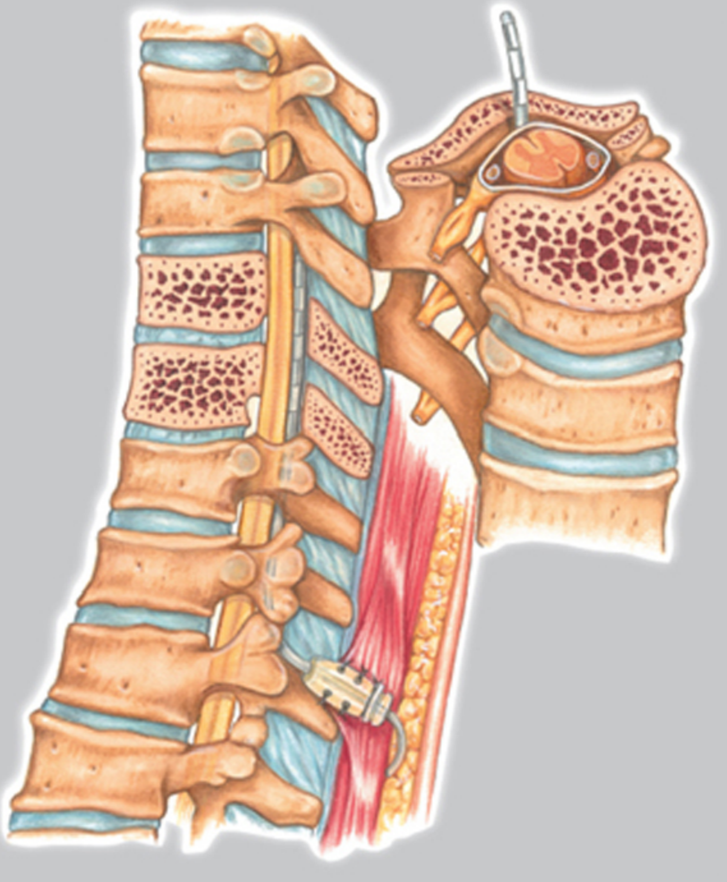
Surgical Management of Pain

Kim J. Burchiel



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Contents

Part I Basic Considerations

Part II Fundamentals of Pain Medicine

Part III Specific Pain Syndromes

Part IV Surgical Procedures

Part V At the Forefront of Pain Surgery

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Foreword

The most often cited article in modern literature on pain and pain research is that published in Science 1965 by Melzack and Wall introducing the gate-control theory. In that paper there is a drawing of an infantlike figurine displaying the entire repertoire of surgical interventions for pain, illustrating that pain surgery was then tantamount to ablative procedures. However, Melzack and Wall also indicated the possibility of modulating by external means the endogenous pain controlling system described in their paper. The presentation of the gate-control theory not only denoted a turning in pain research, but it soon led to the first experimental trials to apply these ideas for therapeutic purposes in humans. Wall and Sweet courageously experimented on themselves, stimulating the infraorbital nerve via percutaneous needles and observing hypalgesia in the territory of the nerve. The observation led to the evolution of electric spinal cord stimulation by Shealy and the subsequent introduction of TENS. The basic concept of gating mechanisms (i.e., the importance for pain perception of a delicate interplay or balance between coarse and thin fibre afferent systems) had in fact been suggested also in the classical pain literature by Head and Holmes and by Zotterman. Although the theory as defined and presented in the famous Science paper attracted much interest, it was also much criticized. However, its role and impact on modern pain research and on the understanding of generation and modulation of pain can hardly be overrated. Of paramount importance was the realization that pain perception cannot be understood as being the result merely of signals transmitted in separate channels with interposed amplifying relays in a straight-through, one-to-one fashion, isolated from and uninfluenced by the rest of the CNS.

This new insight also represented a turning point for pain surgery, and in the 1970s many previous extensively-practiced destructive procedures such as posterior rhizotomy soon became obsolete. The new knowledge also offered likely explanations for the seemingly mysterious recurrence of pain after extensive lesioning of the "pain system."

The introduction of electric stimulation of the CNS denoted an entirely new phase of treatment in pain surgery since it mainly replaced destructive interventions with reversible modes of modulating central pain processing. It seemed that this new approach to pain management had the unique feature of providing surprisingly long lasting relief. I well remember that Sweet once said that in his experience-which was indeed extraordinary-all forms of pain treatment inevitably failed. There are, however, in the literature many reports of patients subjected to spinal cord or intracerebral stimulation who have enjoyed a durable relief for decades. In fact, I was recently contacted by a patient because of malfunctioning of a spinal cord stimulation system implanted in 1973 and continuously in use since that time.

To date, ablative pain surgery is justified only for a limited number of patients with specific indications. For example, no one would consider cordotomy for non-cancer related pain. Nevertheless, this operation, introduced in 1911 by Martin and Spiller, is perhaps the most rational and effective type of pain surgery. It still has an important place in the armamentarium for managing some patients with severe pain due to malignancy and resistant to advanced pharmacotherapy. Otherwise, the development of modern pain surgery is characterized by efforts to minimize invasiveness and postoperative neurological deficits. The treatment of trigeminal neuralgia is a good example of the evolution of pain surgery as it has evolved from neurotomy and rhizotomy to microvascular decompression.

The less invasive procedures such as intracisternal phenol injection and ganglion/rootlet electrocoagulation have been replaced by selective thermorhizotomy, graded root compression and glycerol injection. Radiosurgery represents the most recent and non-invasive development.

A notable, and non-controversial, exception to the tendency to abandon destructive pain surgery is dorsal-root-entry-zone (DREZ) operations which still appear to be the only efficacious way of treating root avulsion pain. In the 60s and 70s, medial thalamotomy was extensively practiced, mostly for pain in malignancy disease. The results were mediocre and with the dissemination of a rational and more liberal usage of opioids the practice of thalamotomy ceased. However, in later years, some proponents of this type of ablative surgery have claimed its usefulness even for non-cancer related pain. It might be that favorable outcomes have been achieved because the original target area has been modified. Are we perhaps witnessing a revival of thalamotomy?

