studies is that placebo effect plays a large role in pain and functional improvement. The recently completed LABEL trial [103], compared the outcomes of patients receiving lidocaine and

bupivacaine injections for vertebral fractures to the outcomes of the blinded control intervention group in INVEST. The INVEST control patients from the lead site experienced significantly greater average pain relief at day 1 and day 3, and significantly greater improvement in functional scores, indicating that factors other than local anesthesia were responsible for their observed improvement in INVEST.

If the placebo effect is the predominant factor in good patient outcomes, then a patient population known to have a decreased expectation-related placebo response would not be expected to respond to vertebroplasty. However, in a study of patients suffering from dementia who underwent vertebroplasty for pain [104], this cohort demonstrated a high rate of pain relief and improved mobility, suggesting that the improved outcomes in vertebroplasty are indeed a true effect.

Although the results of the INVEST and Buchbinder trials are provocative, several issues including statistical underpowering, single site influence, asymmetrical crossover, inhomogeneity of the enrolled population with respect to imaging, and possible secondary gain through workman's compensation potentially affect the results of the studies. In addition, few of the patients in the INVEST trial suffered from acute fractures (less than 6 weeks old) as they were initially ineligible for enrollment because PMMA had been given Investigation Device Exemption (IDE) status. The authors of the VERTOS II trial suggest that the older age of the vertebral fractures treated in INVEST and the Buchbinder trial, and the fact that edema on MRI was not an inclusion criterion for either study, may account for the differences in outcomes between the sham trials and VERTOS II.

Unfortunately, insurance companies and Medicare carriers are already denying vertebroplasty coverage based on the INVEST and Buchbinder studies. It can be argued that these sham trials are not applicable to the real-world scenario because they only compared VP to an intervention, and each lacked a medical arm. The positive results of the VERTOS II study provide the clinician with relevant information on how to best treat patients suffering from compression fracture pain and disability.

Clinical Outcomes in Kyphoplasty

Kyphoplasty (see Chapter 47) was introduced in 2001 [13] as an alternative approach to vertebral augmentation, and rocketed to popularity primarily in the surgical community, based in large part on the positive outcomes seen with vertebroplasty. The vertebral body is accessed in a similar manner as vertebroplasty, but a balloon-tipped bone tamp is inserted through the trocar into the hemivertebra and inflated for the purpose of reducing the fracture and creating a cavity prior to acrylic injection. The procedure is quite similar to vertebroplasty, differing only in the use of the balloon tamp, and has been referred to as "balloon-assisted vertebroplasty." Kyphoplasty proponents claimed their technique's superiority over vertebroplasty rests in its ability to restore physiologic spinal column alignment in addition to providing pain relief [105].

The clinical outcomes data in kyphoplasty are not as extensive as vertebroplasty. Several published series [106–109] demonstrate reduction in pain scale scores similar to those seen with vertebroplasty, with one study showing durability of pain relief and improved mobility to 3 years [110]. But these reports are hampered by the same methodologic flaws that plague vertebroplasty studies.

The only prospective, randomized, controlled trial of kyphoplasty was reported in February, 2009. The Fracture Reduction Evaluation (FREE) trial [2] described the outcomes of a group of patients suffering from acute osteoporotic compression fractures who were randomized to either kyphoplasty or nonsurgical therapy. The primary outcome was the difference in change from baseline to 1 month in the short form (SF)-36 physical component summary (PSC) score. QOL and other safety and efficacy measures were analyzed at 1 year. Three hundred patients were enrolled, with 266 patients completing the 1-month follow-up evaluation. There was a statistically significant improvement in the SF-36 PSC score in the kyphoplasty group over the nonsurgical group, and the frequency of adverse outcomes was no different between the two groups. For most outcome measures, the differences between kyphoplasty and the nonsurgical group were not significant at 1 year, most likely because of pain reduction associated with fracture healing. This finding has also been described in prospective, nonrandomized trials of vertebroplasty [99,100], although the VERTOS II study showed continued benefit of vertebroplasty at 1 year [3].

Several recent studies have compared outcomes between vertebroplasty and kyphoplasty [111–115]. In short, the two procedures show similar outcomes with respect to pain reduction and improved mobility. Correction of kyphotic deformity and restoration of anterior vertebral body height was better with kyphoplasty in one study, but evidence that improved anatomical alignment leading to an increased clinical benefit over vertebroplasty was lacking [112]. One study described better and more durable results with kyphoplasty [113], but others showed comparable clinical outcomes between the two at any time point, indicating that vertebroplasty and kyphoplasty are complementary techniques [111,114,115].

Complications associated with kyphoplasty are similar to those seen in vertebroplasty. Six major complications in 531 patients (1.1%) treated with kyphoplasty were reported in a multicenter collection of patients, four of which were neurological complications [116]. This complication rate is similar to the 1.3% complication rate seen in vertebroplasty for osteoporotic fractures [78].

Controversy Between Vertebroplasty and Kyphoplasty

The major points of contention between kyphoplasty and vertebroplasty advocates relate to height restoration, kyphosis correction, and safety. Currently there are multiple studies that show improved height and reduced kyphosis can occur with both vertebroplasty [71–73] and kyphoplasty [13,107,117]. However, a standardized

method of measurement has not been established. Using four different methodologies commonly employed to measure vertebral height restoration, McKiernan et al. [118] demonstrated the extreme variability in the apparent magnitude of restored height that is reported, making comparisons between studies and techniques difficult. Regardless of the degree of height restoration or kyphosis correction, there is no data to support that these changes provide any additional clinical benefit to the patient.

Another bone of contention between opposing camps is the risk of acrylic leakage during treatment. Kyphoplasty advocates state that the risk of extravasation is diminished because of the creation of a cavity, thus making kyphoplasty safer than vertebroplasty. Lee et al. [83] found a higher percentage of acrylic extravasation in vertebrae treated with vertebroplasty than with kyphoplasty. However, the kyphoplasty levels were more likely to leak in to perivertebral veins, whereas the vertebroplasty levels leaked into the perivertebral soft tissue. Regardless of the detection rate of extravasation, only two cases of pulmonary acrylic embolism occurred—one with each technique. Other studies have shown that the rate of leakage is similar, and in both instances the rate of clinical relevant complication due to leakage remains small.

Because of additional equipment, anesthesia, and hospital costs, kyphoplasty can be 10 to 20 times more expensive than vertebroplasty [119]. This cost differential should diminish significantly as surgeons become more comfortable with performing kyphoplasty under conscious sedation, and the introduction of competing balloon bone tamps, as the proprietary bone tamp patent expires. It is possible that certain subgroups of patients may derive more benefit from one particular procedure [120]. Features that might affect choice of procedure include degree of compression deformity, age of the fracture, and the presence of neoplastic involvement, but the benefits of kyphoplasty relative to vertebroplasty in such subgroups currently remain totally undefined. With the considerable added financial expense of kyphoplasty, a significant clinical benefit over vertebroplasty would have to be proven to justify this cost. A convincing benefit of kyphoplasty relative to vertebroplasty has yet to be shown in the comparative, prospective, randomized studies that have been completed to date.

Sacroplasty

Sacral insufficiency fractures (SIF) are a type of stress fractures that occur as a result of normal physiological stress applied to the sacrum ala that are osteoporotic or affected by a neoplastic process. As with VCFs, most patients are postmenopausal women, but any of the conditions that result in bone loss such as corticosteroid use, renal failure, or hyperparathyroidism can result in SIF. The diagnosis at early stages can be difficult to make on plain film, but the fractures are readily seen on MR as edema in the sacral ala, on CT as fracture lines, or on bone scan as an "H"-shaped tracer uptake in the sacroiliac joints and across the sacrum ("Honda sign"). Like VCFs, SIFs are associated with a reduction in the level of self-sufficiency and an increase in mortality [121].

The treatment of SIFs is usually immobilization and analgesic administration. In 2002, Garant described the first percutaneous puncture of the sacrum with injection of acrylic material for pain relief [122]. Dubbed "sacroplasty," (see Chapter 48) this technique was an offshoot of that used for vertebroplasty with modifications of the approach to address the relative flatness of the sacral ala, and the proximity of fractures to the neural foramina. Since then, 14 other reports have been published in the English literature regarding sacroplasty for SIF [123]. These reports are limited to technical notes, case reports, and small case series, and only five have 1-year follow-up. Only one prospective observational cohort study had been published [124]. Also noted is a smattering of reports of sacroplasty used in the treatment of multiple myeloma or metastatic disease. Sacral kyphoplasty has also been described.

Two posterior techniques have been described, with cannula placement either along the short axis of the sacrum in a posterior-to-anterior direction similar to vertebroplasty (Figure 46.15), or along the long axis, parallel to the sacroiliac joint and the neural foramina, in a

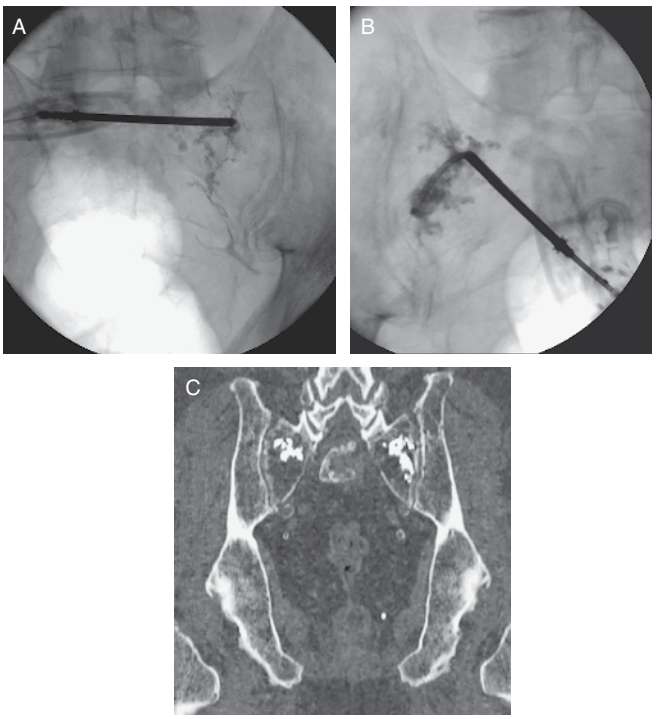

Figure 46.15 Sacroplasty was performed for pain control on this patient with widespread multiple myeloma lesions. Both sacral ala were injected with either a standard cannula (**A**) or with the use of a curved cannula (**B**). Coronal CT reconstructions following sacroplasty (**C**) shows PMMA throughout the sacral ala without compromise of the neural foramina.

caudal-to-cephalad direction [125]. The advantage of the latter approach is that acrylic can be deposited along the length of the fracture as the fracture line tends to parallel the sacroiliac joint. However, much of the sacrum can be reached from the short axis approach when a curved cannula is used (Figure 46.15B). Regardless of the approach, many operators describe a combined imaging approach, with CT for cannula placement for better fracture visualization, and live fluoroscopy for acrylic injection [123]. Injection volumes have varied from 2 to 10 cc of acrylic applied to each sacral ala.

Of the series where the VAS was utilized as a clinical outcome measurement, the average VAS improved from 8.9 pretreatment to 2.6 post-treatment [123]. Frey et al. [124] demonstrated durability of this result out to 1 year. Although the numbers are small, the clinical response to sacroplasty mimics that seen with vertebral augmentation. A prospective, randomized trial is now needed to confirm that this procedure is superior to best medical management.

SUMMARY

Vertebral augmentation procedures have evolved rapidly over the past 20 years, growing in size and scope, and fostering new developments, research, and products. Although safety has been well established in vertebroplasty and kyphoplasty, it has been only in the last 2 years that prospective, randomized, controlled trials have shown what has long been suggested—augmentation diminishes pain and improves mobility in patients refractory to best medical management. However, the results of INVEST and the Buchbinder trial raises the question of how much of this benefit can be attributed to the "placebo effect" or other unrecognized factors. Only further study will determine if vertebral augmentation will remain in the clinician's armamentarium as a treatment of VCFs, or will become another historical footnote in medicine.

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47 Percutaneous Augmentation Using Void Creation

Wade Wong, Douglas P. Beall, and Izzy Lieberman

EPIDEMIOLOGY OF VERTEBRAL COMPRESSION FRACTURES

In the United States, 700,000 vertebral compression fractures (VCFs) occur each year. This exceeds the number of hip and wrist fractures combined. Approximately 150,000 people in the United States are hospitalized because of pain in medical management associated with VCFs. This results in costs in excess of $1.6 billion annually. Osteoporosis-related disability confines the patients to more immobile days in bed than stroke, heart attack, or even breast cancer. It is estimated that the national direct expenditures for osteoporotic and associated fractures are $17 billion in 2001, and the cost is rising according to the National Osteoporosis Foundation [1].

Conventional medical therapy for osteoporosis has consisted of providing supplemental vitamin D and calcium as well as medications for maintaining bone mineral density (the antiresorptive medications, including bisphosphonates, calcitonin, hormones, and selective estrogen receptor modulators) and an anabolic medication to build bone mineral density (teriparatide). High stress physical activity such as high-impact aerobics, running, and weight lifting are also very effective at building and maintaining bone mineral density. When osteoporotic VCFs occur, conventional medical management has consisted of bed rest, bracing, and analgesics. Open surgical treatments are fraught with instrumentation failures when placed in soft bone, such that surgery is reserved primarily for cases with neurological compromise or drastic instability [1]. Percutaneous options to stabilize painful VCF have a viable place in the treatment algorithm for such fractures.

In 1984 Dr. Herve Derramond, a French Interventional Neuroradiologist, performed the first vertebroplasty on a painful C2 hemangioma. This resulted in impressive, almost immediate pain relief [2]. Since that occurrence, numerous polymethylmethacrylate injections of the spine (vertebroplasty) for osteoporotic and malignant VCFs have been performed. The success rates for vertebroplasty as determined by pain relief have ranged from 85% to 90% (see Chapter 47). The complication rate has been low, less than 2% for osteoporotic vertebral compression fractures, but as much as 5% to 10% for malignant VCFs [3,4]. A decade later, as a modification of vertebroplasty, balloon tamps are percutaneously placed into the fractured vertebrae in an attempt to better restore vertebral height and reduce kyphosis.

RATIONALE FOR REDUCING KYPHOSIS

Proponents of kyphoplasty feel that it is important to reduce the kyphosis for a number of reasons (Table).

VCFs: Consequences of uncorrected kyphosis
- Biomechanical: impaired balance, risk of subsequent fracture (see Chapter 45)
- Disability, reduced quality of life
- Reduced lung function
- Early satiety, gastric distress
- Increased mortality

From a biomechanical standpoint, a kyphotic deformity from a VCF causes the center of gravity shift further forward of the spine leading to increased stress on the paraspinous muscles. This scenario leads to early fatigue, and like the leaning tower of Pisa, older patients may be more prone to loss of balance and forward falls that could result in further injuries. Furthermore, as a result of shifting the center of gravity forward, there is increased leverage on adjacent vertebra leading to a higher likelihood of adjacent level fracture [5].

Ross found a fivefold risk of subsequent vertebral compression after the first compression fracture, increasing to a 12-fold increase after two VCFs. This further projects to a 75-fold increase after two or more VCFs in patients with low bone mass (below the 33rd percentile) [6]. Studies by Lindsay et al. reported that 20% of the patients with one or more VCFs reported an additional VCF within the ensuing year [7].

Quality of life studies have found that untreated VCFs have led to physical, functional, and psychological consequences that dramatically impair quality of life [5,8]. More specifically Silverman et al. reported a linear correlation between the number of VCFs and progressive decreases in health-related quality of life in physical function, emotional status, and overall health-related quality of life. Early satiety and abdominal discomfort from compression of the ribs on the abdominal contents is one example [9]. Schlaich et al. have reported that pulmonary

function is significantly reduced in patients with osteoporosis as opposed to those without osteoporosis because of the high prevalence of VCFs in patients with osteoporosis [10]. Leech et al. have calculated that for each thoracic VCF a 9% decrease in forced vital capacity could be expected [11].

Studies by Kado et al. of osteoporotic fractures with a cohort of 9515 women older than age 65 concluded that women with prevalent VCFs had a 23% higher mortality rate than those without VCFs. They also concluded that VCF patients are two to three times more likely to die of pulmonary causes [12]. The presence of VCFs and kyphosis is associated with a number of health-related problems including additional fracture risk, diminished quality of life, diminished pulmonary function, and shortened longevity. In that regard, the rationale to correct the kyphosis, reducing its sequelae, while ameliorating the pain by cement injection seems warranted.

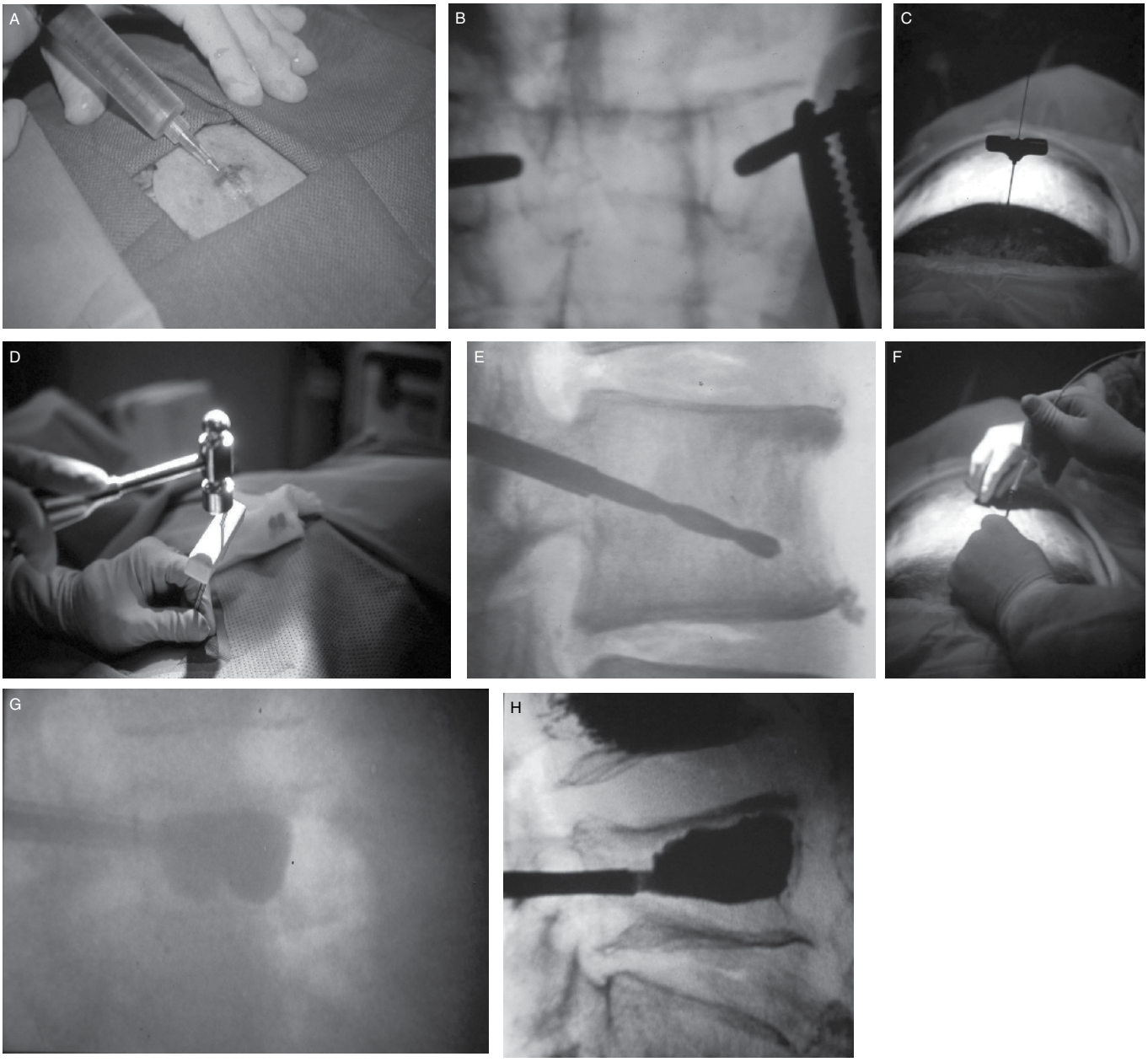

Figure 47.1 Kyphoplasty technique performed with 11-gauge needle. **(A)** Commonly performed with local anesthetic and moderate sedation; **(B)** eleven-gauge Jamshidi needles are directed under fl uoroscopic guidance; **(C,D)** exchange K wire is passed into the 11-gauge needle for its removal and exchange for an 8 gauge canula, which may be tapped into the posterior aspect of the vertebral body; **(E)** 3-mm precision drill is then passed to the anterior quart of the vertebral body; **(F)** balloon passed through the canula; **(G)** balloon is infl ated to create a void and to elevate the endplate; **(H)** bone cement is deposited into the void created by the balloon by way of bone cement filler devices.