In a way, it is surprising that new neuroanatomical data still appear. The "discovery" of a spinal ascending pathway, located in the center of the dorsal cord and subserving visceral nociceptive pain, has generated a technique using a common hypodermic needle.

A major advance with pivotal importance for all pain treatment modalities is the differentiation of various forms of pain. It should be remembered that not until the last decade has it been recognized that pain can no longer be conceptualized as an entity (e.g., "cancer pain") and that the dichotomy of nociceptive and neurogenic pain is not a sufficient base for an adequate pain diagnosis, which should instead identify the underlying pathophysiological mechanisms. This novel approach to pain analysis has evolved as a prerequisite for adequate therapy. Postherpetic neuralgia, which may present with a variety of symptoms, can serve as an example of when a mechanism-oriented pain diagnosis is virtually mandatory for therapeutic efficacy. Such a more refined and rational way of diagnosing pain is actually of paramount importance for defining selection criteria for all forms of pain surgery.

It is now more than six decades since the first textbook on pain surgery appeared: René Leriche, *La chirugie de la douleut* (1937). For many years, the "bible" and standard book in the field was *Pain and the Neurosurgeon,* by White and Sweet, published in 1955, with a subsequent edition appearing in 1969. This thorough treatise covered virtually all aspects of pain and is characterized by the many detailed case reports described in the meticulous way that was typical for Sweet. In 1989, Gybels and Sweet published an extensive textbook, *Nerosurgical Treatment of Persistant Pain,* which, apart from practical guidelines and evaluations, contains comprehensive accounts on the physiological background of each procedure in the light of modern pain research. A more recent publication, partly based on two consensus conferences, is *Neurosurgical Management of Pain,* edited by North and Levy. It should also be noted that many chapters in Gildenberg and Tasker's *Textbook of Stereotactic and Functional Neurosurgery* are devoted to pain surgery as well.

There is reason to remind readers of the fact that for many decades, neurosurgeons were pioneers, playing leading roles in the advancement of pain treatment. Pain management was then an indispensable part of neurosurgical training, but it appears that with the evolution and diversification of neurosurgery, the number of neurosurgeons presently choosing pain surgery as their preferred subspecialty is decreasing. As a consequence, the art of performing, for example, percutaneous cordotomy or analyzing facial pain other than trigeminal neuralgia is fading. This is regrettable, because for many patients a surgical approach to the management of their chronic pain remains the only option. It is indeed our obligation to spread this message to the medical community in general and to our anaesthesiological colleagues in particular, who presently care for the great majority of pain patients.

There is no doubt that a great need exists for an updated text on pain surgery, one that covers the entire field and could serve as a source of knowledge and inspiration for both clinicians and basic scientists. A special merit of the present volume is that it includes a section on the medical aspects of pain, with guidelines for analysis, assessment, non-surgical treatments, pain clinic organization and so forth, solid knowledge of which is mandatory for a neurosurgeon who must function in a multidisciplinary context. The list of contributors is truly international and virtually represents a "who's who" in pain medicine, management, and surgery. A characteristic feature is that each chapter, written by a recognized and experienced specialist, is followed by a commentary by someone representing different perspectives and opinions. In this way, a balanced presentation of issues that may be controversial is attained and the usefulness of each surgical procedure is evaluated from different aspects. The task of organizing and editing such a publication is huge and the accomplishments of Dr. Burchiel are indeed admirable. I know that most of the authors are clinically and scientifically extremely busy and it must have required repeated communications from the editor to finally gather all contributions. Now, looking at the final product, I realize that this volume is a worthy follower of the classics in the field of pain in general and of pain surgery in particular. It is my conviction that this book will be most useful for everyone interested in advanced and interventional pain therapy, and it is my hope that it will serve as a source of inspiration for young neurosurgeons to embark on the fascinating field of pain surgery.

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Dedication

Generations of neurosurgeons will remember the erudite, but crusty, Yankee who dominated the field of pain surgery for so many decades. My personal recollection is of a man who always seemed to be sitting in the front row of every session, taking extensive notes, and asking the tough, probing questions. On January 22, 2001, the field of neurosurgery was diminished by his passing. He remains a role model and inspiration to those of us who have chosen this area for our personal professional path. It is for this reason that this book is dedicated to Dr. William H. Sweet.

Preface

The origin of this textbook stems, in no small measure, from a conversation between a senior neurosurgeon of some notoriety and a chief resident in neurosurgery, deep in the hunt for a job in a prestigious academic medical center. Early in the interview it became clear to the seasoned faculty member that this tyro wanted to pursue the neurosurgical management of pain as his scholarly niche in academics. Skeptical, the question was put to the soon-to-be academician: Specifically, what would he propose as possible surgical procedures for pain? After some verbal foot shuffling, the young neurosurgeon opined that well, of course, there were many highlights in the vast sweep of operative therapies for pain, at least two or three that he could think of right off. Lives are changed by small moments like this.

In the intervening years, I have mulled that question. Is there a definable specialty of neurosurgery devoted to the treatment of pain? The answer is unequivocally, yes. It is part of the larger discipline of pain medicine that took root and flourished during the past four decades since its development by John Bonica and his associates at the University of Washington in the 1960's. The pantheon of pain research and treatment, is, in fact, teeming with neurosurgical heroes. In the vanguard were men like Fred Kerr and William Sweet. They were followed by the likes of Ron Tasker, Bjorn Myerson, Bob King, Hu Rosomoff, Blaine Nashold, John Loeser, Don Richardson, Yves Lazorthes, Peter Jannetta, Marc Sindou, Takashi Tsubokawa, Phil Gildenberg, Don Long, Yucel Kanpolat, and many others. These men helped to define a field that continues to grow and evolve, as does any healthy discipline of medicine. This text is a testament to the status of surgical pain management at the beginning of the twenty-first century.

Early in the planning for this book, I decided to employ a format similar to that used by a number of currently successful medical journals. These journals have developed a format of scientific article followed by expert commentary. Most notably in my field *Neurosurgery* exploits this style to good advantage. I know that I, and I suspect many of my colleagues, read these comments (at times to the exclusion of the article!) as a way of gaining perspective on the content and significance of the contribution. In this book I have emulated this motif. I also wanted to include new perspectives on topics related to pain treatment. Therefore, in as many cases as possible, I have asked representatives of the coming generation of pain surgeons to prepare the more traditional didactic textual material. This, I hoped would help avoid the syndrome of "cloned chapters," written by a small cadre of senior authors, so common in many of our major medical texts. In the present book, the graybeards get their revenge by the application of incisive and sage comments at the conclusion of each chapter.

It has been the utmost personal honor to work with so many luminaries from the fields of neuroscience, neurosurgery, neurology, anesthesiology, rehabilitation medicine, internal medicine, plastic surgery, psychiatry, psychology, and dentistry in the preparation of this book. My intent was to produce a veritable "who's who" of pain surgery. The final author list exceeds even my original admittedly expansive concept.

I have included topics in this book that might seem somewhat ancillary to the knowledge base of the clinician interested in surgical pain treatment. Beyond the obligatory reviews of the anatomy, physiology, and pharmacology of pain and nociception, and a recitation of specific pain diagnoses, some topics may appear to be off the mark of what might be expected to be, for the most part, a procedural text. But, in fact, discussions on the assessment of pain patients, the rehabilitative treatment of patients with chronic pain, management of opiates and other analgesics, myofacial treatments, and the ethics of pain control in the dying patient, are as central to the practice of surgical pain management as knowing how to place a spinal cord stimulator or perform a DREZ operation. In my mind, being an effective pain surgeon requires a broad knowledge of the field of pain medicine, with all of its ramifications.

As with any successful campaign, the production of a textbook was dependent on organization to successfully prosecute the plan. For this I am deeply indebted to my publications assistant, Beth Fee for her tireless patience, dogged persistence, and indefatigable good humor. Her contribution to this project is particularly poignant given that it occurred during a time that all of us who know her so well were saddened by the untimely death of her husband, Larry Fee. This book, in large measure, bears witness of her love for him, and will be a lasting tribute to his memory.

I also thank Joanie Mastrandrea and Todd Ellingston for keeping the lid on administrative and clinical concerns of a department and a neurosurgical practice, respectively, in the throes of finalizing this book. This work started with the encouragement of Ave McCraken, and continued through the capable management of Kathy Lyons and production skills of Becky Dille. I am beholden to all those at Thieme who helped make this book a reality. Most importantly, words cannot express my love and appreciation for my wife, Debra, and to our family for their tolerance of my day job.

There are numerous excellent textbooks devoted to the problem of pain diagnosis and treatment. Most notable among these are *Bonica's Management of Pain (3rd* edition), edited by John Loeser, and the *Textbook of Pain*, edited by Pat Wall and Ronald Melzack. More specific to the topic of the surgical treatment of pain is *Neurosurgical Management of Pain*, edited by Richard North and Robert Levy. Comprehensive textbooks on neurosurgery such as *Neurosurgery (2nd* edition), edited by Robert Wilkins and Setti Rengachary, and *Youmans Neurological Surgery*, edited by Richard Winn, also have good overview sections on the topic of surgery for pain. The *Textbook of Stereotactic and Functional Neurosurgery*, edited by Phil Gildenberg and Ronald Tasker, contains an extensive section on pain and its surgical management. These books should all be part of the library of any serious student of the surgical treatment of pain.

With all due admiration for the texts noted, the book that I have edited still risks disappearance into the penumbra that continues to radiate from the monumental works by White and Sweet: *Pain, Its Mechanisms and Neurosurgical Control,* published in 1955, and *Pain and the Neurosurgeon*, published in 1969. The later addition of *Neurosurgical Treatment of Persistent Pain*, by Gybels and Sweet, in 1989, simply confirmed Dr. Sweet's preeminence as teacher and mentor to a generation of clinicians interested in the surgical treatment of pain. These books are the standard against which future textbooks on pain surgery will likely be compared.

Basic Considerations

Section I

The scientific basis of pain treatment has advanced in parallel with the explosive growth of neuroscience in the past 45 years since the publication of *Pain, Its Mechanisms and Neurosurgical Control.* Details continue to be added to our knowledge of nociceptive and antinociceptive systems. Perhaps the most significant advancements have occurred in our understanding of neuropathic pains, e.g., pains due to nervous system injury. Important progress has also has been made in the manipulation of antinociceptive systems by so-called "neuromodulation." In this area, in particular, experimental studies on intrathecal opiates and stimulation of the central and peripheral nervous system have led to substantial improvement in what has come to be known as "interventional pain management." These basic considerations are fundamental to an understanding of the surgical techniques discussed later in this book.

You might be somewhat taken aback by what appears to be a fundamental disagreement between the author and commentator of Chapter 2. This apparent conflict is emblematic of a genuine, and sometimes virulent, controversy on the nature and basis of chronic neuropathic pains that run under banners such as "Sympathetically-maintained Pains," "Reflex Sympathetic Dystrophy," and "Complex Regional Pain Syndrome" (types I and II). My charge to the reader is to take in both sides of the controversy, suspending judgement on the ultimate veracity of either argument. I am reasonably confident that in the fullness of time, continued study and a reliance on evidence-based medicine will unravel what appears at present to be the legendary Gordian knot.

Chapter 1

Physiologic Anatomy of Nociception

Thomas K. Baumann

Nociceptive neurons are responsible for the sensory–discriminative aspects of pain. This chapter describes the anatomic connections and physiologic properties of peripheral and central neurons that contribute to nociception, beginning at the level of nociceptive primary afferent neurons and proceeding through the ascending pathways that lead to the cortex of the brain.

PHYSIOLOGIC PROPERTIES AND PERIPHERAL PROJECTIONS OF PRIMARY AFFERENT NOCICEPTIVE NEURONS

First-order (primary afferent) nociceptive neurons are sensory neurons that are specialized to detect the presence and signal the location, quality, and intensity of tissue-damaging stimuli.[1,2] All tissues of the body (with the exception of the neuraxis) are innervated by these nociceptors. Most tissues are innervated by both nociceptive and nonnociceptive (low-threshold mechanoreceptor and thermoreceptor) neurons, but some tissues (the cornea, dental pulp, internal surface of the tympanic membrane, as well as the dura, venous, and bony sinuses within the cranium) are innervated mainly, if not exclusively, by nociceptive neurons.