KYPHOPLASTY

Technique

Kyphoplasty is commonly performed with local anesthetic and moderate sedation (Figure 47.1A). Eleven-gauge Jamshidi needles are directed under fluoroscopic guidance through the upper outer aspects of the pedicles of lumbar vertebra in this example L3, passing from posterior to anterior with a lateral to medial angulation (Figure 47.1B). An exchange K wire is passed into the 11-gauge needle for its removal (Figure 47.1C) and exchange for an 8 gauge canula, which may be tapped into the posterior aspect of the vertebral body (Figure 47.1D). A 3-mm precision drill is then passed to the anterior quart of the vertebral body (Figure 47.1E) so that a balloon can be passed through the canula (Figure 47.1F). The balloon is inflated to create a void and to elevate the endplate (Figure 47.1G). The endpoints of inflation are when the vertebral body kyphosis is elevated to its normal height based on adjacent normal vertebra, when balloon contact with any cortical surface, or when no further expansion of the balloon as denoted visually or by no further drop in pressure. Bone cement is deposited into the void created by the balloon by way of bone cement filler devices (Figure 47.1H).

Another variation of the kyphoplasty technique can be performed with the smaller 10 gauge express system. In this example, a VCF T8 is treated (Figure 47.2A). Ten-gauge needle trocars are passed through the pedicles of T8 with a posterior to anterior, lateral to medial angulation Figures 47.2B and 47.2E). They are passed to the anterior quarter of the vertebra (Figures 47.2C and 47.2F) and then pulled back to the post quarter of the vertebral body (Figures 47.2D and 47.2G). Balloons are then inserted and inflated to create voids and make an attempt to reduce the kyphosis (Figure 47.2H). Bone cement is then deposited into the created voids (Figures 47.2I and 47.2J).

Efficacy

Kyphoplasty has been found to be highly effective in ameliorating the pain of VCFs. A study of VCFs (n = 143) treated by kyphoplasty by Wong et al. found a 94% significant reduction in pain [13]. A retrospective analysis by Garfin et al. of 603 patients found a significant reduction in pain that correlated with a discontinued or reduced use of narcotics for fracture-related pain postoperatively [14]. Coumans et al. prospectively followed 78 consecutive patients for 12 to 18 months and reported substantial improvement in bodily pain as measured by SF 36 [15].

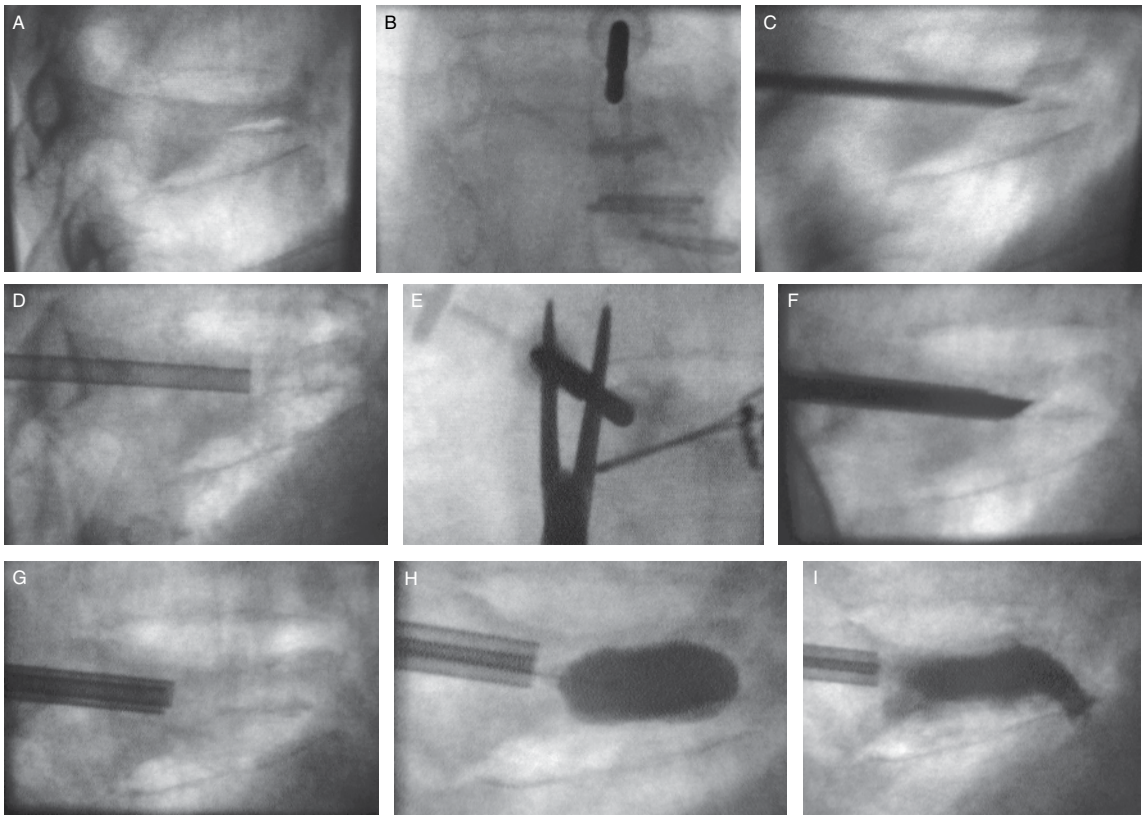

Figure 47.2 Kyphoplasty technique performed with the smaller 10-gauge express system. **(A)** In this example, a VCF T8 is treated; **(B and E)** ten-gauge needle trocars are passed through the pedicles of T8 with a posterior to anterior, lateral to medial angulation; **(C and F)** they are passed to the anterior quarter of the vertebra; **(D and G)** then pulled back to the post quarter of the vertebral body; **(H)** balloons are then inserted and inflated to create voids and make an attempt to reduce the kyphosis; **(I)** bone cement is then deposited into the created voids.

In a prospective multicenter US study by Kyphon, a 60% reduction in the level of pain was found (average preoperative Visual Analogue Scale (VAS) scores 7.5 vs average postoperatively VAS of three following kyphoplasty). The follow up in this study was continued out to 2 years [16]. Leadlie et al. reported similar long-term results with improvement continuing up to 1 year [17]. Lieberman et al. found an average decrease in pain as measured by the VAS to be from 6.18 to 2.84 in patients with multiple myeloma and fractures treated by kyphoplasty [18].

In regard to improvement in function, Leadlie et al. found 80% of 79 patients treated by kyphoplasty to be fully ambulatory at 1 week. They also followed 27 of those patients out to 1 year and found all to be maintaining full ambulatory status. In addition, 90% of the patients who had been wheelchair bound preoperatively were fully ambulatory at 1 week after kyphoplasty [17]. Coumans found a 15% improvement in Oswestry disability index at early follow up, which persisted at 12 and 18 months [15].

Kyphoplasty has been shown to improve quality of life. Coumans et al. reported that in 78 patients with 188 fractures, patient quality of life was significantly improved following balloon kyphoplasty according to SF 36 measurement in seven domains, including physical functioning, role functioning, bodily pain, social functioning, role-emotional, vitality, and mental health. Only general health remained unchanged [15]. In a Kyphon multicenter prospective US study, 100 patients post kyphoplasty were asked how many days they had spent in bed, in the last month, due to back pain, as well as how many days their activity had been limited due to back pain. Patients reported 100% reduction in median number of days spent in bed at the first time point (1 month), and this was maintained when followed out to 2 years [16].

When comparing the risk of subsequent fractures, Komp et al. compared a controlled prospective study between 21 patients who underwent balloon kyphoplasty and 19 patients who underwent conservative treatment. Of conservatively treated patients, 67% were found to have developed new fractures, whereas 37% of patients treated by kyphoplasty had developed new fractures [19].

In regard to fracture reduction and height restoration by kyphoplasty, Wong et al. found height change from 79% of the normal expected height (based on next adjacent normal vertebra) preoperatively to 99% postoperatively in fractures less than 6 months [13]. Garfin et al. found height restoration as measured anteriorly from 83% of the normal preoperatively to 99% postoperatively. At the midportion of the vertebral body, the fracture was reduced from 76% of the normal vertebral body height preoperatively to 92% postoperatively. They also concluded kyphoplasty to be most effective on VCFs that were less than 3 months old [14] (Figure 47.3). When a percent of the predicted anterior body height is calculated, Leadlie et al. found that patients treated by kyphoplasty improved from average preoperative height of 66% of the predicted normal height (based on adjacent normal vertebra) to 89% postoperatively [17]. Theodorou et al. reported an average change of 79% preoperatively to 92% postoperatively [20]. Therefore, a reasonable expectation of height restoration following kyphoplasty should be between approximately 70% preoperatively to 90% postoperatively of normal vertebral body height. Theodorou et al described change in endplate angle from 26° preoperatively to 16° postoperatively after kyphoplasty [20]. Philips et al. found comparable reductions from 17.5° preoperatively to 8.8° postoperatively [21]. Crandall et al. found acute VCFs treated by kyphoplasty improved from 15° preoperatively to 8° postoperatively, while chronic

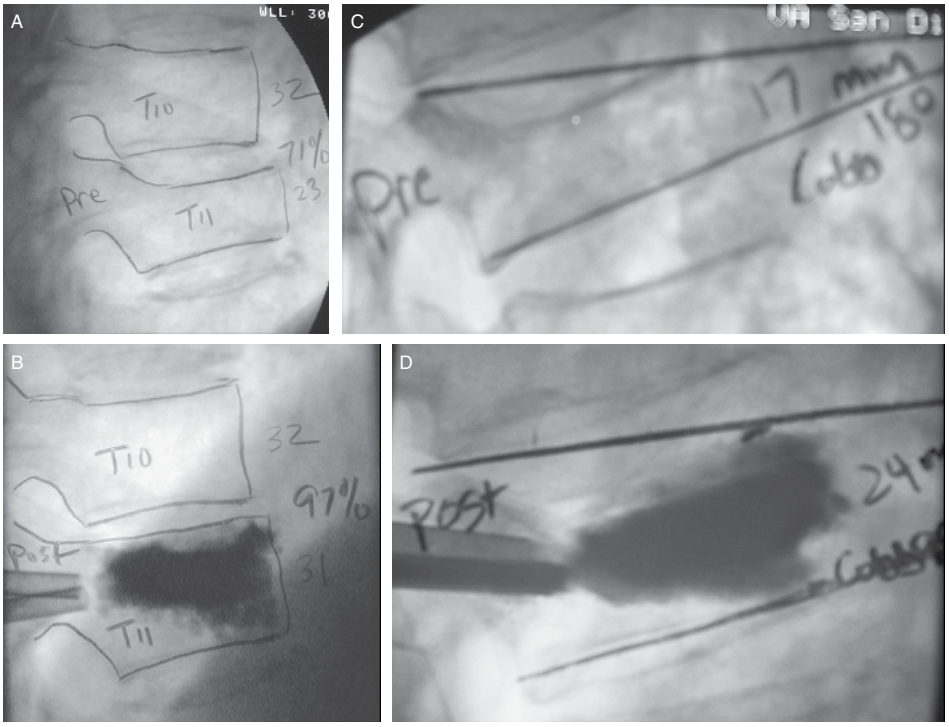

Figure 47.3 (A) An 87-year-old male complains of acute back pain for t2 months. Examination and plain film radiograph correlate with acute compression fracture at T11, which is 71% of normal expected height (based on adjacent normal T10). **(B)** Same patient plain film radiograph post kyphoplasty of T11 resulting in excellent fracture reduction with nearly return to normal height (97%). **(C)** Same patient plain film radiograph who also had painful 13-month-old L3 vertebral compression fracture that was preoperatively 58% of normal height. **(D)** Following kyphoplasty plain film radiograph of the older L3 fracture height restoration resulted in fracture reduction to 74% of normal height.

VCFs underwent somewhat less reduction from 15° preoperatively to 10° postoperatively [22]. Based on these findings, one might expect nearly a 10° change in endplate angle following kyphoplasty for each acute or subacute fracture treated and slightly less change in chronic fractures.

Vertebroplasty is not designed to provide fracture reduction and Belkoff et al. found no kyphosis reduction following vertebroplasty [23]. The lack of reduction with vertebroplasty is not always the case. For example, Hitawashi et al. reported an average increase in vertebral body height following vertebroplasty of 2.5 mm anteriorly, 2.7 mm centrally, and 1.4 mm posteriorly [24]. Teng et al. reported a mean reduction of the kyphosis angle of 4.3° with vertebroplasty and a wedge reduction angle reduction of 7.4°. For VCFs containing gas (for which the gas or internal cleft may indicate instability or maleability), the average wedge reduction angle was as much as 10.2° [25]. Hitawashi et al. more recently found no significant difference in vertebral body height restoration between kyphoplasty and vertebroplasty as their study reported similar degrees of height restoration [26]. The fractures that may be reduced vary from the fractures that have limited reduction. To maximize fracture reduction, fracture age selection and degree of bone softness should be taken into consideration.

It may or may not be possible to consistently expect significant height restoration by vertebroplasty. The task of attaining height restoration by vertebroplasty by forcefully injecting cement, may lead to increased risk of complications from cement leakage [27,28]. It is interesting that Hitawashi et al. [26] noted a much higher cement leakage rate with vertebroplasty (49% into paravertebral soft tissues and 25% into disc) as opposed to kyphoplasty (18% and 12%, respectively).

Safety

In a review article by Taylor et al., pulmonary embolism was reported to be 0.3% for balloon kyphoplasty compared with 1.8% for vertebroplasty. In the same manuscript, spinal cord compression was 0% for kyphoplasty as compared to 0.5% for vertebroplasty and nerve pain (including radiculopathy) was 0.3% for kyphoplasty, versus 2.5% for vertebroplasty [29]. A review article by Hulme et al. found clinical complications associated with kyphoplasty to be present 2.2% of the time (1288 patients), while vertebroplasty was associated within a 3.9% complication rate per patient (2958 patients) [30]. In regard to bone cement-related complications, a Kyphon examination of the literature found kyphoplasty to be associated with a cement-related complication rate of 0.22% (897 patients) and non-bone cement-related rate of 0.68%, while vertebroplasty was associated with a cement-related complication rate of 3.07% (2408 patients) and non-bone cement-related complication rate of 2.55% [16].

Eck et al. performed a meta-analysis to assess pain relief and risk of complications associated with vertebroplasty versus kyphoplasty. The authors identified a total of 1036 abstracts. Of these, 168 studies met the inclusion criteria. Mean pre- and postoperative VAS scores for vertebroplasty were 8.36 and 2.68, respectively, with a mean change of 5.68 ($P \leq 0.001$). The mean pre- and postoperative VAS scores for kyphoplasty were 8.06 and 3.46, respectively, with a mean change of 4.60 ($P \leq 0.001$). There was statistically greater improvement found with vertebroplasty versus kyphoplasty ($P \leq 0.001$). The risk of new fracture was 17.9% with vertebroplasty versus 14.1% with kyphoplasty ($P \leq 0.01$). The risk of cement leak was 19.7% with vertebroplasty versus 7.0% with kyphoplasty ($P \leq 0.001$). The authors concluded that vertebroplasty not only had a significantly greater improvement in pain scores but also had a statistically greater risk of cement leakage and new fracture [31].

Regarding cement extravasation, Hadjipavlou et al. performed a systemic review of studies published between 1983 and 2004. Overall 1279 vertebral bodies were treated by kyphoplasty and 2729 vertebral bodies were treated by vertebroplasty. The reviewers found that there was a difference in cement leakage: 8.4% for kyphoplasty as compared with 29% for vertebroplasty. They further identified the location of the leaks:

Epidural: kyphoplasty 1.2% vs vertebroplasty 10.7%

Neuroforaminal: kyphoplasty 0% vs vertebroplasty 0.6%

Intradiscal: 4% for kyphoplasty vs 8.4% for vertebroplasty

Paraspinal: 4.6% for kyphoplasty vs 6% for vertebroplasty

Intravenous: 0% for kyphoplasty vs 5% for vertebroplasty [32]

Even though cement leakage does not necessarily mean that there is a complication, it would, however, seem plausible that if an operator wanted to avoid cement leakage, kyphoplasty would lend itself more toward that goal than vertebroplasty. The use of a technique that limits the risk of leakage may be especially important in high-risk situations such as fractures of the posterior wall, malignancy, and complex fractures along multiple cortical margins. In such cases, kyphoplasty might be preferred over vertebroplasty.

However, some operators feel that there may not be a practical difference in safety between kyphoplasty and vertebroplasty. Mathis et al. [33] point out: "... both procedures relieve pain and can be performed with acceptable complication rates by prudent, well-trained physicians."

Indeed both vertebroplasty and kyphoplasty can be performed very safely as long as basic guidelines are followed. These guidelines include:

1. High-quality fluoroscopy and a sound understanding of radiographic anatomy
2. Adequate opacity of the polymethylmethacrylate (i.e., 30% barium by weight)
3. Adequate viscosity of polymethylmethacrylate (cement should be puttylike and nondripping)
4. Avoid overfilling of the vertebral body (fill anterior 2/3 to 3/4 of the vertebral body) [26,27]

Mathis et al. further point out their opinions of the value between the two procedures, "We do note the large cost differential between the two procedures. If KP [Kyphoplasty] is going to be worthwhile, it should reliably produce significantly more height restoration than does PV [Vertebroplasty]. In our practices, we believe we employ

KP differently but agree to its use when height restoration (beyond that usually achieved by PV) is feasible and would be beneficial. Our implementation of KP is driven by the 'time since fracture' and is markedly different within our own ranks. One extreme requires fractures of 3 weeks or less (J.M.M.), while another tack includes fractures of less than 3 months (O.O.)" [33].

Limitations to Use of the Balloon

Contraindications to vertebroplasty also apply to kyphoplasty. These include symptomatic VCF, unfractured vertebra, prophylactic treatment, osteomyelitis of the target vertebra, myelopathy, uncorrected coagulopathy, allergy to the bone cement, or opacifying agent. Relative contraindications include significant retropulsion of the fracture fragment or tumor extension, radiculopathy in excess of vertebral pain, and ongoing systemic infection [34].

Unstable fractures, many of which are posttraumatic and involve fractures of the posterior elements, and severe ligamentous injury are high-risk situations for neurological compromise and should therefore be evaluated for surgical reduction and stabilization rather than being considered for kyphoplasty or vertebroplasty.

A significant problem to effectively and consistently gain height restoration by kyphoplasty is encountered with hard bone situations (i.e., older fractures perhaps greater than 6 months of age, young patients, and blastic metastatic bone disease), which may be unyielding to balloon expansion. Nevertheless, advanced techniques such as the adjunctive use of high-pressure balloons or the curette as described in emerging technologies section may indeed help to affect a degree of fracture reduction and height restoration when the balloon alone is not enough.