Dorsal Root Ganglia and Trigeminal Ganglia

The cell bodies of primary afferent nociceptive neurons are located in dorsal root ganglia and trigeminal ganglia. The posterior half of the head and the rest of the body are innervated by cervical, thoracic, lumbar, and sacral dorsal root ganglion neurons. The innervation follows the well-known pattern of spinal (*radicular*) dermatomes (Fig. 1–1). Neurons that innervate the anterior aspect of the head (Fig. 1–2) have cell bodies in trigeminal ganglia, except for slowly adapting mechanoreceptors that innervate the gums and masticatory muscles (the cell bodies of which are in the trigeminal mesencephalic nucleus). Within dorsal root ganglia and trigeminal ganglia, the cell bodies and axons of nociceptive and nonnociceptive neurons are intermixed, arranged in a loosely somatotopic fashion.

Nociceptive neurons make up approximately half the population of the neurons in the dorsal root and trigeminal ganglia (the rest of the neurons are devoted to innocuous tactile, thermal, and kinesthetic sensations). Axons of nociceptive neurons that innervate the skin, muscle, or joints project through peripheral nerves accompanied by axons of nonnociceptive somatosensory neurons (Fig. 1–3); axons of visceral nociceptive neurons project through visceral nerves along with the axons of sympathetic and parasympathetic neurons. Some primary afferent nociceptive neurons have large-diameter, thickly myelinated axons that conduct action potentials rapidly (i.e., in the Aβ-fiber range),[3] but the vast majority of nociceptive sensory endings are supplied by small-diameter axons, which are either thinly myelinated or unmyelinated. The former conduct action potentials at velocities between 2 and 40 m/s and traditionally are designated Aδ (when referring to fibers that innervate the skin) and group III (in the case of fibers innervating skeletal muscle and joints). Transmission along unmyelinated (C- or group IV) fibers is quite slow (≤1.5 m/s), meaning that brief, simultaneous activation of nociceptive Aδ- and C-fibers in distal extremities can give rise to “first” and “second” pain because the conduction distance to the spinal cord is sufficiently long to allow temporal separation between the Aδ- and C-fiber action potential volleys.

Nociceptive Neuron Response to Different Sensory Submodalities of Noxious Stimulation

Neurophysiologists recognize several physiologic types of primary afferent nociceptive neurons and classify them according to the conduction velocity of the axon and the types of noxious stimulation that excite the neuron. Tissue-damaging or noxious stimuli may be mechanical, thermal, or chemical. Nociceptive neurons that respond to more than one type (or submodality) of noxious stimulation are often referred to as *polymodal.* Neurons that respond only to intense mechanical stimuli are called *high-threshold mechanonociceptors* (Fig. 1–4), and nociceptive neurons that respond to both noxious heat and mechanical stimuli are referred to as *mechanoheat nociceptors,* many of which also respond to noxious cold stimuli (Fig. 1–5).[4]

Among cutaneous mechanoheat nociceptors with Aδ-fibers, neurophysiological experiments revealed two sub-

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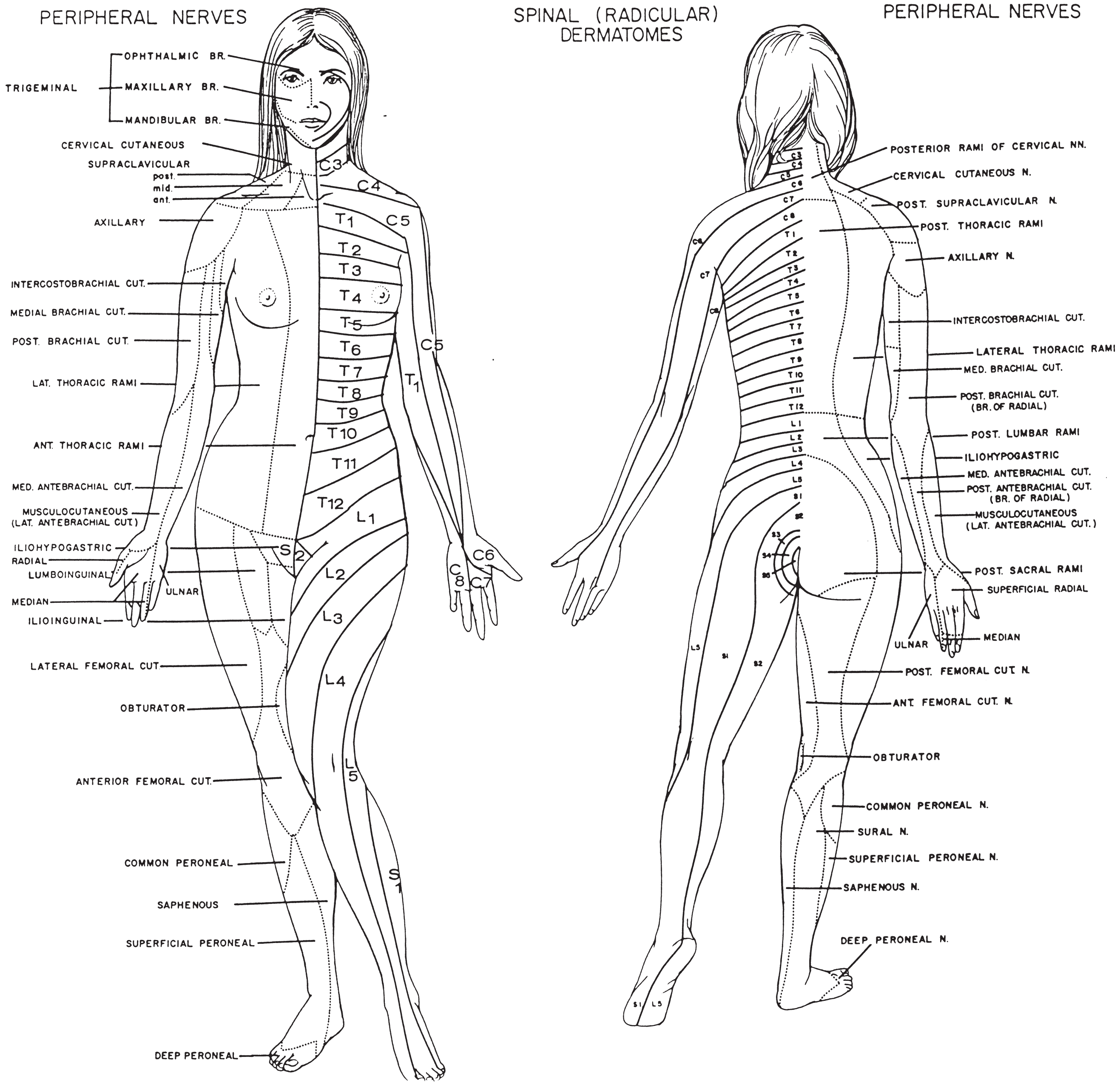

Figure 1–1 Comparison of radicular (dermatome or segmental) and peripheral nerve innervation. (From Marcus EL. Clinical considerations of the spinal cord. In: Curtis BA, Jacobson S, Marcus EM, eds. *An Introduction to the Neurosciences.* Philadelphia: WB Saunders; 1972:150–206)

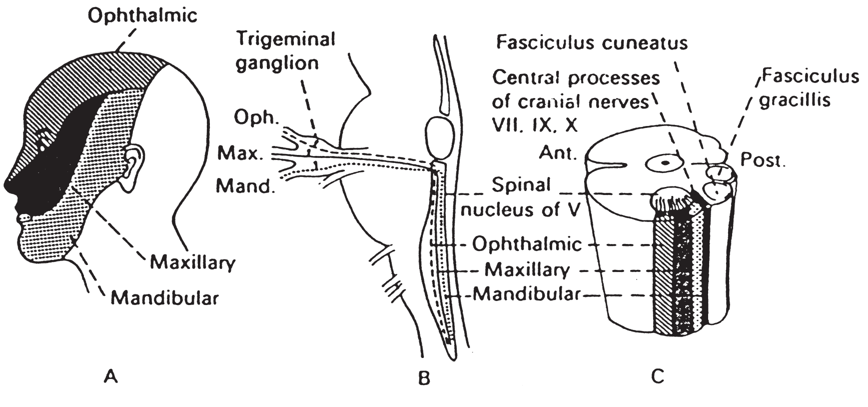

Figure 1–2 The cutaneous distribution of the three divisions of the trigeminal nerve and their termination in the trigeminal brainstem nuclear complex. (From Bonica JJ. Anatomic and physiologic basis of nociception and pain. In: Bonica JJ, ed. *The Management of Pain.* 2nd ed, vol. 1. Philadelphia: Lea and Febiger; 1990:28–94, with permission.)

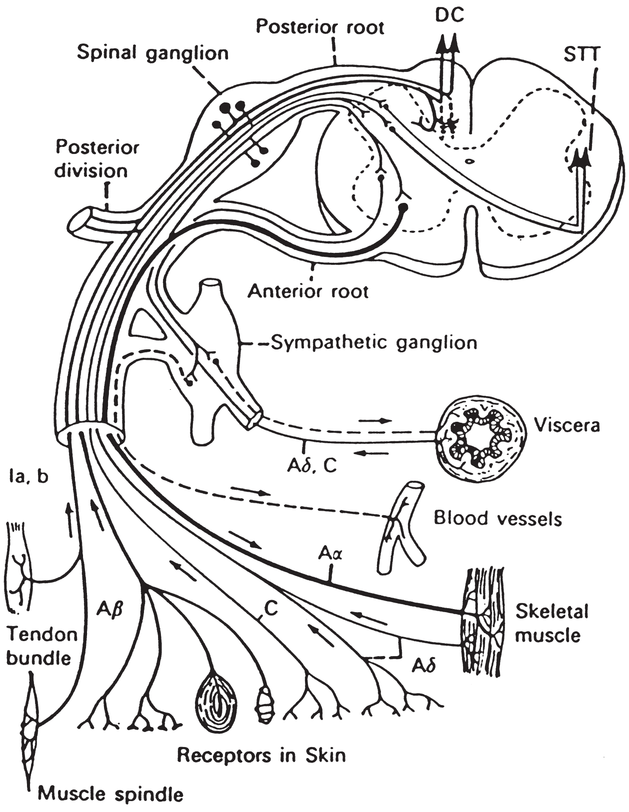

Figure 1–3 A schema of a spinal nerve and the different types of fibers it contains. DC, dorsal column; STT, spinothalamic tract. (From Bonica JJ. Anatomic and physiologic basis of nociception and pain. In: Bonica JJ, ed. *The Management of Pain.* 2nd ed, vol. 1. Philadelphia: Lea and Febiger; 1990:28–94, with permission.)

groups (called type I and type II A-fiber mechanoheat nociceptors). Type I A-fiber mechanoheat nociceptors tend to be readily excited by noxious mechanical stimuli (such as pinch) but have a relatively high heat threshold (typically >51°C), whereas type II A-fiber mechanoheat nociceptors tend to be less sensitive to mechanical stimuli but have a lower heat threshold (45°C).[5] The heat threshold of C-fiber mechanoheat nociceptors and C-fiber heat nociceptors is even lower and corresponds to the threshold for the perception of heat-evoked pain in humans (42°C).

SPECIAL CONSIDERATION

Human judgments of the intensity of pain evoked by prolonged, intense heat stimuli reflect combined activity (i.e., frequency of action-potential discharge) in both C-fiber mechanoheat nociceptors and A-fiber mechanoheat nociceptors (Fig. 1–6).[5]

In addition to physical stimuli, many primary nociceptive neurons are excited by one or more endogenous chemicals that are released into the extracellular space by cell injury [e.g., adenosine triphosphate (ATP) and potassium] or tissue inflammation (glutamate, bradykinin, serotonin, histamine, prostaglandin E_2 and I_2, protons, nitric oxide, and other radicals).[6] Many nociceptors also respond to exogenous chemicals of either plant origin (e.g., capsaicin, piperine, resiniferatoxin, or allylisothiocyanate in hot spices)[6–9] or insect origin (e.g., melittin in bee venom).[10] Figure 1–7 shows a comparison of the response of C-fiber and A-fiber nociceptors to capsaicin and heat.