EMERGING CONCEPTS IN VOID CREATION

Unique Applications of the Balloon and Adjunctive Void Creating Tools

In situations with hard bone, a balloon may fail to expand effectively and possibly not at all. In these situations, a technique of leaving a balloon at maximum pressure (400 psi) for an extended length of time (perhaps 8–10 minutes) may enhance expansion as the continued pressure of the balloon finally weakens the adjacent bone (Figure 47.4).

The use of the curette to score and break up sections of hard bone may provide a softer platform for the balloon to initiate expansion in situations where the balloon is unable to expand (Figure 47.5).

In situations where a vertical fracture line is encountered, a balloon may expand along the path of least resistance (i.e., the fracture line) leading to ineffective and asymmetric expansion. The use of the curette may help

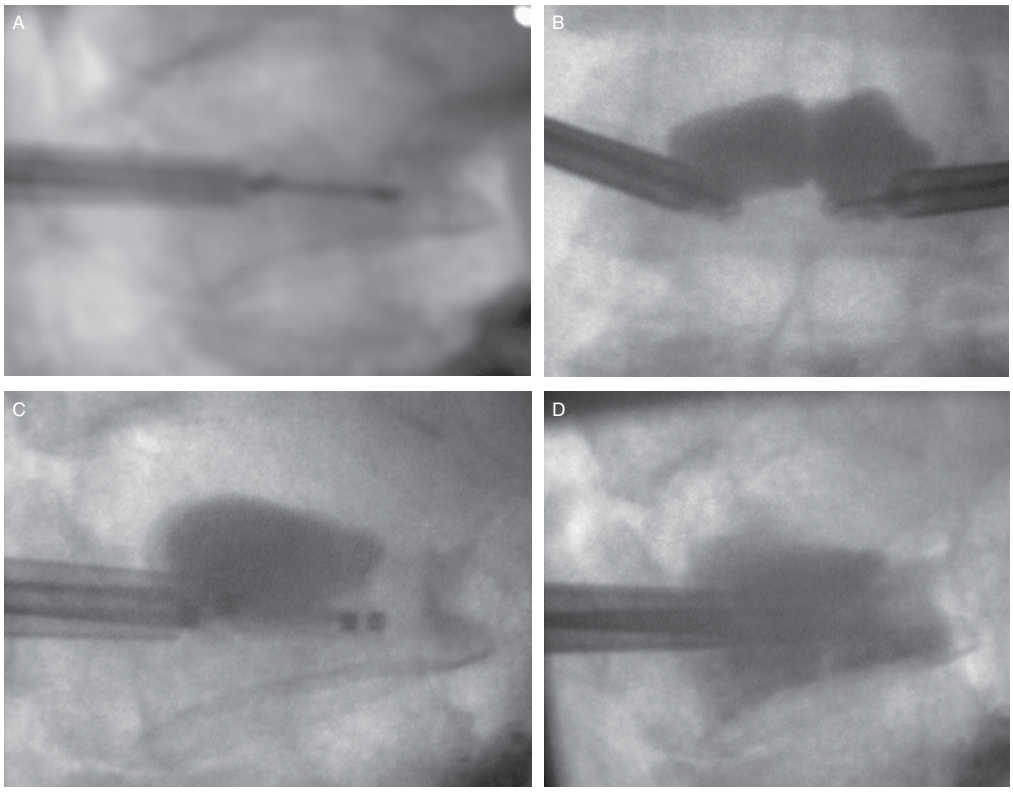

Figure 47.4 **(A)** A 6-month-old fracture is reluctant to respond to reduction by kyphoplasty balloons set at maximum pressure (400 psi). **(B–D)** lateral fluoroscopic image: AP, lateral postreduction and lateral postcementation views. The balloons were left in place instead of aborting the reduction attempt. Finally after 7 minutes, the bone yielded and partial reduction occurred.

to provide a different plane for more symmetric balloon expansion (Figure 47.6).

In cases of severe vertebra plana (especially in the thoracic region) where there may be a steep cranial to caudal angle where the pedicles meet the residual vertebral body, vertebroplasty needle entry may not be feasible to gain access to the anterior one fourth of the vertebral body. In such cases, the use of the kyphoplasty balloon with a curved stylet and deliberate progressive expansion may allow the balloon to produce a cavity out to the anterior portion of the vertebral body for effective cementation (Figure 47.7).

Retropulsion is commonly considered a relative contraindication to both vertebroplasty and kyphoplasty.

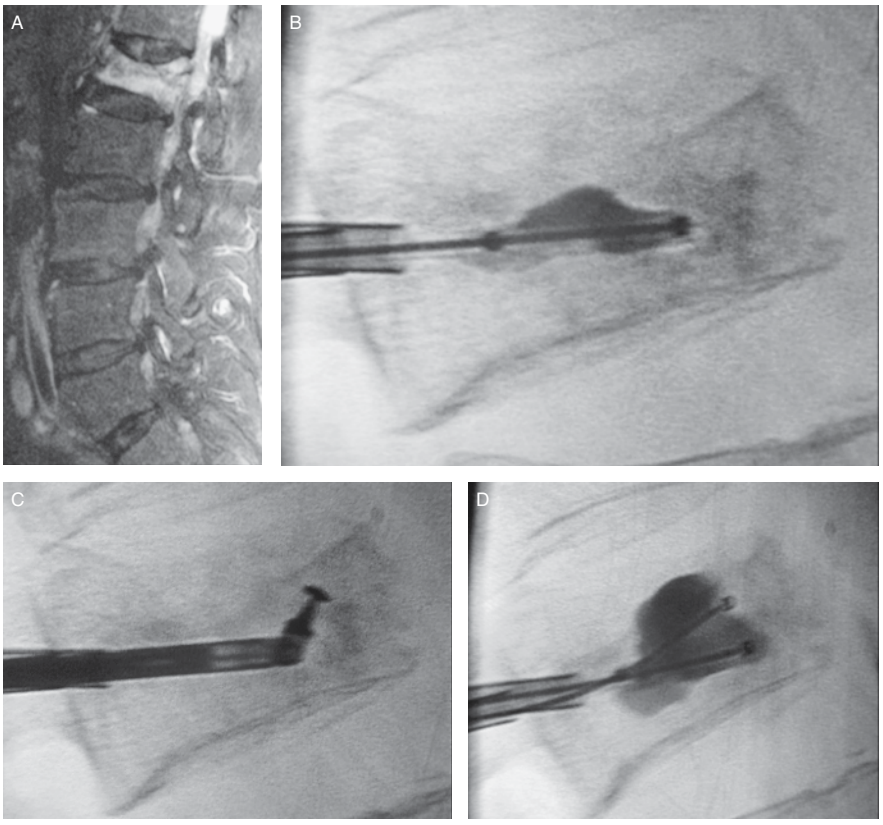

Figure 47.5 **(A)** T2-weighted sagittal MRI of a 7-week-old vertebral compression fracture L1 with considerable edema in a 57-year-old male who fell from a scaffold. **(B)** Lateral fluoroscopic image: Balloons set at 400 psi make progress in reducing the fracture after 9 minutes. **(C)** Lateral fluoroscopic image: The curette is deployed and this helps to enable the balloons to reduce the fracture. **(D)** Lateral fluoroscopic image: Following cavity creation by the curette, the balloons are now able to reduce the fracture.

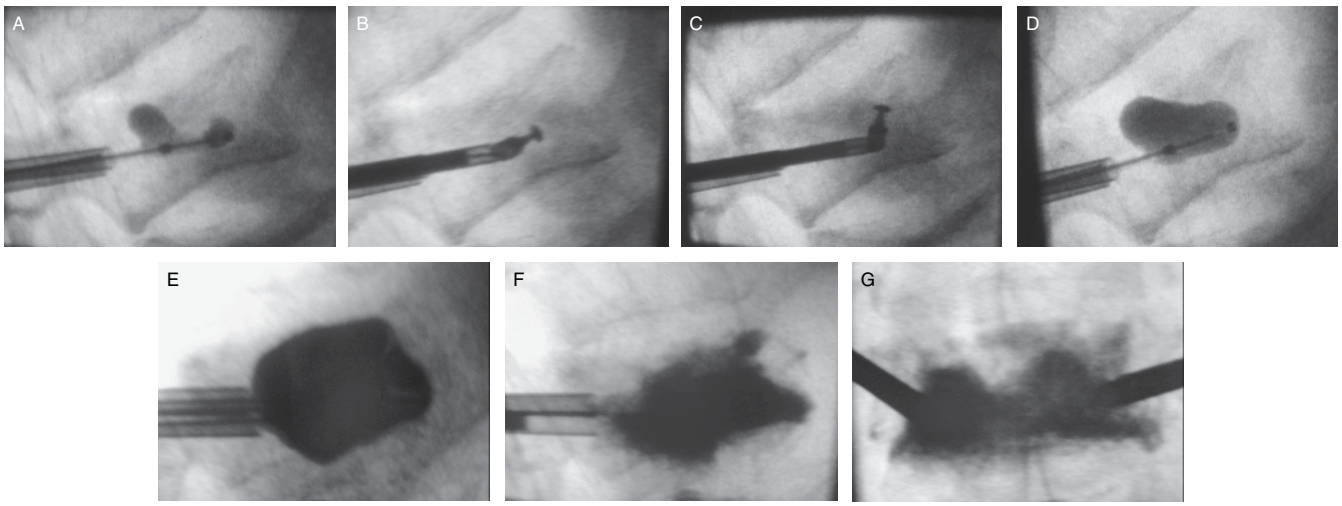

Figure 47.6 **(A)** The balloon finds a path of least resistance along a vertical fracture line. **(B,C)** The curette is deployed to create a forward cavity to allow the balloon to disperse its lifting force to the superior fracture margin. **(D,E)** Demonstrate resulting balloon expansion and lifting of the anterior aspect of the superior fracture margin. **(F,G)** Demonstrate the cementation of the reduced vertebral compression fracture.

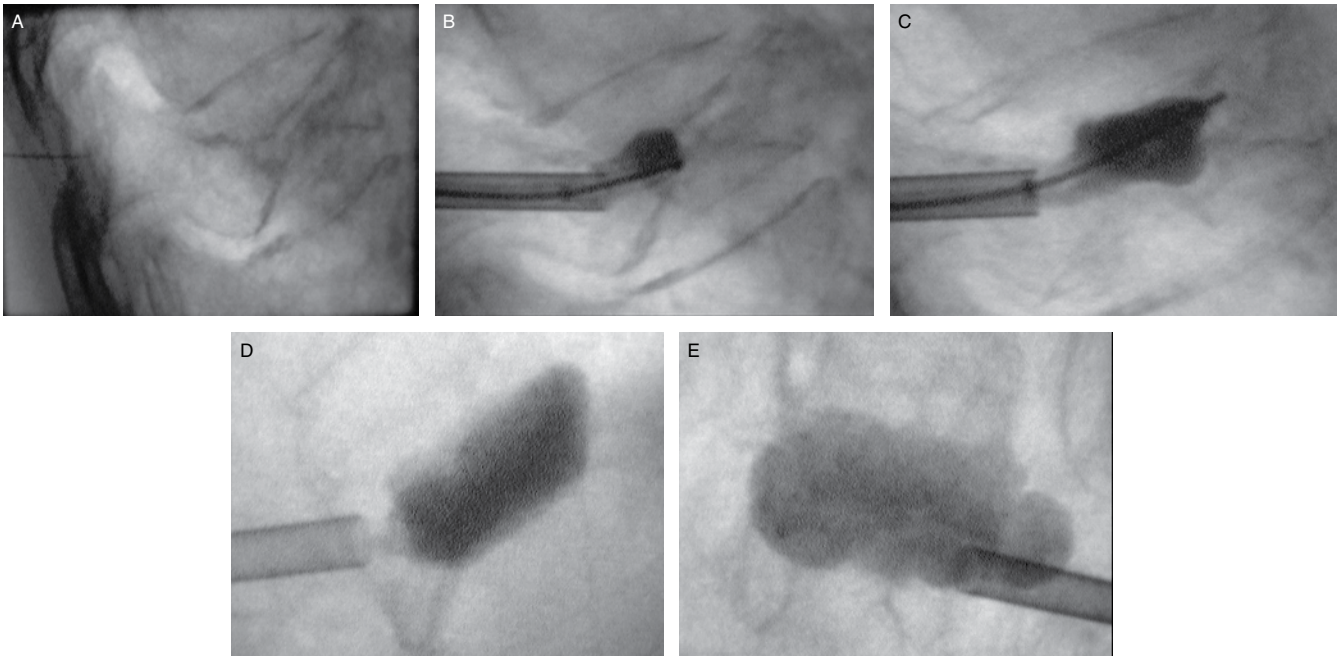

Figure 47.7 **(A)** Painful T4 severe vertebra plana. **(B,C)** Single costovertebral approach avoiding the neuroforamen. The stylet of the balloon is curved. The balloon is used as a blunt dissector to pass to the midline anteriorly. Balloon inflation proceeds until the internal cavity reaches lateral cortical margins. **(D,E)** Final cementation filling the anterior three fourth with excellent side to side filling. The patient experienced dramatic pain relief within an hour after the procedure.

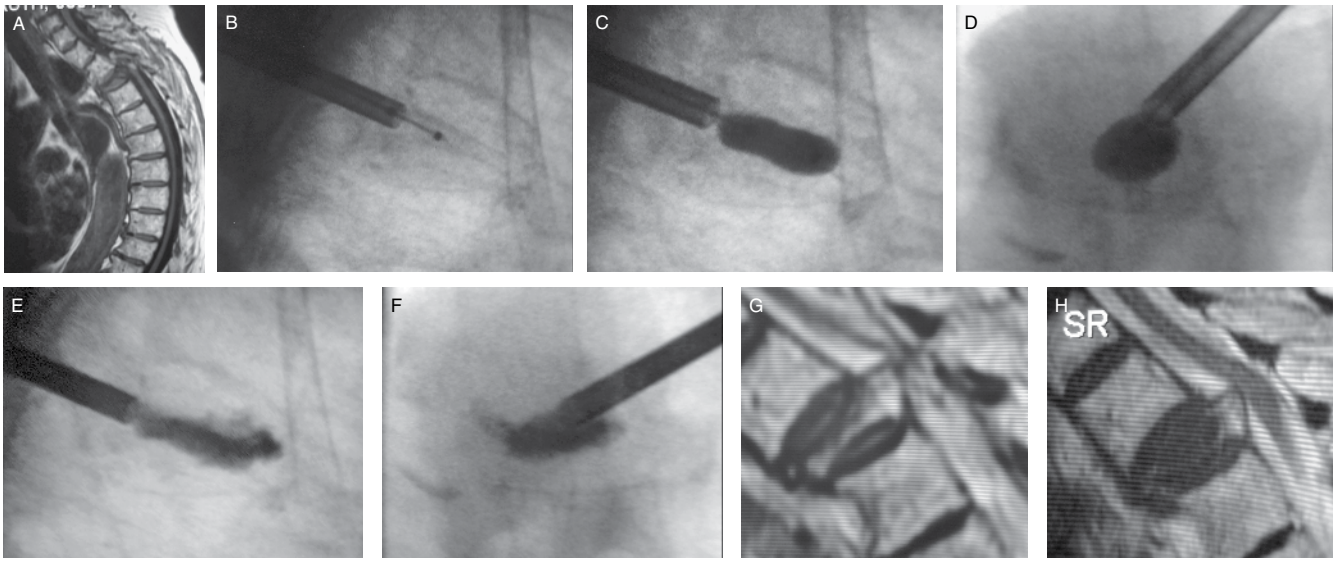

Figure 47.8 **(A)** Sagittal T1-weighted MRI demonstrates vertebra plana deformity of T3 with retropulsion and resulting stenosis in a poor surgical candidate. ((G) is the corresponding T2-weighted MRI of the same vertebra preoperatively.) **(B,C)** (lateral fluoroscopic views), and **(D)** (AP fluoroscopic views): demonstrate progressive balloon expansion. **(E)** (lateral fluoroscopic view) and **(F)** (AP fluroscopic view): demonstrated optimal cement distribution. **(G,H)** (T2-weighted sagittal MRI): (G) is prekyphoplasty that demonstrates severe central canal stenosis by a posteriorly displaced fracture fragment while(G) is postkyphoplasty, which demonstrates reduction not only in the extent of compressive deformity of the vertebral body, but even more important reduction in the degree of central canal stenosis from severe to moderate. It is theorized that by using the balloon to elevate the fracture anteriorly the posteriorly directed force on the posterior fracture fragment is reduced thereby allowing it to be redirected forward by the elastic posterior longitudinal ligament. This further demonstrates another advantage of void creation by the use of the balloon.

However, initial studies by Wong et al. have demonstrated that VCFs with retropulsed fragments in nine neurologically intact patients were treated by kyphoplasty safely. Because it was hypothesized that retropulsed fragments occur because of severe anterior compression causing a fragment of bone to be driven posteriorly, elevation (reduction) of the anterior compression deformity may permit posterior fragments to be moved forward by reduction and subsequent traction placed on the posterior longitudinal ligament. All nine patients had severely compressed vertebral bodies with retropulsed fragments leading to severe central canal stenosis. After kyphoplasty, the extent of stenosis was reduced from severe to moderate central canal stenosis. All patients experienced significant pain relief and there were no complications [35] (Figure 47.8).

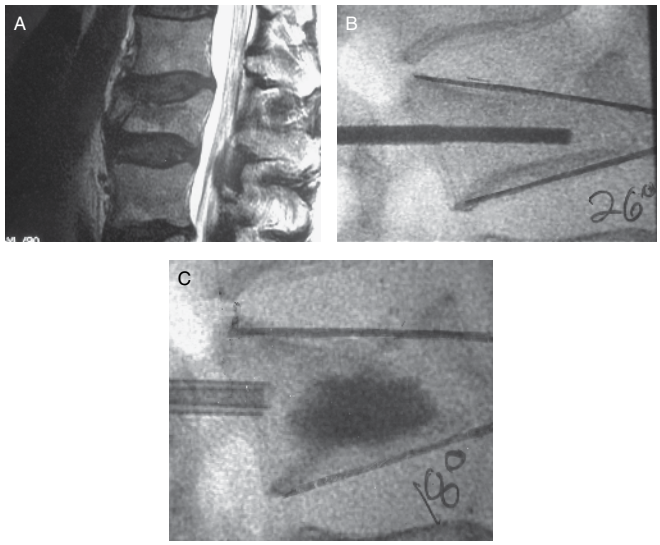

Figure 47.9 **(A)** T2-weighted sagittal MRI of L1 posttraumatic vertebral compression fracture with the anterior quarter being sheared off. Cementing this without a void may result in profuse leakage. **(B)** Lateral fluoroscopic view pre void creation by kyphoplasty. **(C)** Lateral fluoroscopic view post void creation by balloon and cementation demonstrating resulting excellent containment of cement without leakage and moderate kyphosis reduction. Patient experience significant pain relief post procedure.

When the likelihood of cement leakage is high, a limited leakage technique can be performed by first creating a cavity in bone and then back filling that cavity with extremely thick viscous cement (putty consistency) thereby permitting extremely precise control on cement deposition. This can be beneficial in situations such as complex fractures extending to the posterior cortex of the vertebral body and in cases of malignancy in which the posterior cortex is destroyed (Figure 47.9).