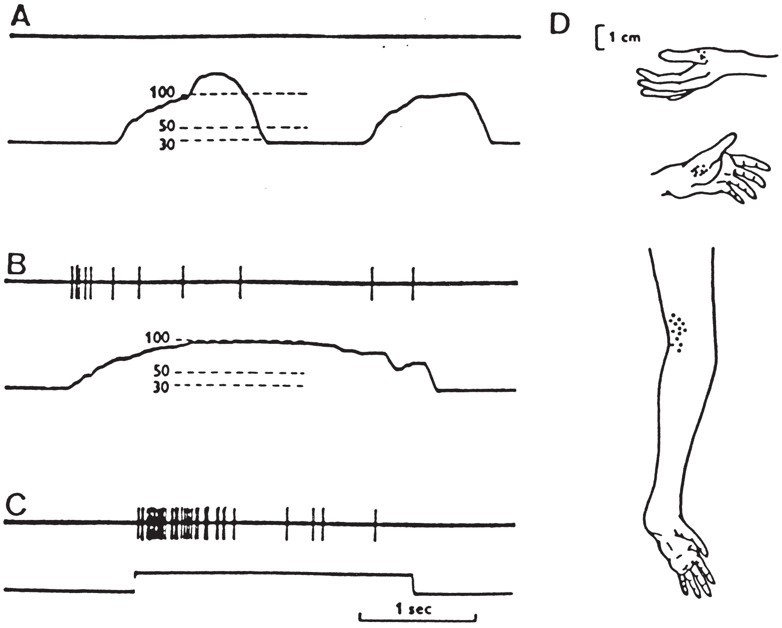

Figure 1–4 Responses of an Aδ mechanical nociceptor in the glabrous skin of a monkey to pressure or to damaging stimuli. **A:** There was no response to pressure by a blunt probe with a 2.2-mm tip diameter (numbers indicate force applied in grams). **B:** There was action potential discharge when similar forces were applied with a needle tip. **C:** The stimulus was provided by pinching with serrated forceps. **D:** Receptive fields of three different Aδ mechanical nociceptors in monkey skin. The receptive fields consisted of sets of punctate spots separated by insensitive zones. (From Perl ER. Myelinated afferent fibres innervating the primate skin and their response to noxious stimuli. *J Physiol (Lond).* 1968;197: 593–615, with permission.)

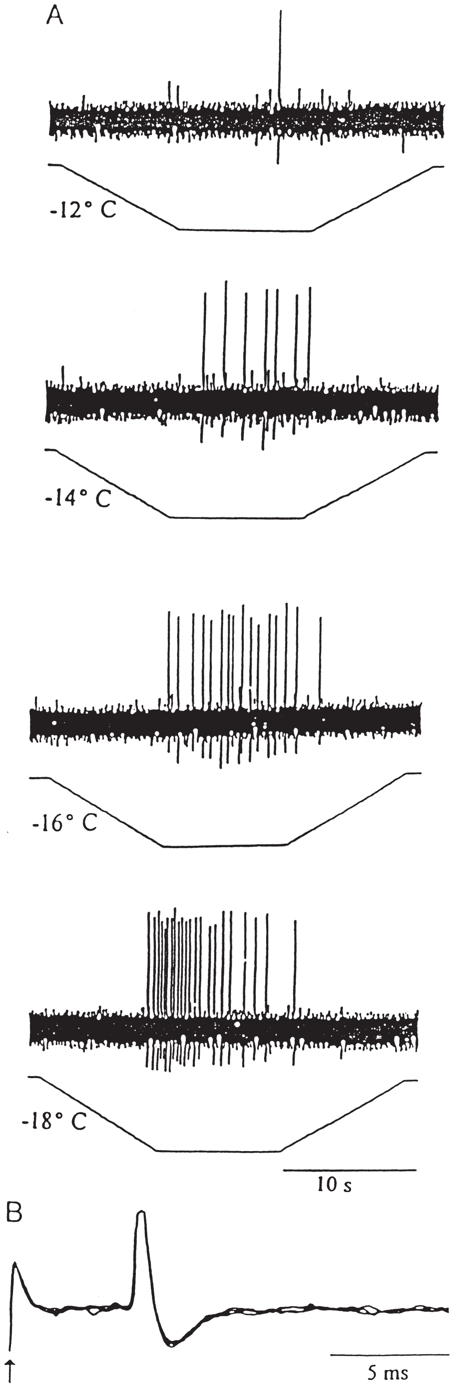

Figure 1–5 **A:** Response of a single Aδ nociceptor evoked by cold stimuli of −12 to −18°C applied to the skin of a rat. Numbers refer to peak stimulus temperatures at the surface of the skin (maintained for a duration of 10 seconds). Response threshold for cold was −12°C. The total evoked number of impulses, the mean discharge rate, and the peak discharge rate increased with stimulus intensity. **B:** Constant conduction latency for this fiber (5 overlapping traces). Arrow: electric stimulus artifact. (From Simone DA, Kajander KC. Responses of cutaneous A-fiber nociceptors to noxious cold. *J Neurophysiol.* 1997;77:2049–2060, with permission.)

Sensitization of Nociceptors by Inflammatory Mediators

In some nociceptors, inflammatory chemicals cause frank excitation (i.e., initiate action-potential discharge), whereas in many nociceptors the effect of inflammatory mediators is more subtle and consists of sensitization (manifested as lowered threshold for action-potential discharge or an increased number of action potentials discharged in response to other stimuli).[6,11] The sensitizing effect of inflammatory mediators is particularly striking in the case of the so-called mechanically insensitive, silent or sleeping nociceptors,[12–14] which normally are quite insensitive to mechanical stimulation but begin to respond to low-intensity mechanical stimuli following exposure to a combination of the inflammatory mediators bradykinin, histamine, serotonin, and prostaglandin E_2.[14] Sensitization of primary nociceptive neurons leads to *allodynia* (i.e., nociception triggered by stimuli that normally are not painful) and *primary hyperalgesia* (i.e., increased pain from normally less painful stimuli) at the site of tissue injury.

Nociceptive Transduction

In contrast to low-threshold mechanoreceptors, nociceptive sensory endings have no corpuscular accessory structures (for this reason, they are often referred to as *free nerve endings*). As an example, Figure 1–8 shows a three-dimensional reconstruction from serial electron-microscopic sections of a nociceptive nerve ending found in the knee joint.[15] The receptor structure consists only of the axon branches and associated Schwann cells (the nerve ending is not surrounded by perineurium, and there is no trace of myelin). Each axon branch assumes a form of a series of spindle-shaped segments ("beads") connected by smaller-diameter segments. The beads and the end bulb at the tip of the sensory axon show ultrastructural features that are believed to be characteristic of receptive sites (accumulation of vesicles, mitochondria, and glycogen particles in the cytoplasm as well as "bare" areas of the axolemma that are not covered by Schwann cells).

SPECIAL CONSIDERATION

It has been suggested that the sensory part of nociceptive nerve endings is formed by the entire terminal tree and that the beads represent multiple receptive sites.[15]

Study of the nociceptive sensory apparatus (free nerve ending) is made difficult by the fact that it is not truly "free." A nociceptive nerve ending is always surrounded by tissue and therefore not directly accessible for investigation with the presently available electrophysiologic methods. Fortunately, many of the membrane elements that are thought to be present in the sensory endings also may be expressed in the soma membrane of nociceptive neurons. In recent years,

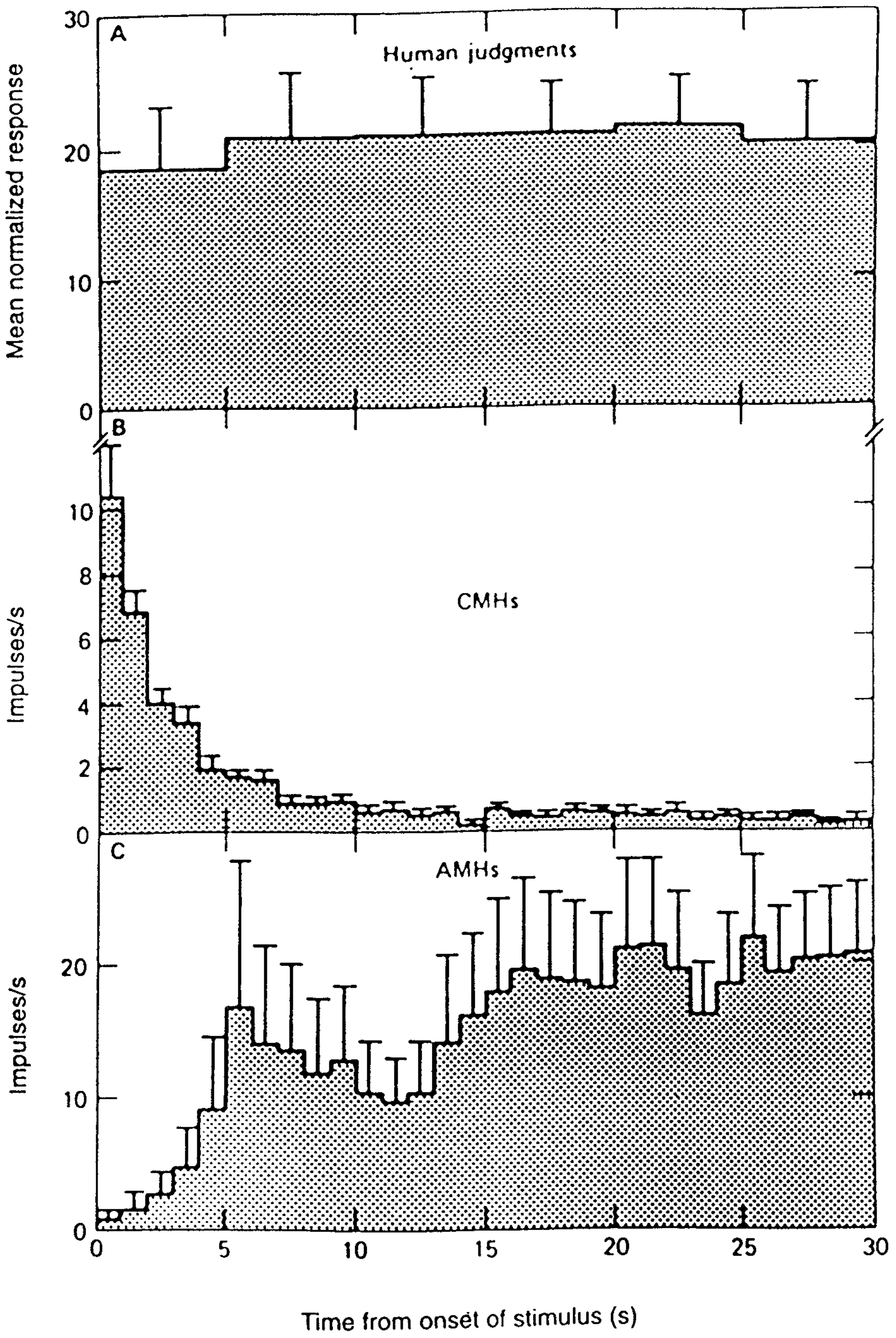

Figure 1–6 Ratings of pain by human subjects during a long-duration, intense heat stimulus (53°C, 30 s duration) applied to the glabrous skin of the hand. Compared are human judgments of the intensity of pain **(A),** the responses of C-fiber mechanoheat (CMH) nociceptors **(B)** and type II A-fiber mechanoheat (AMH) nociceptors **(C)** to an identical heat stimulus. (From Campbell JN, Meyer RA. Cutaneous nociceptors. In: Belmonte C, Cervero F, eds. *Neurobiology of Nociceptors.* Oxford: Oxford University Press; 1996:117–145, with permission.)

this apparent anomaly allowed the study of ion channels, which may be responsible for the transduction of nociceptive stimuli. Patch-clamp recordings from the cell bodies of nociceptive neurons in dissociated cell cultures disclosed ionic currents that are gated (or modulated) by algesic and inflammatory chemicals,[16] heat,[17–22] or mechanical[23–25] stimuli. Studies of nociceptive transduction mechanisms are currently an active area of research at the crossroads of neurophysiology and molecular biology. Several cDNA sequences for ion channels that are preferentially expressed by nociceptive neurons have been cloned.[26–32]