Alternative Methods to Cavity Creation

It has been shown earlier that the use of the balloon to create a void combined with highly viscous, puttylike cement consistency, can be used to reduce the incidence of cement leakage. Recently techniques other than the balloon are also being utilized to create a void with the idea that void creation can enhance control of cementation and limit unwanted leakage. An example of mechanical void creation is the use of the curette, which can be deployed and rotated in a circular fashion to create a void within the bone (Figure 47.10). Cardinal's AVAflex nitinol needle and Athrocare's nitinol curette can be applied similarly (Figure 47.11). Spine

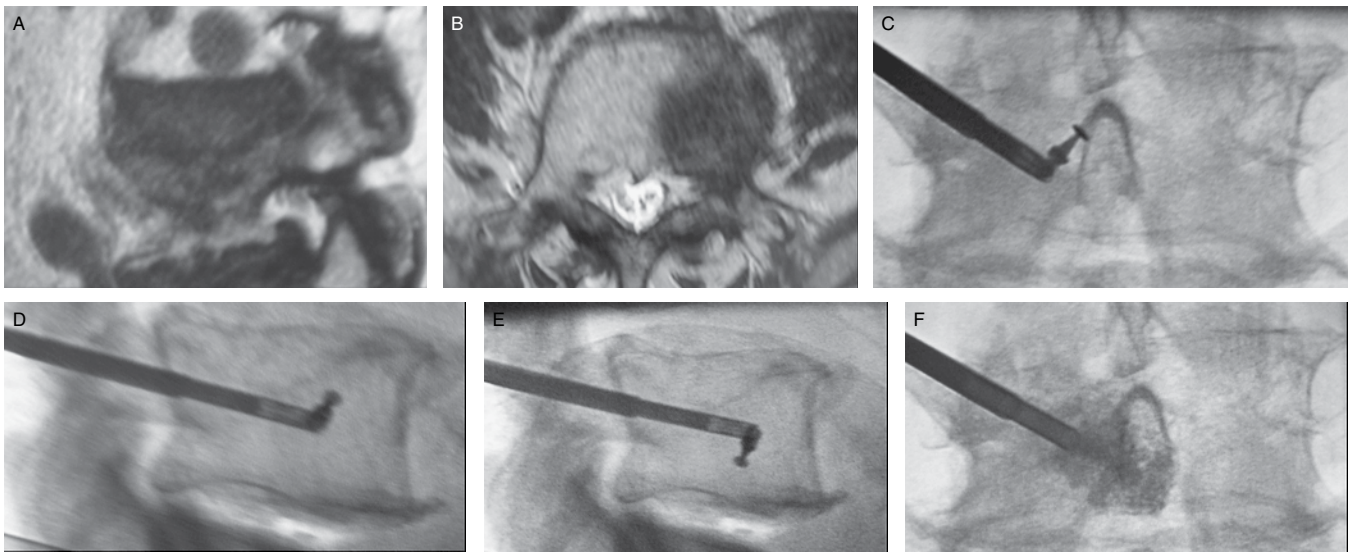

Figure 47.10 **(A,B)** Sagittal T2-weighted and an axial T2-weighted MRIs of 69-year-old male with prostate cancer resulting in painful L5 left lateral vertebral body metastasis with ipsilateral fracturing. The bone is very brittle and hard making needle entry and controlled cementation challenging. **(C–E)** Demonstrate the use of the curette to create a void in the area of blastic metastasis by rotating the curette circumferentially. **(F)** Demonstrates resulting controlled cementation in the area of blastic metastasis. The patient experienced a significant pain relief (8/10 down to 3/10) following the procedure.

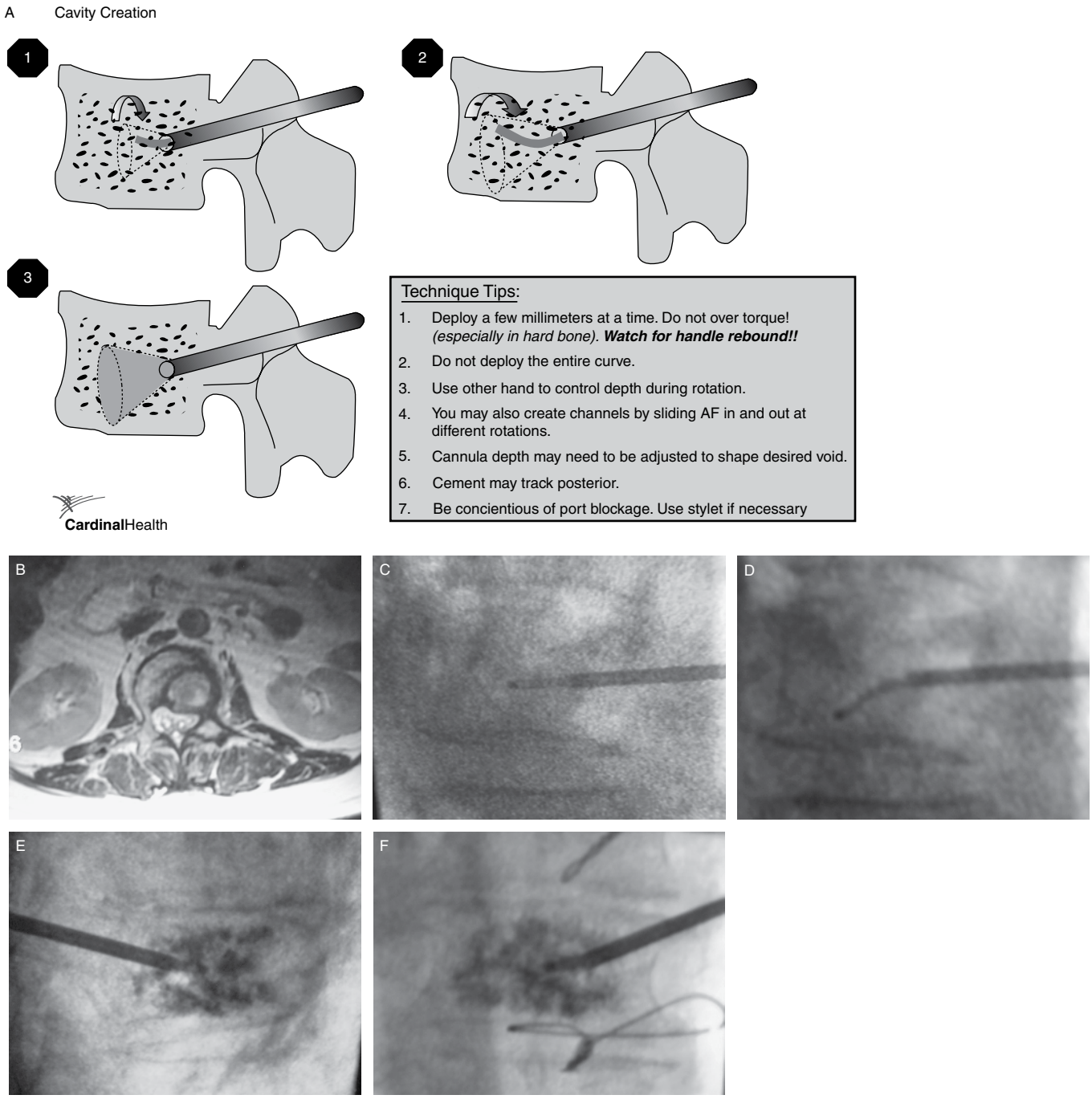

Figure 47.11 **(A)** Diagram for the technique of using of the AVAflex needle for void creation by first pushing the needle inward in various directions and then rotating to create a void. **(B)** T2-weighted MRI axial image: Case example of a painful Colon metastasis to bone with loss of the posterior cortex. **(C,D)** (lateral fluoroscopic images): The AVAflex needle is pushed inward in different directions several times before being rotated up and down to create a cavity. **(E,F)** (lateral and AP fluoroscopic images): Contained controlled cementation results in no adverse leakage and excellent pain relief. From Cardinal Health.

Wave has introduced its Staxx system of polyetheretherketone wafers to gain height and promote void creation (Figure 47.12). Spineology has introduced a mesh bag containing bone graft material in an attempt to augment and gain height restoration. At this time any significant results of these technologies are yet to be entered into the literature.

CONCLUSIONS

The benefit of void creation prior to cementation has been shown to be associated with a decreased leakage rate of cement and a slightly lower complication rate than vertebroplasty such that void creation prior to cementation may be particularly desirable in high-risk situations. It is also

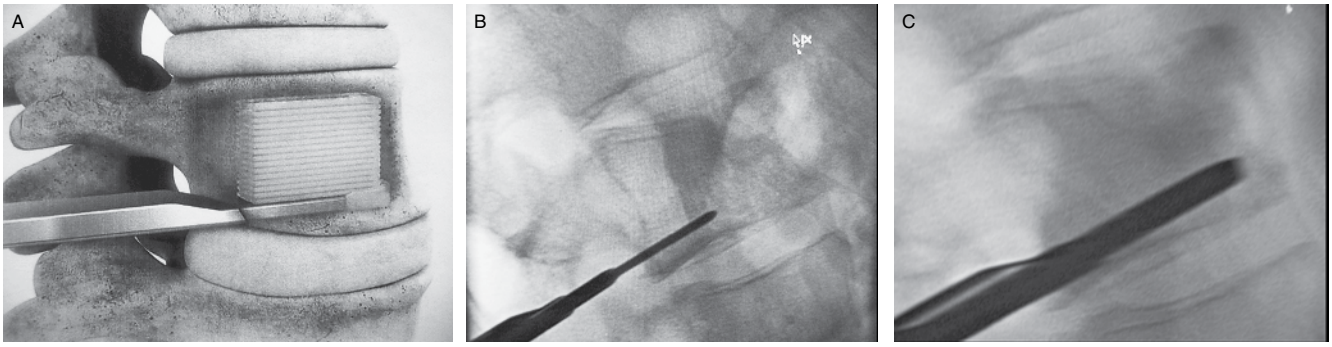

Figure 47.12 **(A)** Manufacturer's diagram (spine wave) describing the Staxx delivery technique for delivering rigid Staxx wafers for void creation and height restoration. Note the manufacturer's recommended low angle approach passing at the level of the neuroforamen. **(B)** (lateral fluoroscopic image): The patient is a 58-year-old male with an L1 vertebral compression fracture. The access is first via K wire over which the Staxx delivery device is pushed and/or hammered into the inferior vertebral body. **(C)** (lateral fluoroscopic image): Staxx wafers are then delivered to into the vertebral body prior to cementation. This patient unfortunately experienced a complicating L1 radiculopathy following the procedure as a result of compression of the nerve root from the entry device.

desirable to reduce the kyphosis resulting from VCFs if reduction is possible. In selected cases of acute fractures, soft bone from advanced osteoporosis, or lytic metastatic disease, kyphoplasty has been shown to have some degree of positive effect on vertebral fracture reduction while providing significant pain relief and improved quality of life by fracture stabilization. Finally in difficult circumstances such as severe compression or retropulsion without neurological compromise, the use of cavity creation and reduction to alter the geometry and to control cement may help to make otherwise untreatable situations treatable.

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48 Evaluation and Treatment of Pelvic Insufficiency Fractures

Douglas P. Beall, Michael Frey, and Wayne Olan

INTRODUCTION

Insufficiency fractures occur when the combined forces acting upon an osseous structure exceed its ability to withstand these forces. The compromised bone strength can result from structural disruption as is seen in tumor invasion or with infection or, more commonly, may be because of alteration of internal architecture from bone mineral loss.

Osteoporosis, which may be primary (age-related) or secondary (steroid use, smoking, rheumatoid arthritis etc.), is estimated to affect approximately 1.5 million people in the United States with worldwide costs of hip fractures alone projected to reach $131.5 billion by 2050 [1]. One in three women above the age of 50 will experience osteoporotic fractures [2]. The prevalence of this common condition is also expected to increase. By the year 2010, it is estimated that more than 52 million women and men will have either osteopenia or osteoporosis and, if current trends continue, the number will exceed 61 million by 2020 [3]. Despite it being one of the most common medical conditions, osteoporosis remains undertreated. In one study, three out of four postmenopausal women in the United States did not receive treatment during the year following an osteoporotic fracture of the hip, wrist, or vertebral body [4].

Sacral and pelvic fractures are also undertreated and under-recognized [5,6]. Patients with these fractures typically present with low back pain, hip pain, or groin pain depending on the location of the fracture. The incidence of fragility fractures of the pelvis is far less than fractures of the spine and sacrum. Previously reported as rare, these fractures are becoming more commonly recognized, and sacral fractures have more recently been reported to account for back pain in 1% to 2% of the elderly female population [6].

Nontraumatic fractures of the pelvis and sacrum have similar etiologies as do vertebral compression fractures (VCFs), and the risks of developing insufficiency fractures of the pelvis are very similar to those of VCFs. In addition to primary osteoporosis (by far the greatest risk factor for developing insufficiency fractures), there are other predisposing factors that may contribute to these fragility fractures including renal osteodystrophy, Paget's disease, hip arthroplasty, osteomalacia, and recent lumbosacral fusion [7]. Radiation therapy increases the incidence of pelvic and sacral insufficiency fractures substantially, and the 5-year cumulative incidence of pelvic insufficiency fractures can be nearly 20% in patients who have undergone radiation therapy for cervical carcinoma [8]. Overall, there is a strong female predominance (10:1) and fractures may also occur spontaneously primarily due to metabolic bone disease [5,8].

FRACTURES OF THE SACRUM AND EXTRASACRAL PELVIS

The incidence of sacral insufficiency fractures is especially common and these fractures include approximately 1% to 2% of all fractures involving the spine and pelvis [9]. Sacral insufficiency fractures, however, are more difficult to visualize on imaging studies and are more difficult to diagnose. Many sacral insufficiency fractures are not detected until much later or are either misdiagnosed or undiagnosed.

The diagnosis of pelvic and sacral insufficiency fractures has been traditionally difficult for several reasons. First, the clinical symptoms are not typical of most fractures and the pain can be described as an ache rather than a severe pain. As a response to the aching pain, some patients may shift in their seats because their back and buttock hurt, while others may experience localized pain around the sacrum or radicular pain most often in an S1 distribution. Second, the cause of the injury may not be entirely apparent. The patients will most often describe a fall on their buttocks or making a slightly jarring step, which produces subsequent severe pain in their back or buttock. The consideration of the underlying cause of the fracture is also important and it is helpful to know about the patient's osteoporotic medications, bone density (by DEXA scanning or Q CT), and history of other fractures (especially of the wrist, hip, or spine). Third, the laboratory evaluation can be fairly involved and may include many different parameters that can contribute heavily to the patient's low bone density. A thorough evaluation should include ionized calcium, parathyroid hormone and 1, 25-hydroxyvitamin D levels, thyroid function tests, alkaline phosphatase, liver enzymes, and free testosterone levels in men. In addition, there is no reliable physical examination test for the diagnosis of sacral insufficiency fractures. Important examination characteristics include a positive standing leg test, tenderness at the sacrum with

compression of the pelvis, no neurologic findings (including no weakness, no reflex changes, and normal sphincter tone), and the patient may have a slow antalgic gait.

Imaging Diagnosis of Sacral Insufficiency Fractures

The imaging diagnosis of sacral insufficiency fractures can be challenging and findings on conventional radiographs are usually subtle and may can be easily overlooked. The primary x-ray finding in sacral and pelvic insufficiency fractures is sclerosis and, in an anatomically complex area like the sacrum fractures are likely to go undetected. Bowel gas and contents also obscure bone detail of the sacrum. Early fractures of the sacrum and pelvic may not be seen until there is adequate reactive sclerosis. The most important reason that pelvic insufficiency fractures have been overlooked is a lack of awareness that insufficiency fractures occur in these areas. An article in the mid-1980s that identified a cohort of 12 patients with sacral insufficiency fractures identified more than a 4-month time period stated that "it seems most likely that this sudden small epidemic is due to recognition of the entity rather than to an actual increase in the number of sacral stress fractures" [5].

The location and radiographic appearance of sacral insufficiency fractures is characteristic and is characterized by vertical regions of sclerosis just medial to and paralleling the sacroiliac (SI) joint (Figure 48.1). The Dennis classification of sacral fractures describes three zones. Zone 1 fractures involve the lateral portion of the sacral ala but do not traverse the central sacral canal or neural foramen. The zone 1 fractures are the most common sacral insufficiency fractures and typically parallel the SI joint producing the sclerotic lines adjacent to the SI joint. Zone 2 fractures involve the sacral foramen but do not involve the central canal. Zone 3 fractures involve the central canal.

Typical sacral insufficiency fractures will parallel the SI joints and they may be unilateral or bilateral. Most commonly, the fractures will be bilateral and a horizontal component of the fracture is also seen extending between the other vertically oriented fracture (Figure 48.2) at the level of the S1-S2 junction. On a frontal view of the sacrum, the transverse portion of the fracture is located adjacent to the S1–2 junction or intervertebral disk if present (Figure 48.3).

Additional diagnostic studies may be necessary to corroborate the presence of the fractures and to further define the degree of involvement, the severity, and the amount of displacement of the fractures. Computed tomography (CT) is an appropriate modality for a cross-sectional evaluation of the pelvis and is effective at displaying the fracture along with the associated sclerosis (Figure 48.4) and at excluding a more destructive or neoplastic process. Despite the value of CT in providing anatomic information about pelvic insufficiency fractures, the sensitivity of CT for detecting these fractures is less than that of either magnetic resonance imaging (MRI) or radionuclide bone scanning [10]. CT also demonstrates the sclerosis surrounding the healing insufficiency fractures that may be misinterpreted as degenerative sclerosis surrounding the SI joints. In a study by Cabarrus et al. [11], MRI was found to be much better

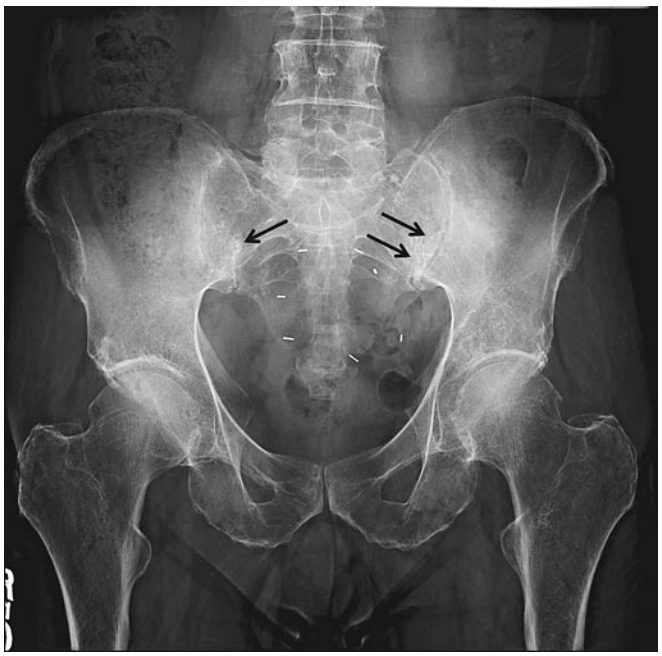

Figure 48.1 Conventional anteroposterior radiograph of the pelvis demonstrates sclerotic regions along the midportion of the sacrum paralleling the inferior portion of the sacroiliac joint (black arrows) indicative of bilateral sacral insufficiency fractures.

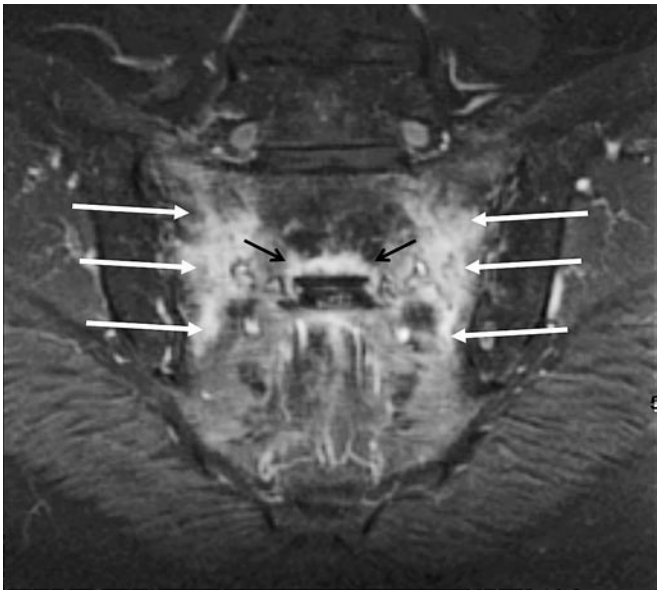

Figure 48.2 Coronal fast spin echo T2-weighted MR image shows regions of increased signal in the lateral portions of the sacrum (white arrows) consistent with sacral insufficiency fractures and a horizontal component of the sacral insufficiency fracture located centrally within the sacrum (region of increased signal located between the black arrows).