CENTRAL CONNECTIONS OF PRIMARY AFFERENT NOCICEPTIVE NEURONS

Central projections of dorsal root ganglion neurons to the spinal cord, spinal segmental processing of nociceptive in-

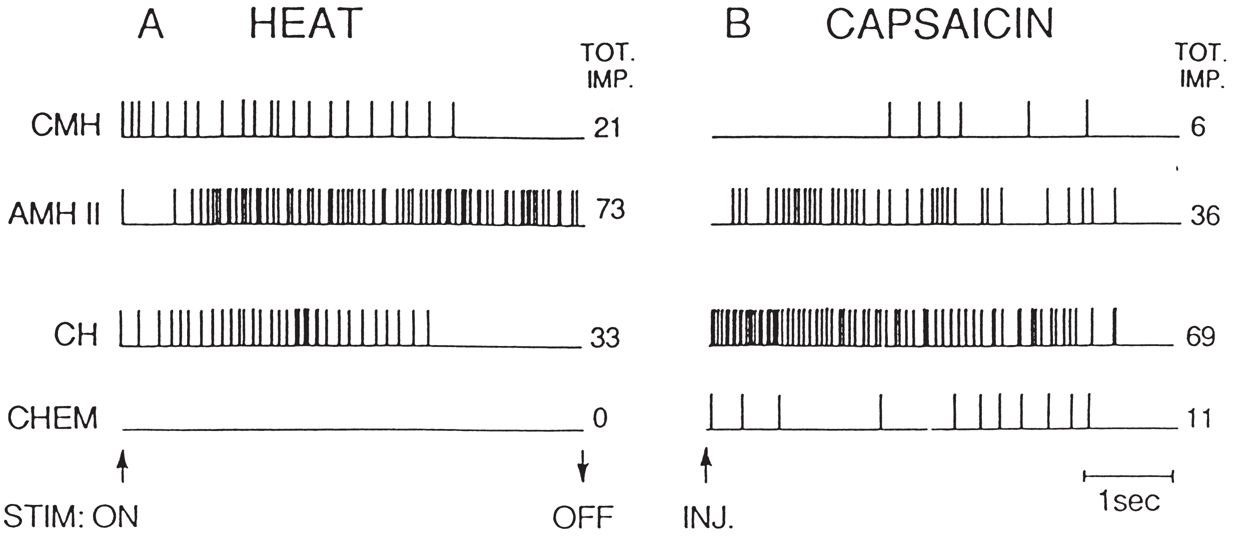

Figure 1–7 Comparison of responses of different physiologic types of nociceptors to heat and noxious chemical stimuli applied to the skin of an anesthetized monkey. **A:** response to a 5-second heat stimulus of 51°C. **B:** Response during the first 5 seconds after intradermal injection of 100 μg of capsaicin. The number to the right of each trace is the total number of action potentials fired by the primary afferent neuron during the 5 seconds. Top three rows illustrate the responses of heat-sensitive nociceptive afferents. CMH, C-fiber mechanoheat nociceptor; AMH II, Type II A-fiber mechanoheat nociceptor; CH, C-fiber heat nociceptor. Bottom row shows the response of a chemonociceptive fiber (CHEM), which was insensitive to heat and mechanical stimuli but responded to capsaicin. Mechanoheat fibers responded more vigorously to heat than to capsaicin. The opposite was true for CH and CHEM fibers. (From Baumann TK, Simone DA, Shain C, LaMotte RH. Neurogenic hyperalgesia: the search for the primary cutaneous afferent fibers that contribute to capsaicin-induced pain and hyperalgesia. *J Neurophysiol.* 1991;66:212–227.)

formation, and spinal nociceptive ascending pathways are addressed next. Subsequent description of trigeminal projections will build on the existing analogies with spinal segmental and ascending systems.

Dorsal Root Ganglion Neurons

Spinal Terminations

The vast majority of dorsal root ganglion neurons reach the spinal cord via the dorsal roots. As the axons approach the spinal cord, nociceptive small-diameter myelinated and unmyelinated axons tend to aggregate in the lateral aspect of the dorsal root; nonnociceptive, large-diameter fibers shift more medially.[33] On entry into the spinal cord, nociceptive dorsal root ganglion neurons either send a primary projection directly into the dorsal horn at the segment of entry, or they form branches that project through the tract of Lissauer (Fig. 1–9). Branches of small-diameter myelinated axons can reach several segments rostrally or caudally, whereas branches of unmyelinated primary afferent fibers travel to a much more limited extent (approximately one spinal segment). Along their rostrocaudal trajectory, the main branches issue several collaterals that leave the tract of Lissauer and enter the dorsal horn. It has been estimated that about 80% of the axons in the Lissauer tract belong to primary afferent neurons,[34] and the remainder belong to a proprioceptive system that originates in lamina II (substantia gelatinosa neurons). In addition to the tract of Lissauer, there is also a lesser known projection of both myelinated and unmyelinated nociceptive primary afferent axons through the dorsal columns.[35,36]

The gray matter of the spinal cord can be clearly divided into 10 layers or regions, each with a distinct cytoarchitecture (Figs. 1–10 and 1–11). Laminae I through VI make up the dorsal horn, and lamina X surrounds the central canal. Cutaneous nociceptive primary afferent neurons with Aδ-fibers terminate in laminae I, IIo (outer lamina II), and V, and C-fiber nociceptors terminate mainly in lamina II (Fig. 1–11). Visceral nociceptas terminate mainly in laminae I and V (in the thoracic cord) or laminae I, II, V, VI, VII, and X (in the sacral cord). Neither lamina that is innervated by nociceptors is innervated exclusively by nociceptors. Lamina I also receives input from innocuous thermoreceptors. Inner lamina II receives substantial input from innocuous thermoreceptors. Inner lamina II receives substantial input from innocuous mechanoreceptors with thin myelinated and unmyelinated fibers. Lamina V receives additional input from many types of myelinated low-threshold mechanoreceptors. Lamina X neurons also are activated by low-threshold inputs.[37]

Some axons of dorsal root ganglion neurons deviate from rule of Magendie[38] (separation of sensory and motor roots) and enter the spinal cord via the ventral (motor) roots instead of dorsal (sensory) roots.[39] The deviant axons traverse the ventral horn to terminate in the dorsal horn (Fig. 1–12).[39]