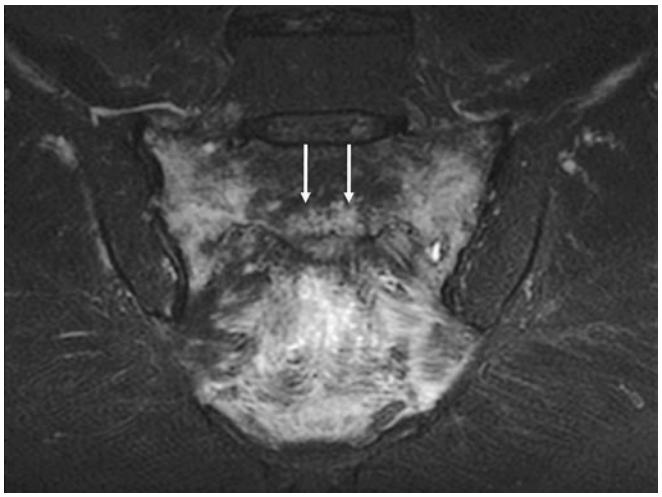

Figure 48.3 Coronal fast spin echo T2-weighted MR image shows regions of increased signal just superior and paralleling the S1–2 intervertebral disk (white arrows). This horizontally oriented region of edema represents the horizontal component of a sacral insufficiency fracture.

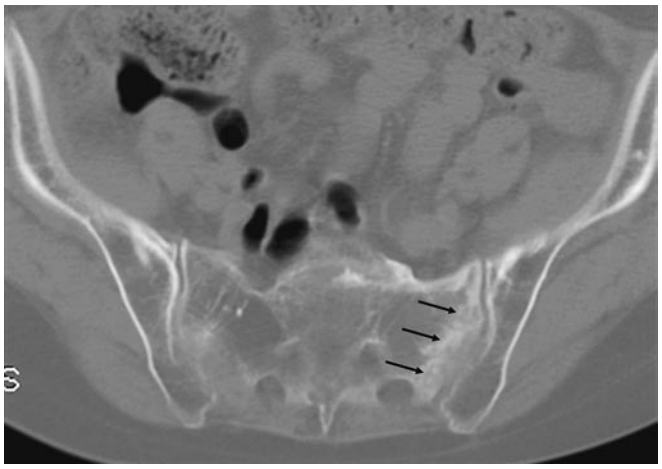

Figure 48.4 Axial CT image of the sacrum demonstrates bony sclerosis in the lateral left portion of the sacrum (black arrows) paralleling the sacroiliac joint. This sclerosis indicates a healing left-sided sacral insufficiency fracture.

in detecting pelvic insufficiency fractures [11,12]. Patients underwent both studies in this report with MRI detecting 128 of 129 (99%) of fractures and CT scanning detecting only 89 of 129 fractures (69%) [11]. The appearance of pelvic insufficiency fracture on radionuclide bone scanning is also characteristic but is less specific because there is increased osseous uptake in anything that will involve the bone and cause inflammatory change or bone destruction. In the appropriate clinical setting, increased uptake bilaterally in the upper sacrum should strongly suggest the presence of sacral insufficiency fractures but sacroiliitis could produce a similar distribution of abnormal uptake. This diagnosis would, of course, be very unusual in an elderly female.

In patients who are at risk of pelvic insufficiency fractures, the most common cause of increased uptake in the axial skeleton is metastatic disease, and correlation of the bone scan with other imaging modalities is important to achieve the correct diagnosis. In the detection of insufficiency fractures, it must also be kept in mind that it may take up to 3 days after the occurrence of the fractures before increased uptake is noted on the bone scan. When the timing of the study is appropriate, bone scanning with technetium Tc99m–labeled methylene diphosphonate is one of the most sensitive examinations for the detection of pelvic insufficiency fractures. An H-shaped pattern of increased uptake within the sacrum is characteristic of sacral insufficiency fractures (this pattern has been also called the "Honda sign"). The reported sensitivity for the detection of sacral insufficiency fractures is 96% and the positive predictive value is 92% [13]. The most effective modalities to detect pelvic and sacral insufficiency fractures are nuclear scintigraphy and MRI, and based on the body of imaging literature, the single best modality for evaluating both the anatomic appearance and for the presence of pelvic insufficiency fractures is MRI [10]. Sacral and pelvic insufficiency fractures usually appear much the same as other osseous fractures with a linear region of decreased signal on the T1-weighted images (indicating the fracture line) and surrounding areas of increased signal intensity on the T2-weighted images indicating edema around the fracture (Figures 48.5a and 48.5b). Fractures of the sacrum are best demonstrated on coronal planes with a dedicated MRI of the sacrum and are best seen on the fluid sensitive sequences such as fat-suppressed T2-weighted images or STIR images. A routine MRI examination of the lumbar spine may not optimally demonstrate sacral insufficiency fractures, and it is important to extend the axial imaging inferior through at least the first sacral vertebrae and to be cognizant of the sacrum on the sagittal images so as not to miss sacral fractures that may be shown on the periphery of an MRI examination of the lumbar spine.

During the diagnostic process, it must be kept in mind that sacral fractures are directly related to fractures of the pubic rami. The superior and inferior pubic rami are most commonly fractured as the result of direct trauma but are

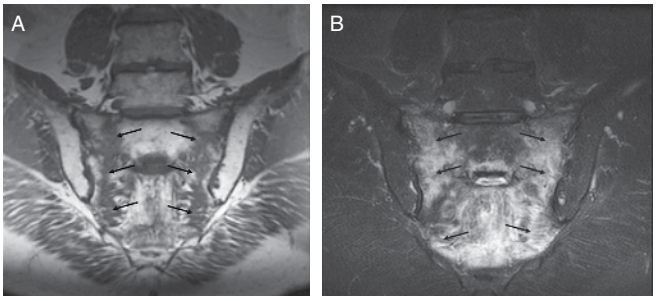

Figure 48.5 (**A**) Coronal T1-weighted MR image of the sacrum shows vertically oriented linear regions of decreased signal within the lateral portion of the sacrum (black arrows) indicative of sacral insufficiency fractures. (**B**) Coronal T2-weighted MR image of the sacrum in the same slice position as Figure 48.5A demonstrates prominent regions of increased signal surrounding the fractures (black arrows). These foci of increased signal represent the osseous edema surrounding the fractures.

susceptible to osteoporotic insufficiency fractures, especially when associated with sacral insufficiency fractures. These fractures tend to occur together and fractures of either the sacrum or pubic rami indicate an increased risk of developing an insufficiency fracture of the unfractured portion of the pelvis. When fractures of the pubic rami or the sacrum are identified, the opposite pubic ramus or the sacrum should be closely evaluated for the possibility of fracture.

TREATMENT OF PELVIC INSUFFICIENCY FRACTURES

Conventional treatment for pelvic and sacral fractures varies according to the patient's age, the condition of the bone, and the presence of comorbidities. If the bone is sufficiently strong and can hold hardware, and if the fracture can be reduced then it is typically repaired with plate and screw fixation. Conservative therapy is traditionally applied to patients who cannot tolerate surgical intervention or if the fracture does not involve weight bearing portions of the pelvis [14,15]. Conservative treatments for osteoporotic fractures focus on pain relief through use of narcotics, analgesics, bed rest, and external bracing with lumbosacral or pelvic corsets. Rehabilitation with early mobilization, partial weight-bearing, and the use of a walker is typically employed and progressive exercise is utilized after the patient is able to tolerate this activity. Despite early conservative treatment with the measures described earlier, pain relief and return to prior mobility may take anywhere from few weeks to several months [16].

Fractures of the spine and pelvis are also associated with significant risks of morbidity and mortality, and this is one of the reasons to consider invasive treatment in patients who may not be able to tolerate conservative therapy. The relative risk of mortality from VCFs can be as high as eight to nine times higher than age-matched controls, and the risk of mortality increases with increasing numbers of VCFs [17,18]. This risk is matched by insufficiency fractures of the pelvis and, when these occur, up to half of the patients will not recover their previous level of functioning, up to 25% of patients will require long-term tertiary care, and nearly one in seven patients will die from complications associated with their sacral insufficiency fractures [19]. Nearly one in seven patients in this patient population died during the first year of conservative therapy [19]. The complications from pelvic insufficiency fractures and the resulting immobilization include deep venous thrombosis, pulmonary emboli, reduced muscle strength, postural hypotension, impaired cardiac function, constipation and fecal impaction, pressure ulcers, depression, and pulmonary complications such as atelectasis and pneumonia [7,20]. Clinical improvement with conservative therapy may occur rapidly but complete resolution of symptoms may not occur for up to 9 to 12 months [19,20]. In patients who fail conservative therapy or cannot tolerate conservative therapy, osseous augmentation with acrylic cements (i.e., polymethylmethacrylate [PMMA]) have been used for several decades to stabilize the fractures of the axial skeleton [8]. Percutaneous administration of PMMA for treatment of insufficiency fractures located in the vertebral bodies has been shown by numerous independent researchers to be a safe, effective means of providing pain relief [9,11,21]. More recent evidence has suggested that osseous augmentation with acrylic cements is also effective for other portions of the axial skeleton, specifically the sacrum and pelvis [22–24]. This augmentation of the skeleton outside of the vertebral bodies may provide an additional treatment method that expands the existing methods of conservative treatment (which is typically applied to osteoporotic pelvic fractures) and to plate and screw fixation (which is typically applied to traumatic fractures in patients with normal bone mineral density).

The principle applied to osseous augmentation with the injection of acrylic liquids is that of stabilization via the liquid that flows into the fractures and provides mechanical stability to the fractured bone itself [8]. The injected liquid will, most often, also fuse the fractured bone to the surrounding intact bone. The injected material has a large surface area of contact with the osseous material it is injected into because of the very porous nature of medullary or cancellous bone. When the liquid hardens, it forms an internal strut that is well anchored into the underlying cancellous bone. This internal position of the injected material and its large surface contact area with the underlying bone provides for optimal support even in severely osteoporotic bone [25].

In this chapter, we will discuss further the principles and techniques for image guided osseous augmentation of insufficiency fractures of axial and appendicular skeleton. We will also describe the patient selection, diagnostic criteria, potential risks, complications, and techniques to achieve optimal outcomes.

Osseous Augmentation

Osseous augmentation of insufficiency fractures began with the treatment of VCFs. The technique was first performed in France in 1984 when PMMA was used to stabilize the C2 vertebral body that contained a prominent and symptomatic hemangioma [26]. Since this initial report, the use of vertebral augmentation with PMMA has become widespread, especially for the treatment of benign osteoporotic VCFs, and the efficacy of this technique has been well documented [27]. Percutaneous augmentation of VCFs has been shown to significantly decrease pain and discomfort and subsequently have a positive effect on early mobility, quality of life, and participation in activities of daily living [28,29]. It has also shown to be very safe and is a minimally invasive procedure that may be done under conscious sedation rather than general anesthesia. Vertebral augmentation with PMMA has played a very important role in the care of patients who respond poorly to conservative medical therapy as well as those who are poor candidates for more invasive surgical treatments.

Insufficiency fractures of the pelvis occur in the same patient population as those affected by VCFs. Pelvic (including sacral) insufficiency fractures usually heal uneventfully without operative intervention, but elderly patients may be too fragile to tolerate the required immobilization and the protracted hospital stay that is typically seen with this patient population, and the process of hospitalization

itself utilizes substantial health care resources [21]. In patients with intractable pain or in patients who cannot tolerate conservative therapy, percutaneous osseous augmentation with PMMA has been provisionally shown to be a viable treatment alternative for patients with pelvic insufficiency fractures [24].

Although the long-term results are not known, a number of studies have shown sacroplasty to be an effective means of pain reduction and a way to facilitate early mobility [11,22–24]. There are also reports of percutaneous augmentation of pelvic insufficiency fractures including pubic rami, acetabulum, and ilium fractures [24,30].

Sacroplasty

Sacroplasty is the injection of an osseous fill material (usually PMMA) into the interstices of the medullary or cancellous bone of the fractured sacrum at the S1 and S2 vertebral levels (because these are the vertebral levels that are most commonly, and most severely, fractured). This procedure may be performed under fluoroscopic or CT guidance and is technically very similar to a vertebroplasty.

Preliminary reports regarding the efficacy of sacroplasty are very encouraging as to the efficacy of this technique. Sacroplasty was first reported in 2001 because treatment of symptomatic sacral metastatic lesions and subsequent reports have documented its safe and effective performance [19,31,32]. Although the initial studies were promising, the short follow-up intervals and relatively small study cohorts preclude a more definitive commentary regarding the safety of the procedure and the durability of initial results [22,33,34].

In 2007, Frey, DePalma et al. published a prospective multicenter study designed to assess the safety and efficacy of sacroplasty, to better define the incidence of complications, and to evaluate the clinical utility of sacroplasty in treating painful osteoporotic sacral insufficiency fractures [22]. The mean age of patients in this study was 76.6 years and they had experienced a failure of conservative care for a mean of 34.4 days. The mean Visual Analogue Score (VAS) at baseline was 7.7, which decreased to 3.2 within 30 minutes after the procedure and was 0.7 at 1 year following the procedure with a steady decline in the interval. These patients experienced a significant reduction in narcotic use and there were no lasting complications as a result of the procedure, and the degree of patient improvement was similar to published reports on vertebroplasty [8,16,25,26].

Other reports have corroborated the safety and efficacy of sacroplasty [11,22–25]. Although the potential risks for the sacroplasty procedure are similar to that of vertebroplasty and include cement extravasation into the presacral space or around the nerve roots, cement emboli, and leakage into the epidural space, there are other potential complications specific to sacroplasty including penetration of the cephalad or superior margin of the sacral ala, or penetration of the anterior cortex with extravasation around the lumbosacral plexus. Despite these potential complications in practice, this is a safe procedure with few reported complications.

In 2008 Frey, DePalma et al. published an additional study that corroborated the previously reported safety and efficacy of sacroplasty. This study described that more than 75% of the patients had their pain reduced by more than half within 30 minutes following the procedure [23]. The authors also reported additional longer-term follow-up on some of their previous patients. As the number of patients undergoing sacroplasty increases and as the length of time these patients are followed up lengthens, the weight of evidence indicates that this is a viable and durable technique for treating patients with sacral insufficiency fractures that are not candidates for (or have failed) conservative therapy.

Sacroplasty Technique

Sacroplasties are most often performed under conscious sedation using fentanyl and midazolam (or other intravenous sedatives) as the agents for providing the sedation. Preoperative antibiotics are often given to the patient and usually include medications that provide coverage for gram-positive bacteria (i.e., cefazolin or clindamycin) and for gram-negative bacteria (i.e., gentamicin).

There are several techniques for performing percutaneous sacroplasty that have been described in the literature but, typically, this procedure is performed with either CT or fluoroscopic guidance [22,23,35]. CT fluoroscopy may also be used as a method to guide the procedure [36]. When the sacrum and the fractures therein are visualized via CT, the needle entry site may vary according to the location of the fracture lines. The horizontal component of the sacral insufficiency fracture, for example, can be accessed by placing the needle posterolaterally through the SI joint. Other alternative access sites are through the sacral ala with angulation of the needle between the spinal canal and the ipsilateral sacral foramen. Some authors have proposed that the use of CT fluoroscopy is superior to that of conventional fluoroscopy to reduce complication rate and improve clinical outcomes [37].

The choice as to which imaging guidance is used is mostly according to operator comfort with the particular modality. Whitlow examined the technical considerations of needle placement and PMMA extravasation while injecting the sacrum with bone cement under fluoroscopy [35]. He performed sacroplasty on cadaveric specimens under biplane fluoroscopy and analyzed these specimens with CT both before and after the injection of PMMA into the sacral ala. The follow-up CTs that were performed demonstrated that safe needle placement and PMMA delivery may be facilitated by orienting the needle parallel to the L5-S1 interspace and to the ipsilateral SI joint (Figure 48.6). When the appropriate alignment was achieved, the targeting was focused at the superolateral sacral ala within the area bounded by a line lateral to the posterior foraminal openings and a line superimposed on the medial edge of the SI joint (Figure 48.7).

The Whitlow technique, applied to patients has been called the short-axis technique as the needles are injected directly into the posterior portion of the sacrum at approximately 90° to the long axis of the sacrum (Figure 48.8) thereby accessing the short axis of the sacrum. The PMMA is injected into the sacrum focally and needles may be used bilaterally to obtain access to both the S1 and S2 segments

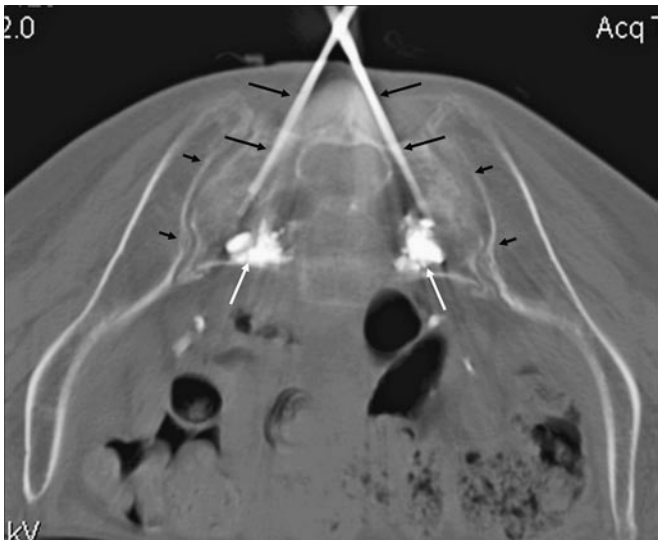

Figure 48.6 Axial CT obtained with the patient in the prone position shows needle placement into the medullar bone within the sacral ala bilaterally (long black arrows). The needles are oriented parallel to the ipsilateral sacroiliac joints (short black arrows). Bone cement is seen at the needle tips within the anterior portion of the sacrum (white arrows).

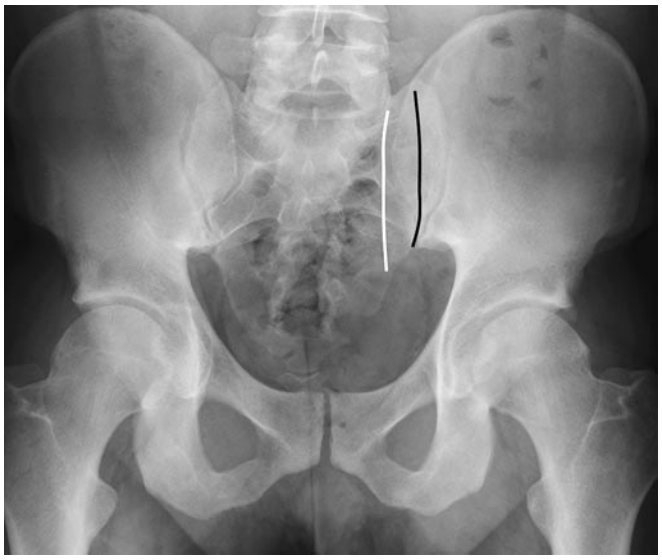

Figure 48.7 Anteroposterior conventional radiograph of the pelvis shows the area of the sacral ala on the patient's left side that is appropriate for target for sacroplasty. The medial border is defined by a line drawn along the lateral portion of the sacral foramina (white line) and the lateral border is indicated by a line drawn along the medial border of the sacroiliac joint (black line). The portion of the sacrum that is visualized in this radiographic orientation is primarily the S1 and S2 levels.

of the pelvis for a total of four needles. The needles are placed with the image intensifier either directly anteroposterior (AP) or with slight caudal angulation to improve the en face visualization of the sacrum and the needle tips are placed lateral to the lateral portion of the sacral foramina and medial to the SI joint. The image intensifier

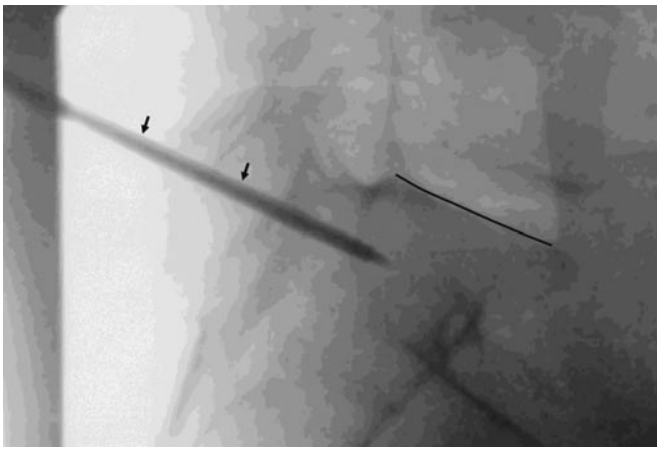

Figure 48.8 Lateral fluoroscopic view of the sacrum shows a needle (short black arrows) entering the S1 segment of the sacrum. The needle is oriented parallel to the superior endplate of S1 (black line).

is angled 25° to 30° contralateral to the side of the sacrum being accessed or at whatever angle allows a view along the long axis of the SI joint. The needle placement should initially be made from the oblique AP view and the lateral view is always used to ensure that the tip of the needle is in the central one-third of the sacrum (Figures 48.9a and 48.9b). The cement is injected with a technique very similar to that used in vertebroplasty. The control of the PMMA is optimal when it is thick and of toothpaste-like consistency. Direct fluoroscopic observation is used to ensure that the cement is placed into the center of the S1 and S2 segments and does not extravasate into unwanted locations such as medially into the location of the neural foramina, anterior to the sacral ala (location of the lumbosacral plexus), and intravascularly. In fractures with a horizontal component through the inferior S1 segment or through the S1-S2 interspace, cement can be seen to extend medially into the fractured bone between the levels of the S1 and S2 foramina (Figure 48.10). Although extension of cement into the SI joint is considered suboptimal, this is often unavoidable and is of questionable significance as it typically stays confined within the joint itself.

The long-axis technique is used to access both the S1 and S2 segments with one needle on each side (two needles totally) and is directed from inferior to superior at an oblique angle along the long axis of the sacrum (Figure 48.11) [38]. The goals when originally developing the long-axis approach were to improve cement distribution along the fracture lines and to decrease inadvertent perforation of the anterior sacral cortex [38]. The initial placement of the needle is at the inferior border of the S2 segment in a similar mediolateral position to the short-axis technique with the needle tip between the lateral border of the sacral foramina and the medial border of the SI joint. The needle is then directed from the inferior and posterior portion of the S2 vertebrae to the central and anterior portion of the S1 vertebrae (Figure 48.12). The normal lordosis of the lumbosacral junction, the dorsally tilted sacrum, the prone position of the patient all facilitate the appropriate superoinferior angle

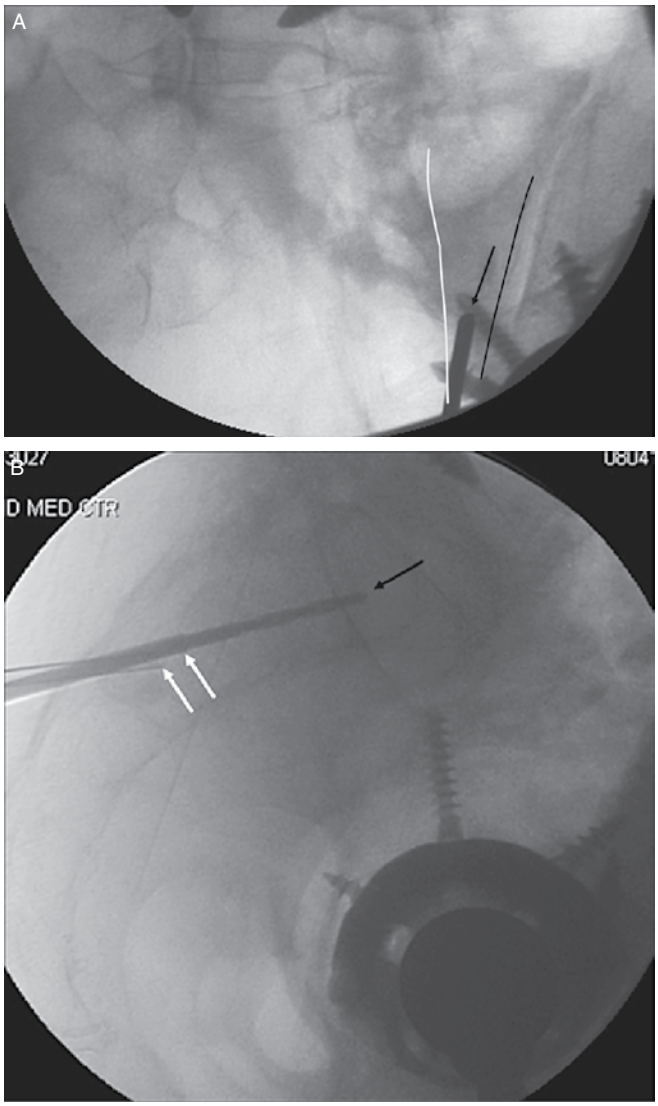

Figure 48.9 (**A**) Oblique posteroanterior fluoroscopic view oriented along the sacroiliac joint with 15° of caudal angulation. The tip of the needle (black arrow) is placed between the medial border of the sacroiliac joint (black line) and the lateral border of the sacral foramen (white line) at the inferior margin of the sacrum that is visualized with this angulation. (**B**) Lateral right-sided fluoroscopic view of the sacrum with two needles in place at the posteroinferior portion of the S2 vertebral segment (white arrows) and an osseous drill placed through the left needle with the tip located slightly superior to the center of the S1 vertebral segment (black arrow). The trajectory of the needles is located through the approximate anteroposterior center of the sacrum.

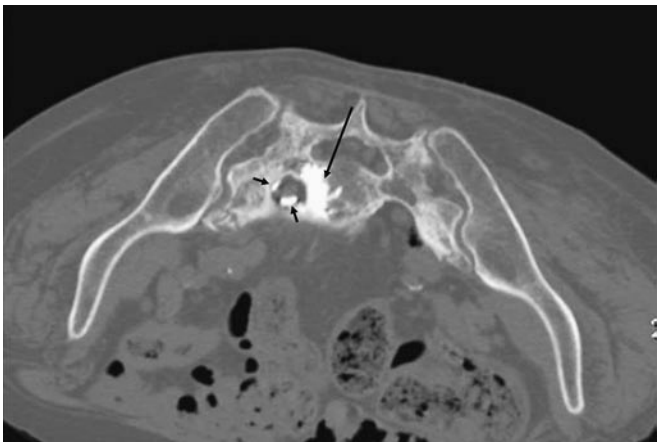

Figure 48.10 Axial CT image at the S1-S2 level obtained with the patient in the prone position shows bone cement extending into the center of the sacrum through the horizontal component of this sacral insufficiency fracture (long black arrow). There is also small amounts of bone cement that has extravasated into the left S1 neural foramen (short black arrows). Despite this extravasation, the patient was asymptomatic.

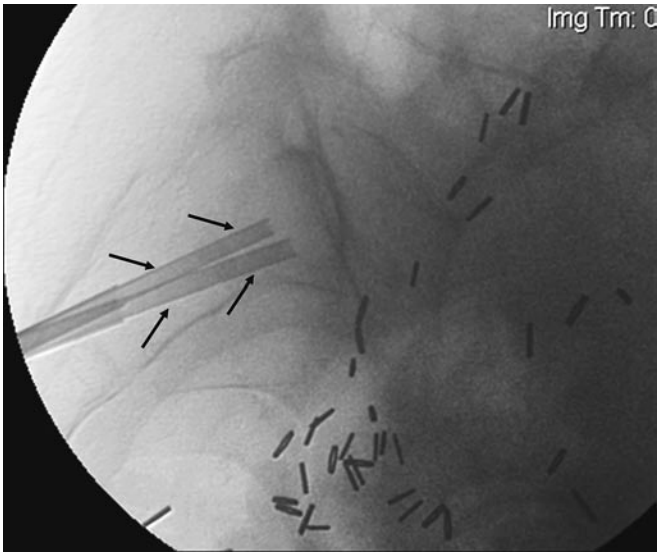

Figure 48.11 Lateral fluoroscopic view of the sacrum shows bone fillers extending from the posteroinferior portion of the S2 vertebral segment to the midanterior portion of the S1 vertebral body (black arrows). This trajectory is oriented through the longitudinal axis of the sacral ala.

of the needle and it is therefore not technically difficult to achieve this degree of superoinferior angulation. Care should be taken not to penetrate the anterior cortex of the S1 or S2 vertebrae as the lumbosacral plexus is located in this region and anterior extravasation from a disrupted anterior cortex can be difficult to visualize and/or control. When the needles are placed and while using the same technical considerations regarding the consistency of the cement and the observation of the cement injection, the sacrum is filled with PMMA from distal to proximal. The steps of the long-axis approach are listed in Table 48.1 and the steps of the short-axis approach are listed in Table 48.2.

Osteoplasty of the Pelvis

Pubic Ramoplasty

Fractures of the pubic rami are very commonly found in conjunction with fractures of the sacrum and have been seen together in 88% of patients [39]. It has been thought

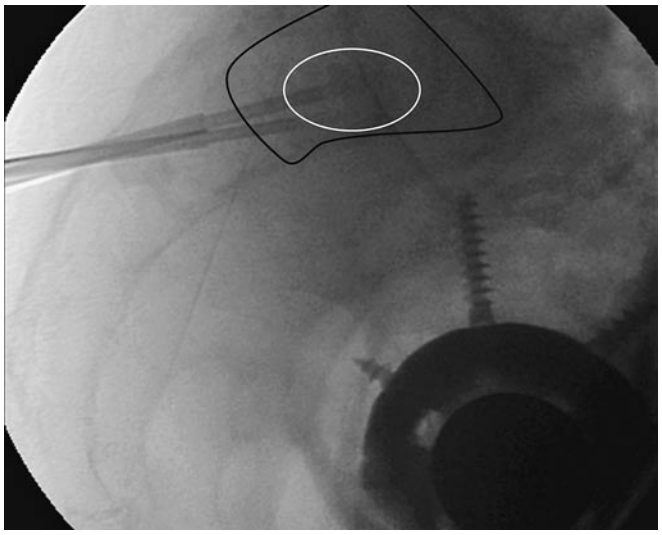

Figure 48.12 Lateral fluoroscopic view of the sacrum shows bone fillers directed to the central and anterior portions of the S1 vertebrae (outlined with black line) with bone cement in place in this location (white oval).

that the sacrum is the initial site of the insufficiency fracture and that this results in an increased amount of stress on the remainder of the osteoporotic pelvis. By the time the patient presents with low back pain typical of that of a sacral insufficiency fracture, they will commonly also have groin pain that is characteristic of pubic rami fractures.

The first description of osteoplasty of the pubic ramus for insufficiency fractures was reported in 2007 when two cases of direct injection of PMMA into the superior pubic ramus and parasymphyseal region was effective for treating pain caused by a chronic superior pubic ramus in one patient and an acute superior pubic ramus in the other [40]. Both of these patients experienced a dramatic decrease in their pain and corresponding increases in their ability to ambulate and they had full and uneventful recoveries from their fractures [40]. Based on these outcomes the authors suggest that this may be a viable alternative to conservative therapy for pubic rami fractures when other conservative therapies fail.

Osteoplasties of the pelvis can prove more technically challenging primarily because of the lack of established percutaneous access pathways to the fracture sites and the

Table 48.1 Step by Step Sacroplasty Using the Long-Axis Technique

1. In the frontal plane, rotate the II cephalad to an AP plane
2. Ensure that a direct AP view is obtained by rotating the II to place the spinous processes in the center of the vertebral body
3. Rotate the II to the side opposite the treatment location to approximately 25° to 30° or to an angle along the longitudinal axis of the sacroiliac joint that aligns the inferior portion of the joint
4. Choose a starting point halfway between the sacroiliac joint and a vertical line that joins the medial borders of the sacral neural foramina
5. Anesthetize the skin in the location of the starting point and make a stab incision with a No. 11 blade scalpel
6. Insert the needle parallel to the II and angle the needle tip 20° to 40° cranially
7. Using a mallet, advance the needle tip just past the posterior sacral cortex
8. Rotate the II to get a lateral view of the sacrum and adjust the superoinferior angulation of the needle so it can be advanced to the level of the superior portion of the S1 vertebral body
9. Return to the angled AP view and ensure that the mediolateral angulation of the needle is appropriate
10. Return to the lateral view and advance the needle to the level of the superior portion of the S1 vertebral body
11. When the PMMA has a toothpaste consistency, inject it into the sacrum using both the lateral and the oblique AP views to monitor the PMMA injection. There should be no anterior extravasation (anterior to the anterior border of the sacrum) as seen on the lateral view and there should be little to no extravasation into the neural foramina as seen on the oblique AP view
12. Place the cement in the sacrum trying to fill the center portion of the sacral ala (between the SI joint and the neural foramina) and intermittently retract the needle from distal to proximal while placing the PMMA along the longitudinal axis of the sacrum
13. When the PMMA has been placed into the sacral ala, remove the needles and place the appropriate bandages over the puncture sites

Abbreviations: AP, anteroposterior; II, image intensifier; PMMA, polymethylmethacrylate

Table 48.2 Step by Step Sacroplasty Using the Short-Axis Technique

1. In the frontal plane, rotate the II cephalad to parallel L5-S1 disc space. This will require significant caudal angulation of the II
2. Ensure that a direct AP view is obtained by rotating the II to place the spinous processes in the center of the vertebral body
3. Rotate the II to the side opposite the treatment location to approximately 25° to 30° or to an angle along the longitudinal axis of the sacroiliac joint that aligns the inferior portion of the joint
4. Choose a starting point in the upper half of the sacrum halfway between the sacroiliac joint and a vertical line that joins the medial borders of the sacral neural foramina
5. Anesthetize the skin in the location of the starting point and make a stab incision with a No. 11 blade scalpel
6. Insert the needle parallel to the II
7. Using a mallet, advance the needle tip just past the posterior sacral cortex and into the center one-third of the sacrum
8. Rotate the II to get a lateral view of the sacrum and the needle within the S1 vertebral body
9. When the PMMA has a toothpaste consistency, inject it into the sacrum using both the lateral and the oblique AP views to monitor the PMMA injection. There should be no anterior extravasation (anterior to the anterior border of the sacrum) as seen on the lateral view and there should be little to no extravasation into the neural foramina as seen on the oblique AP view
10. Place the cement in the sacrum trying to fill the center portion of the sacral ala (between the SI joint and the neural foramina) and intermittently retract the needle from distal to proximal while placing the PMMA along the longitudinal axis of the sacrum
11. If necessary, insert a second needle into the inferior half of the sacrum with the same mediolateral positioning as the first needle. Repeat the visualization and injection process as with the first needle
12. When the PMMA has been placed into the sacral ala, remove the needles and place the appropriate bandages over the puncture sites

Abbreviations: AP, anteroposterior; II, image intensifier; PMMA, polymethylmethacrylate.

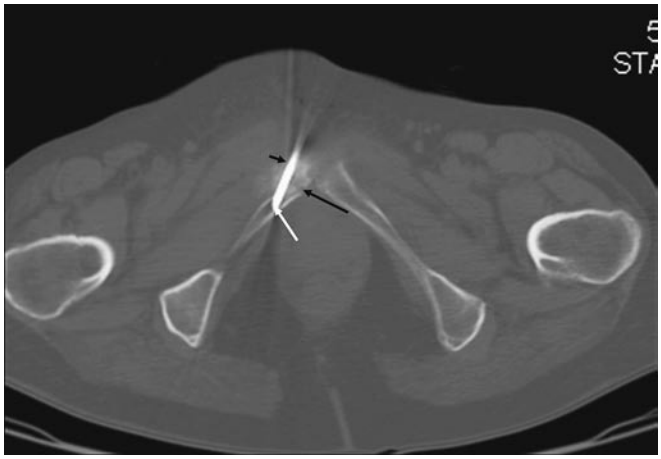

Figure 48.13 Axial CT image with needle in place (short black arrow) demonstrating the technique of advancing the needle tip (white arrow) across the fracture site (long black arrow). The pubic ramus will then have bone cement added to the medullary portion of the bone and the cementing will be performed from distal to proximal to include the fracture itself with a goal of providing a strut of stability across the fracture.

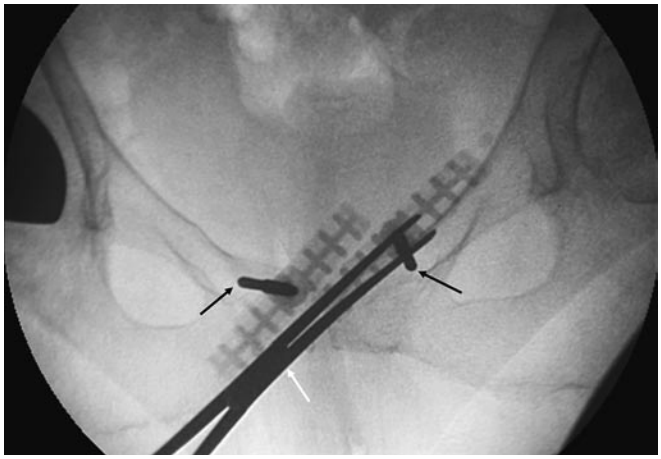

Figure 48.14 Anteroposterior fluoroscopic image showing needles in place with the tips (black arrows) in the medial portions of the superior pubic rami. A Kocher (white arrow) functions as a needle holder.

relatively decreased amount of cancellous bone as compared with vertebral bodies. Fluoroscopic and CT-guided techniques have been described for the sacrum but there are no standard percutaneous approaches to the pubic rami, the superior pubic root, or the supra-acetabular ilium. The use of CT offers direct visualization of the underlying structures, and safe and effective approaches to the pelvis have been introduced with CT and then converted to a fluoroscopically guided procedure when a greater degree of familiarity with the approach was gained [24].

It is also uncertain as to the implications of cement extravasation because the pelvic fracture sites are typically located farther away from critical neural structures than when performing vertebral augmentation with PMMA. Extravasation into the hip joint, around the lumbosacral plexus, or into the sacral foramina would be obvious untoward events but the implications of extravasation into many of the surrounding soft tissue structures of the pelvis remains uncertain. The risk for cement extravasation may also be elevated given the ease with which cement fills the fracture site and its adjacent structures without encountering significant feedback pressure from the injection system [35].

Pubic Ramoplasty Technique

The technique described initially involves CT guidance and placing an 11-gauge needle through the outer cortex and up to the fracture site, drilling through the fracture, and cementing back across it. Subsequent techniques for osteoplasty of the pubic rami have adopted a fluoroscopically guided approach that involves the penetration of the anterior cortical bone to access the underlying medullary bone and injecting the PMMA into the medullary bone until it crosses the fracture site (Figure 48.13) [24].

The access to the fractured pubic rami is usually not difficult and access point is typically in the medial half of the ramus (Figure 48.14). The inferior epigastric and the femoral neurovascular bundles are located lateral to this access point and the obturator and internal pudendal bundles are central to the pubic ramus. The external pudendal artery arises from the medial portion of the proximal femoral artery and can be located in the access pathway to the superior pubic ramus. Despite the anatomic location of the external pudendal artery, the vessel is small and does not appear to pose a significant risk to the anterior access of the pubic ramus. The pubic ramus is typically relatively superficial and a large bore needle can be used to access the medullary bone in the middle of the ipsilateral parasymphyseal pubis and a second needle can be used to access the superior pubic ramus in the location of the pubic tubercle (Figure 48.15).

Fractures also commonly involve the superior pubic root (which is located at the iliopubic junction). The needle located in the center of the pubic ramus can typically be directed laterally into the lateral portion of the superior pubic ramus and the PMMA will typically flow into the superior pubic root and into the medial portion of the superior acetabulum (Figure 48.16).

Acetabuloplasty and Ilioplasty

Fractures of the acetabulum and ilium are usually associated with severe osteoporosis, previous radiation therapy, or surgical anatomic alterations of the pelvis (i.e., large iliac bone graft donor sites) [19]. Patients who present with acetabular insufficiency fractures will typically complain of hip pain that can be indistinguishable from pain originating from the hip joint itself. Patients with ilium fractures usually complain of low back pain or pain in the posterior hip.

The diagnosis of acetabular and ilium fractures can be difficult, especially in patients with previous malignancy

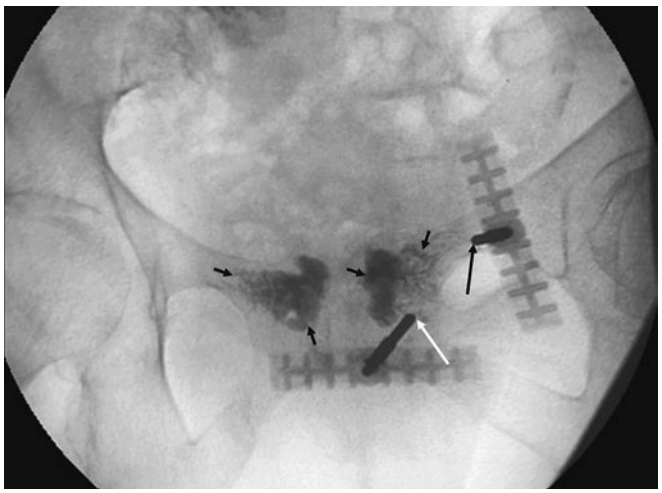

Figure 48.15 Anteroposterior fluoroscopic view demonstrating needles in place with one needle tip placed in the midportion of the superior pubic ramus (long black arrow) and the second needle tip place in the midportion of the parasymphyseal pubis (white arrow). Bone cement has previously been added to the pubic rami (short black arrows).

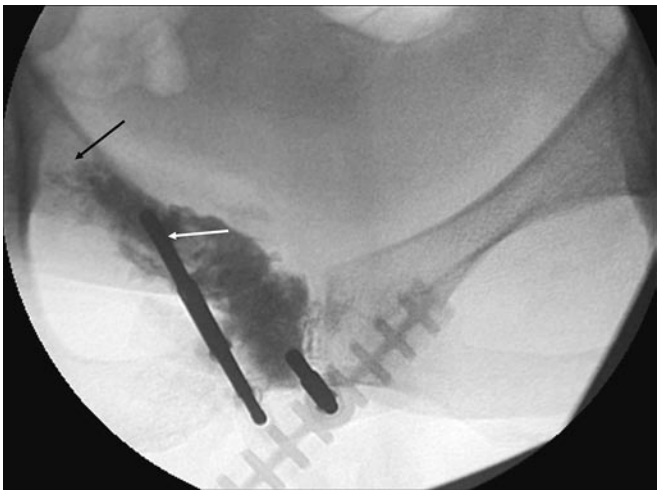

Figure 48.16 Anteroposterior fluoroscopic view of the pubic rami shows a needle in the lateral portion of the right pubic ramus (white arrow) directed laterally toward the ipsilateral pubic root. The bone cement injected into this region will flow into the superior pubic root (black arrow) and further on into the medial portion of the acetabulum.

or prior radiation therapy. As with vertebral and sacral fractures, MRI is the modality best suited for diagnosing insufficiency fractures of the extrasacral pelvis [13,39]. As with fractures in the sacrum, there is typically a characteristic linear region of low signal intensity on the T1-weighted images and increased signal consistent with edema on the T2-weighted images. In the acetabulum, this signal abnormality is usually oblique or curvilinear, parallels the roof of the acetabulum, and is present in the supra-acetabular ilium [39]. CT or conventional radiographs may show sclerosis in the supra-acetabular ilium in a similar location but are not as sensitive as MRI. As with VCFs, nuclear medicine bone scintigraphy is very sensitive for detecting fractures but lacks the anatomic information that is supplied by cross-sectional imaging. If the patient is unable to undergo MRI, a combination of bone scintigraphy and CT scanning is effective for the detection and demonstration of the fracture, respectively.

Ilium fractures also have typical locations [41]. Some fractures extend diagonally across the ilium originating from the greater sciatic notch and are known as oblique iliac fractures, whereas other fractures are located adjacent to the SI joint paralleling the joint itself and are known as superomedial iliac fractures (Figure 48.17). Fractures of the ilium are identical in appearance to other insufficiency fractures of the pelvis on the various imaging studies that demonstrate them. Knowledge of the typical locations and appearances of these fractures will prevent diagnostic confusion when presented with these types of insufficiency fractures.

After identification of insufficiency fractures of the acetabulum or ilium, patients will most often be treated with conservative therapy including analgesics, narcotics, bedrest, pelvic corset bracing, and early mobilization.

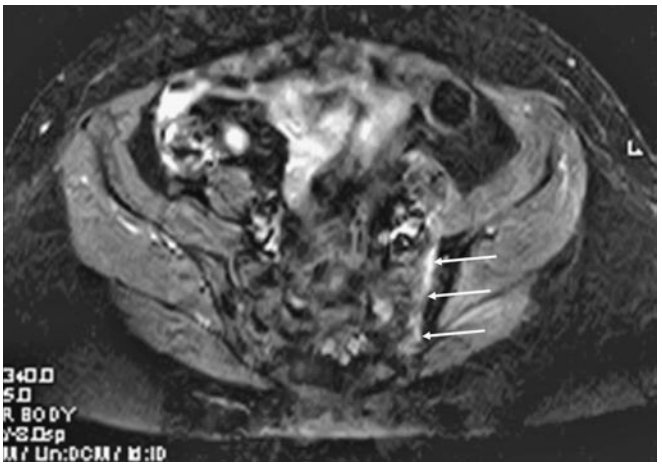

Figure 48.17 Axial STIR MR image shows a left-sided ilium fracture appearing as a linear region of increased signal (white arrows) paralleling, and lateral to, the left sacroiliac joint. This fracture is known as a superomedial iliac fracture.

Patients who do not heal their fractures and demonstrate subsequent clinical improvement may be candidates for percutaneous osseous augmentation.

Reports of the efficacy of osteoplasty of pelvic insufficiency fractures are uncommon. The authors of the largest series of percutaneous osteoplasty for pelvic insufficiency fractures reported 40 consecutive fractures that were treated with osseous augmentation with PMMA [24]. The fractures included sacral (n=26) and nonsacral (n = 14) fractures. The nonsacral group included 10 pubic ramoplasties, 3 ilioplasties, and 1 acetabuloplasty. The VAS for pain status was reduced from a mean of 8.9 (preprocedure) to 2.0 in the sacral fracture group and from 8.0 to 2.7 in the nonsacral fracture group. The difference

in VAS score was found to be highly significant in both groups ($P < 0.001$). No major procedure-related complications such infection, injury to adjacent structures, or hematoma were seen.

To our knowledge, there has been no reported complications related to pelvic augmentation osteoplasty. Theoretical complications include extravasation of PMMA into the surrounding soft tissues or vasculature, hematoma formation, puncture of underlying structures, infection, or leakage of PMMA into the hip joint. Neurovascular injury or compromise would also be possible if sufficient PMMA would extravasate into specific locations such as the femoral tunnel. Real-time or repetitive intermittent visualization with fluoroscopy of CT while injecting the fill material is imperative to identify areas of early extravasation. The implications of extravasation have yet to be adequately described.

Acetabuloplasty and Ilioplasty Technique

Osteoplasty of the acetabulum (acetabuloplasty) and of the ilium (ilioplasty) may be performed with either fluoroscopic or CT guidance [24]. Access to the anatomic region of the acetabulum that usually sustains an insufficiency fracture (the supra-acetabular ilium) is most often achieved via an anterior or anterolateral approach (Figures 48.18a and 48.18b). A safe approach to the acetabulum involves an approach that avoids important anatomic structures such as the femoral neurovascular bundle, the hip joint, the sciatic nerve, and the obturator nerve and its branches (Figure 48.19). The target is the body of the ilium that is at approximately the level of the anterior inferior iliac spine (AIIS) and that is between the inferior gluteal line and the insertion point of the articular capsule of the hip joint (Figure 48.20). The appropriate portion of the ilium can be easily and safely targeted by using CT as the imaging guidance modality of choice. The needle is advanced to the anterior or anterolateral cortex of the ilium and tapped into the outer portion of the osseous cortex with a mallet. When a safe trajectory is confirmed, the needle(s) is advanced into the medullary bone and bone cement is injected into the interstices of the ilium (Figure 48.21). An alternative to the use of CT is fluoroscopic guidance. Using the lateral border of the body of the ilium at the level of the inferior AIIS as a landmark, the operator can effectively avoid the femoral neurovascular bundle that typically crosses the medial one-third to one half of the femoral head and lies even more medially at the level of the AIIS. Either one or two needles may be inserted into the medullary bone of the body of the ilium (Figure 48.22). Once the needles are in place, the cement may be injected into the interstices of the medullary bone. Real-time or rapid intermittent observation of the injection process is of the utmost importance to detect extravasation and to monitor the flow of the PMMA. The goal of the injection process is to place the PMMA into the location of the fracture so as to provide stabilization to the heretofore fractured bone (Figures 48.23a and 48.23b).

Postprocedure care is limited to coverage of the puncture wounds with steri-strips or a similar adhesive occlusive dressing and a temporary absorbent layer (such as surgical gauze) that may be removed the next day. There is very little limitation of the patients activities following the procedure other than typical precautions to prevent additional falls and small lifting restriction in an attempt to prevent VCFs. As with other osseous augmentation procedures, follow-up either in person or by phone is typically accomplished within 24 to 48 hours of the procedure and again at 2 weeks following the patient's discharge.

Access to fractures of the ilium is achieved by way of a posterior paramedian approach and, as with other percutaneous osseous augmentation procedures, may be done with either CT or fluoroscopic guidance. The primary anatomic structures to avoid during ilioplasty are located medial to the starting location of the procedure and include the sacral foramina (and sacral nerve roots inside),

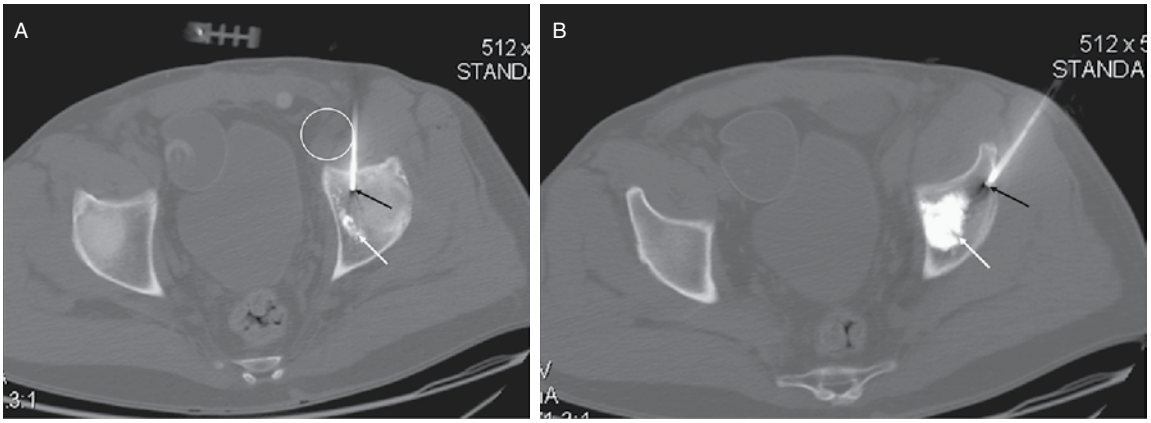

Figure 48.18 (**A**) Axial CT image shows an anterior approach to the acetabulum with the needle in place in the anterior portion of the acetabulum (black arrow). The needle path is located just lateral to the femoral neurovascular bundle (white circle). Bone cement is seen in the posterior portion of the acetabulum (white arrow). (**B**) Axial CT image shows an anterolateral approach to the acetabulum. The needle is directed from lateral to medial into the lateral portion of the acetabulum (black arrow). Bone cement is seen in the central portion of the acetabulum (white arrow).

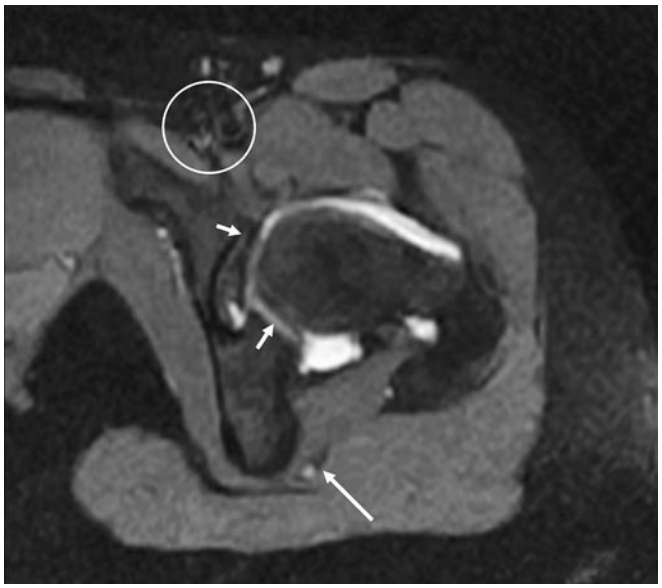

Figure 48.19 Axial T1-weighted MR image with fat saturation shows some of the anatomic structures to avoid during percutaneous access to the acetabulum including the femoral neurovascular bundle (white circle), the hip joint (short white arrows), and the sciatic nerve (long white arrow). The obturator nerve and its branches are located more inferiorly between the adductor brevis, adductor magnus, and the obturator internus.

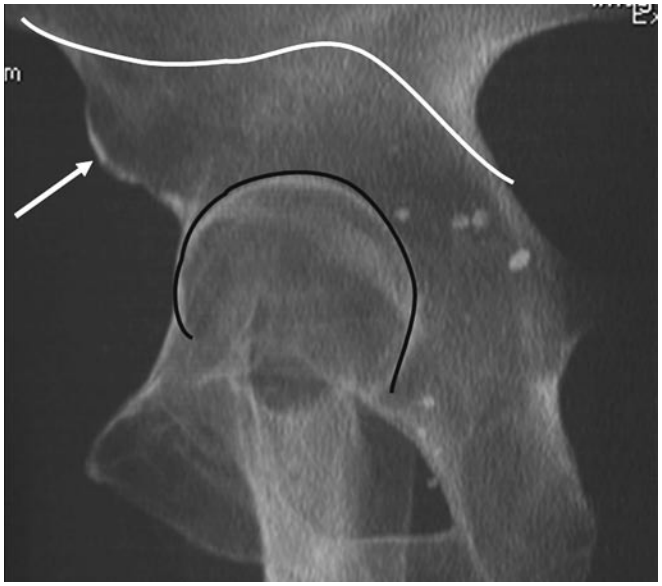

Figure 48.20 Three-dimensional volume rendered reconstruction of the right hip as seen from a sagittal orientation demonstrates the target for percutaneous acetabuloplasty. The target is the body of the ilium at the level of the anterior inferior iliac spine (white arrow) and between the inferior gluteal line (white line) and the insertion point of the articular capsule of the hip joint (black line).

the internal iliac vessels, and the lumbosacral plexus. In addition, the sciatic nerve passes through the greater sciatic foramen just inferior to the portion of the ilium that is most commonly fractured and care must be taken to avoid

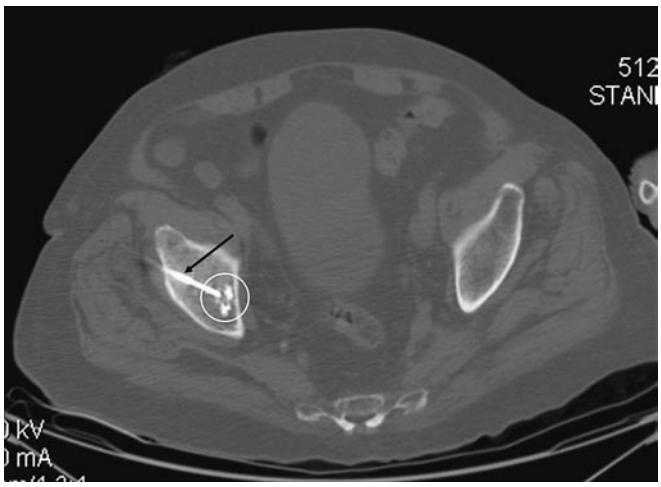

Figure 48.21 Axial CT image of the pelvis shows the needle within the right acetabulum above the right hip joint (black arrow). A bone filler has been inserted through the needle cannula and bone cement has been injected into the interstices of the ilium (white circle).

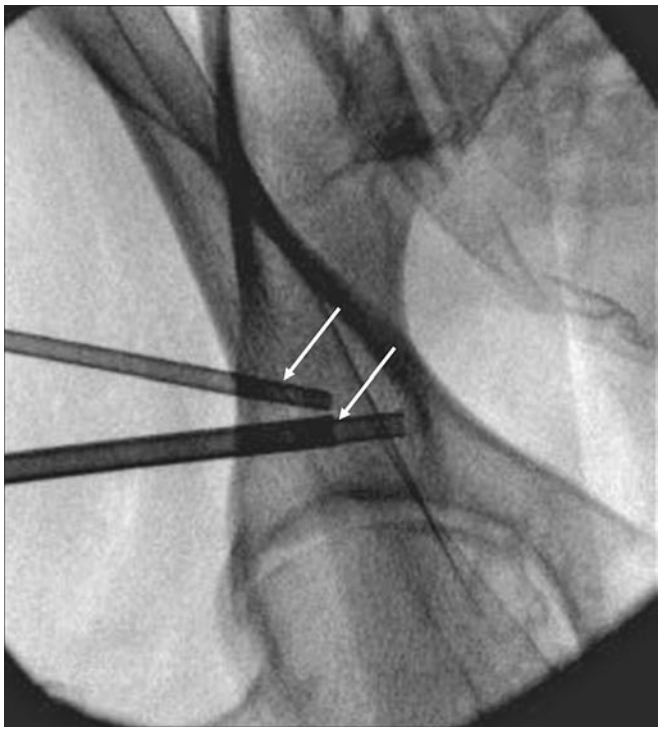

Figure 48.22 Anteroposterior fluoroscopic image shows two needles placed in the acetabulum (white arrows). These needles were placed anterolaterally into the acetabulum.

extravasation of PMMA into the region surrounding the sciatic nerve.

Once the location of the fracture is identified, the patient is placed prone on the CT table or the special procedures table and the posteromedial portion of the ilium is accessed along the center of the iliac tuberosity for the SI ligament attachment and the needle is angled parallel to the

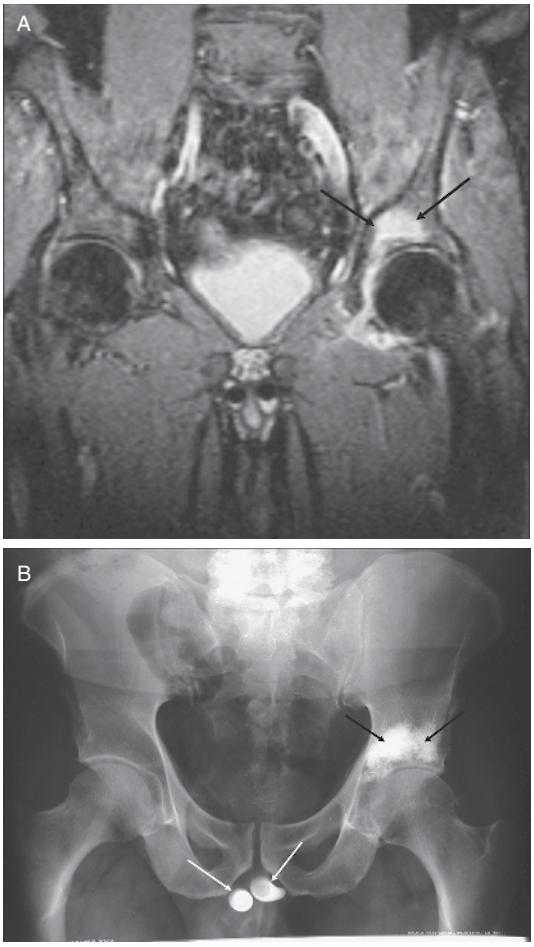

Figure 48.23 (**A**) Coronal T2-weighted MR image with fat saturation shows an area of increased signal within the left acetabulum (black arrows) indicating an acetabular insufficiency fracture. (**B**) Anteroposterior conventional radiograph of the pelvis obtained after percutaneous acetabuloplasty demonstrates bone cement within the left acetabulum (black arrows). The bone cement is located in the region of the fracture as shown in **A**. Incidentally noted is the patient's penile prosthesis (white arrows).

mediolateral angulation of the ilium (Figure 48.24). After the appropriate lateral angulation of the needle has been achieved, it is driven across the fracture site (Figure 48.25) and PMMA is injected from distal to proximal to provide stabilization proximal and distal to the fracture site as well as within the fracture itself (Figure 48.26). Injections into the ilium should be closely monitored (as with other osseous injections) to avoid unintended extravasation outside of the intended injection locations and to ensure that PMMA flows to the appropriate regions surrounding the fracture and into the fracture itself. Postprocedure care is identical to other osseous augmentation procedures and the patient is encouraged to ambulate as soon as they are able.

EFFICACY OF AUGMENTATION OF PELVIC INSUFFICIENCY FRACTURES

Initial reports of efficacy of percutaneous osseous augmentation of the pelvis have indicated that this is a viable

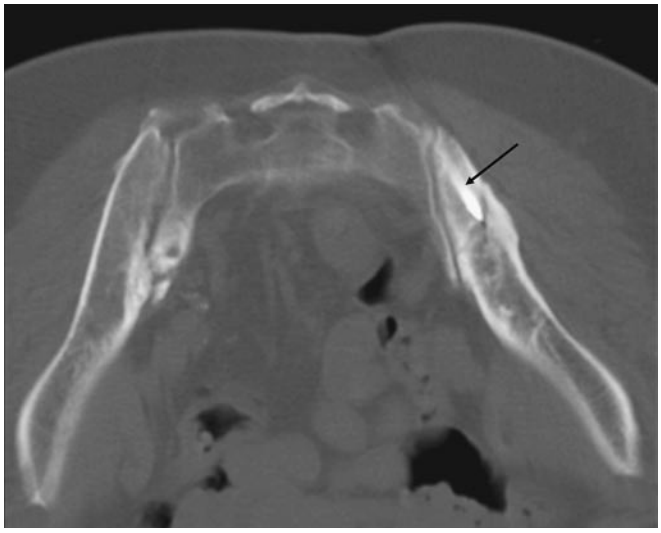

Figure 48.24 Axial CT image obtained with the patient in the prone position shows the needle in the posterior portion of the right ilium (black arrow) in a trajectory that parallels the longitudinal axis of the ilium.

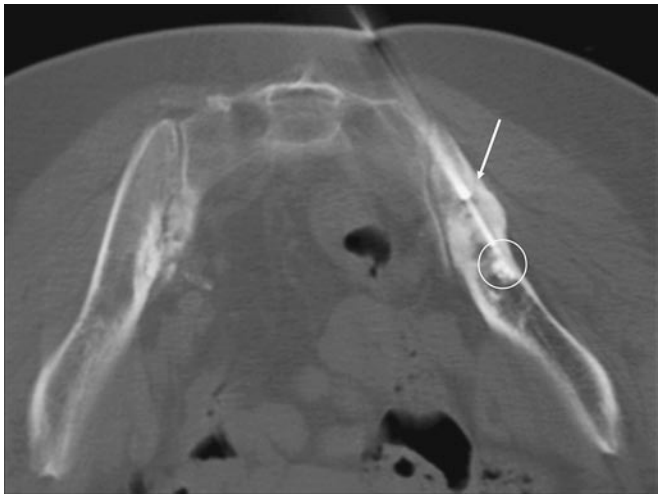

Figure 48.25 Axial CT obtained with the patient in a prone position shows a bone filler injecting bone cement into the interstices of the right ilium (white circle) distal to the ilium fracture site (white arrow).

treatment option for patients with insufficiency fractures of the pelvis and sacrum. There is a paucity of literature in this regard and much of the original experience with the effectiveness of percutaneous osteoplasty of the axial and proximal appendicular skeleton was obtained through treating neoplastic lesions in these locations [42]. The response of patients to the direct injection of bone cement into these neoplastic lesions has been encouraging for those clinicians eager to offer an additional form of palliative care to their patients and a typical patient response to this technique is substantial pain relief that is durable over the course of the typical follow-up [42,43].

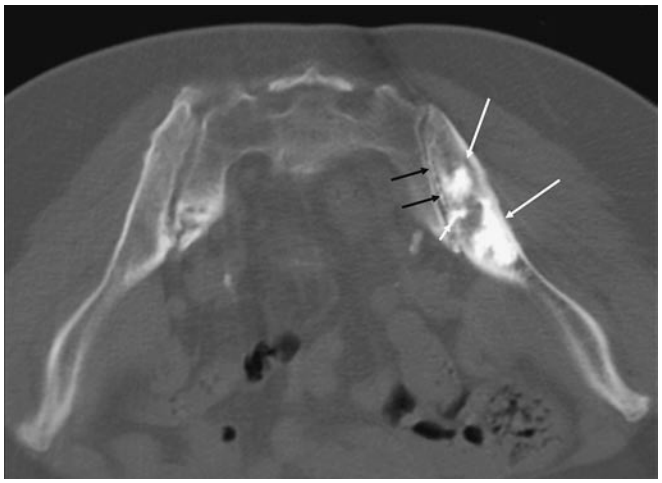

Figure 48.26 Axial CT image obtained with the patient in a prone position shows bone cement within the right ilium (long white arrows) including within the ilium fracture site as well (short white arrow). There is also some bone cement that has extravasated into the right sacroiliac joint (black arrows).

The efficacy of the treatment for osteoporotic fractures with percutaneous osteoplasty techniques are very well known in the spine and are becoming more well known and established in the sacrum, but data regarding the post-treatment outcomes in patients with insufficiency fractures of the extrasacral pelvis and proximal appendicular skeleton is far less common [24,30,44,45]. As mentioned previously, the largest reported series of pelvic fractures treated with percutaneous osteoplasty involved 14 nonsacral pelvic fractures and 26 sacral fractures, but other reports of pelvic fracture treatments are limited to case reports, small case series or retrospective analyses [24,30,44,45,46]. Despite this limited amount of information, the existing literature related to percutaneous osteoplasty of the pelvis is similar to the early vertebroplasty literature in terms of efficacy of the procedures and the low complication rates.

SUMMARY

As the osteoporotic population increases, the complications arising from this disease process will also increase including insufficiency fractures of the axial and appendicular skeleton. The initial experience with percutaneous osteoplasty of the sacrum and extrasacral pelvis has shown that this technique may be a viable treatment option in addition to conservative therapy in those patients whose bone density is insufficient to undergo osseous stabilization with metal plates and screws. The body of the literature to date has indicated that percutaneous augmentation of insufficiency fractures is safe and effective in treating patients with painful fractures and may be the treatment of choice for patients who do not improve with or cannot tolerate conservative therapy.

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49 Functional Restoration of the Osteoporotic Patient

Peter E. Pidcoe

BACKGROUND

Physical therapy is commonly prescribed in the treatment of patients suffering with osteoporosis or osteopenia [1]. Osteoporosis is a skeletal disorder that can result in compromised bone strength. This can lead to increased risk of fracture. A position paper by The North American Menopause Society stated that the primary goal of osteoporosis therapy is to prevent fractures [2]. This can be accomplished by slowing bone loss, maintaining bone strength, and minimizing factors that may contribute to fractures. The Surgeon General proposed that physical therapy should be part of the base of a pyramidal approach in the treatment of patients who suffer with osteoporosis [3,4]. This nonpharmacological component of management includes fall prevention and exercise programs and has been shown to be effective [5]. Increasing bone strength, improving balance, and increasing muscle force generation capacity are all steps toward functional restoration.

Patients who are referred to therapy may have already suffered a fall or have been identified at risk for fall. Their treatment cannot be provided via a single plan of care. Treatment frequently includes components of exercise and balance training. Titrating these activities can be difficult. There is no known dose-response relationship between exercise and bone strength [6]; therefore an understanding of physiology, anatomy, kinesiology, and biomechanics are all important components in designing an exercise progression for these patients. It is important to note that although there are guidelines in the application of exercise, there is not a single progression that fits all patients. The treatment plan-of-care must take into account patient age, gender, general fitness level, current bone density, and the area to be treated. This chapter will provide treatment guidelines to assist the practitioner in the exercise progression decision-making process.

EXERCISE

Exercise is a broad term that describes a variety of physical activities. These can be classified as aerobic or anaerobic depending on the source of the energy. Common to both is that muscles are activated and produce force. Since muscles are attached to bone, these contractions stress and strain bone tissue. Bones provide the mechanical integrity for locomotion and movement. Strains produced by muscle activity and mechanical loads (e.g., exercise) affect bone mass, density, and architecture [7]. Exercise has been shown to have a positive impact on these attributes [8]. In short, bone remodels in response to new loads. Physical therapy is often employed to elicit positive changes in bone mass and to reduce fall and fracture risk [9].

Bone mass varies among adults [10]. The development of bone mass is related to both age and activity. Some researchers support the belief that peak skeletal bone mass in the first three decades of life accounts for the bone mass variability in elderly adults [11]. Bone mass typically increases during growth, plateaus in adulthood, and declines during aging. It can be influenced by activity intensity. As an example, gymnastic loading during growth appears to yield significant enlargement of total and cortical bone geometry (+10% to 30%) with increased trabecular density (+20%) in the forearm. This results in an elevated index of skeletal strength (+20% to +50%) [12].

It is impossible to determine if adult variations are a direct result of activity level in developmental years since no longitudinal studies have been performed. The more important questions relate to the maintenance of bone mineral density (BMD) since decreases are related to adult fragility and the potential for fracture [13].

The Need for Site-Specific Exercise

While aerobic exercise is used to promote endurance and general fitness, anaerobic (or resistive) exercise is typically used to promote strength (a muscles ability to generate force during contraction). Physical activity can be considered exercise; in fact it has been shown that the osteoporotic patient can benefit from physical activity to counteract the progressive loss of bone and muscle mass associated with aging [8]. But in order to improve the effectiveness of the training, these exercises need to be site-specific.

Site-specific exercises have been shown to have a more favorable effect in improving BMD in targeted areas [6]. This was illustrated in a randomized controlled trial of 56 postmenopausal women who exercised in a controlled fashion for 1 year [14]. In the resistance trained group, BMD increases at the greater and lesser trochanter correlated with leg press exercise ($P < 0.05$) and in Ward's triangle with hip extension and adduction exercises ($P < 0.05$).

In a similar study, high-load isokinetic resistance training was found to increases site-specific BMD in women [15]. In this study, 70 women were randomly assigned to eccentric and concentric training groups and asked to perform unilateral flexion and extension exercises on their nondominant upper and lower extremities. Dual-energy X-ray absorptiometry was used to evaluate BMD changes over a 5 month period. The results showed increases in BMD in the proximal femur and forearm of the exercised limbs. These changes were not found to be correlated to the exercise mode, eccentric and concentric exercise produced the same results. Strength training has also been shown to increase BMD in the lumbar spine (2.0% gain) and femoral neck (3.8% gain) of men following 4 months of training [16].

These studies suggest that BMD improvements can be targeted to specific areas by prescribing and titrating appropriate exercise programs. In addition, improvements in strength and coordination resulting from these activities have also been shown to decrease fall risk when compared with general wellness programs that include a physical activity component [17].

Is Bone Mass Maintained After Cessation of Exercise?

Studies evaluating the cessation of exercise on bone mass show contrasting results. Many studies have demonstrated that BMD is positively impacted by physical activity [7,8,18], but that this gain is only temporary and BMD returns to pre-exercise levels once the activity is stopped [19–22]. Other studies suggest that BMD changes do persist long after the cessation of exercise, but that these effects may be different for men and women. Men appear to retain BMD increases several decades into the retirement from their sport [20,23]. Women show similar effects, but for a shorter time period [24,25]. If this is the case, then women would need to maintain some level of activity to retain the BMD benefits achieved with exercise; however, other extraskeletal benefits of exercise may also be important.

A more recent prospective study investigated the effect of a 2-year course of exercises on vertebral BMD and fracture rate in postmenopausal women [26]. The exercises focused on back extensor muscles using a progressive resistive weight lifting technique performed 5 days per week. The control and exercise group BMD was not significantly different at baseline or the 2-year follow-up, suggesting no apparent bone gain in the exercise subjects as a result of the training. At a 10-year follow-up, however, BMD had significantly decreased in both groups and the difference between the groups had become significant ($P = 0.0004$). Not only was the exercise group BMD higher than the control group, but they also reported fewer than half the number of vertebral fractures. This study suggests that improvements in muscle strength may be more important at reducing the risk for vertebral fractures than BMD alone [26,27].

Exercise programs designed to improve bone mass, muscle force production, posture, and balance have to consider many factors, safety being paramount. No single plan will fit all individuals. Below are some guidelines that may help in the development and progression of an exercise program.

Age and Gender Factors

Certain sports and exercises promote skeletal development in children and adolescents; augmenting bone strength in adulthood [28,29]. These sports are typically high impact in nature. These high impact components may not be appropriate for the older adult, but there is evidence suggesting that women at middle and older ages need to train at higher intensity levels than men to improve bone mass [30].

Types and Order of Exercises

Bone adaptation is limited to loaded regions. As a result, exercises must be chosen to act on specific sites to be affective [14]. As an example, exercises that load the hip and greater trochanter, intertrochanteric and femoral neck regions include hip flexion, extension, and abduction activities. These can be accomplished with open kinetic chain exercises like leg press and extension machines, or by using closed chain activities like front step-ups, side step-ups, jumping, and running [31].

Impact exercises should be progressively increased within the capabilities of the subject [32]. The age of the subject, general fitness level, and fall risk should be part of the equation when selecting exercises. These exercises can include jumping, running, and stair climbing, although the osteogenic potential of these exercises is reduced in postmenopausal women [13]. Activities like swimming (aquatic therapy) may not be appropriate for this population since it is not site-specific and is non-weight-bearing [33]. However, the combination of weight bearing and non-weight-bearing activity has been shown to be effective at reducing physiologic bone loss in postmenopausal women [34].

Intensity and Frequency of Training

There is no consistent threshold reported in the literature to enhance bone mass. Most programs use intensities of 70% to 90% of a 1 repetition maximum as a guideline [13]. Training frequency is usually 2 to 3 days per week, but can increase to 5 to 6 days per week dependent on the subjects training experience and tolerance. The higher intensity training does result in substantial and continued increases in strength in postmenopausal women, with the greatest gains seen in the first 3 months [35]. It is thought that this high-intensity physical activity must be continued to maintain the improvement [22].

Training Velocity

Explosive muscle contractions produce greater osteogenic stimulus [30]. As a result, training progression should move from medium speed to high speed as soon as the subject can perform the activity safely and with good form.

BIOMECHANICS

Exercise programs also need to consider the underlying biomechanics of the activities. The forces produced in all activities are governed by physical principles. In this context, these forces include gravity and muscle contraction. Gravitational forces are considered to be external and muscle contraction forces are internal. External forces are easily measured with instrumentation like force plates, scales, and load cells. Internal forces are more difficult to measure in vivo and are often estimated using inverse dynamic techniques that require measurements of body segment positions, measurements of external loads, and estimates of segment mass distributions. These two combine to produce forces that impact bone by mechanically stressing it.

Impact Forces

External forces are often characterized in units of body weight (BW). An example of peak external forces acting between a subject and the floor include standing (1 × BW), level surface walking (1.3 × BW), running (3 × BW), walking downstairs (6 × BW), and falling (>10 × BW). In all but the standing example, these are considered impact loads. Impact loads are attenuated by soft tissue, but are ultimately passed to bone. If the bone is unable to absorb the transmitted energy, a fracture occurs. The decline in bone mass and the associated changes in structural integrity increase the risk of fractures [13].

Reaction Forces

External and internal forces occur during exercise and create reaction forces. During running and walking, impact loads occur during the initial phases foot contact and produce loads that are transmitted from soft tissue to the bones and joints. Reaction forces are those associated with muscle reactions. These act to counter the external torques and balance the subject during an activity. These forces also stress the bone, but typically in a more controlled manner than the aforementioned impact forces.

Exercise or activity progression involves assessing these loads and advancing the individual in a controlled and calculated way. Consider a person jumping with both feet in a forward direction on a level surface. More force is typically generated during the landing phase than the take-off phase. The forces seen at the foot-floor interface can be modified by using combinations of one and two foot take-offs and landings. A simple progression from small loads to larger loads might include the following: (1) two foot take-off/two foot landing, (2) one foot take off/two foot landing, (3) two foot take-off/one foot landing, and finally (4) one foot take-off/one foot landing. Jumping up onto a higher surface would act to decrease landing forces, while jumping down would increase these forces.

Surface Choice

Surface stiffness also impacts the forces transmitted to the individual. These are dictated by the impulse-momentum relationship. This relationship states that a body with momentum will produce less force, when brought to a stop it is allowed to decelerate over a longer period of time. Landing on a concrete floor produces a quick deceleration. Landing on a padded or carpeted surface increases the time for deceleration and therefore decreases the landing or impact force.

Using the same impulse-momentum relationship, speed (or velocity) can also be used to control loads. Objects moving at a slower velocity have less energy and therefore produce less force when they are decelerated. This is why jumping up onto a higher landing surface produces a smaller landing force. There is less time for gravity to accelerate the individual and therefore less velocity at impact.

BALANCE AND FALL PREVENTION

Fracture risk is obviously going to be reduced with fall risk reduction since falls are often associated with fracture [36,37]. Back extensor strengthening may also help to reduce fracture potential [38] even in patients who have had vertebroplasty [27]. The mechanism has been theorized to relate to posture and the effect poor posture has on vertebral compression forces. The loss of lordotic curves in the lumbar spine and an increase in thoracic kyphosis result in increased vertebral compressive loads [39]. Increased extensor muscle support may decrease the compressive forces on the spine [38]. This may be especially important in patients who have had vertebroplasty since they may be prone to accelerated failure of adjacent bodies [40].

Improved posture may also improve balance [41,42], so the result of better posture is twofold: reduced vertebral loading and reduced fall risk. Fall risk increases in older populations [36] with recurrent falls more likely in women [37]. Balance training is often employed to reduce this risk and is often part of a therapeutic exercise program [8,43,44]. Physical activity has been shown to reduce postural sway and unsteadiness in postmenopausal and osteopenic women [45,46]. Decreased postural sway equates to reduced fall risk. Tai Chi has been shown to improve balance, increase lower extremity strength, and reduce fall risks [7,47]. Balance, posture, strength, and bone density are all interrelated. Improving balance and posture typically reduces fall risk [7]. Activities that improve balance also improve strength through exercise [7,47,48]. Exercise promotes increases in bone density [8]. In short, staying active has an overall positive impact on well being. The focus is on prevention since fall-related healthcare costs are typically much higher than the cost of a prevention program [49].

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