PRENTICE

# THERAPEUTIC MODALITIES

## IN SPORTS MEDICINE

SECOND EDITION

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# Preface

There is little argument that professional athletic trainers and physical therapists use a wide variety of therapeutic techniques in the treatment and rehabilitation of sports-related injuries. One of the more important aspects of a thorough treatment regimen often involves the use of therapeutic modalities. At one time or another, virtually all sports therapists make use of some type of modality. This may involve a relatively simple technique such as using an ice pack as a first aid treatment for an acute injury or more complex techniques such as the stimulation of nerve and muscle tissue by electrical currents. There is no question that therapeutic modalities are useful tools in injury rehabilitation. When used appropriately, these modalities can greatly enhance the athlete's chances for a safe and rapid return to athletic competition. Unfortunately, the sports therapists' rationale for using a particular modality is too often based on habit rather than on analysis of effectiveness. For the sports therapist, it is essential to possess knowledge regarding the scientific basis and the physiologic effects of the various modalities on a specific injury. When this theoretical basis is applied to practical experience, it has the potential to become an extremely effective clinical method.

What role should a modality play in injury rehabilitation? An effective treatment program includes three primary objectives: (1) management or reduction of pain associated with an injury, (2) return of full nonrestricted range of movement to an injured part, and (3) maintenance or perhaps improvement of strength through the full range. Modalities, though important, are by no means the single most critical factor in accomplishing these objectives. Therapeutic exercise that forces the injured anatomic structure to perform its normal function is the key to successful rehabilitation. However, therapeutic modalities certainly play an important role in reducing pain and are extremely useful as an adjunct to therapeutic exercise.

It must be emphasized that the use of therapeutic modalities in any treatment program is an inexact science. If you were to ask ten different sports therapists what combination of modalities and therapeutic exercise they use in a given treatment program, you would probably get ten different responses. There is no way to "cookbook" a treatment plan that involves the use of modalities. Thus what this book will attempt to do is to present the basis for use of each different type of modality and allow the sports therapists to make their own decisions as to which will be most effective in a given situation. Some recommended protocols developed through the experiences of the contributing authors will be presented.

The sports therapist continues to gain acceptance in the medical community as a highly qualified and well-educated paramedical professional concerned with the treatment and rehabilitation of injuries to athletes. It is essential for the programs educating student trainers and therapists to provide classroom instruction in a wide range of specialty areas including injury prevention, care and management, injury evaluation, and therapeutic treatment and rehabilitation techniques. Detailed instructions in the use of therapeutic modalities should be of primary concern to those who intend to pursue a career in sports medicine.

The use of therapeutic modalities in the treatment of athletic injuries by individuals with various combinations of educational background, certification, and licensure is currently a controversial issue. Formal classroom instruction in the

use of therapeutic modalities is included in all physical therapy programs and is also provided in the majority of athletic training education programs. Physical therapists who are licensed to practice have been given permission to legally use modalities in their patient treatment programs. Likewise, some states have also granted licensure to athletic trainers, thus allowing them full use of therapeutic modalities. Specific laws governing the use of therapeutic modalities vary from state to state. How should modalities be used by athletic trainers who are not licensed by the state in which they are working?

The use of therapeutic modalities has traditionally been in the hands of physical therapists and athletic trainers. The laws of the various states place limitations on this use. The reader of this book should be careful that any use he or she makes of a modality is within the limits allowed by the law of his or her particular state. I do not intend for the reader to interpret anything in this book as encouraging him or her to act outside the scope of the law of his or her state.

The editor hopes that this text will be a useful tool in the continuing growth and professional development of all individuals concerned with and interested in the field of sports injury rehabilitation. The following are a number of reasons why this text should be adopted for use.

**COMPREHENSIVE COVERAGE OF THERAPEUTIC MODALITIES IN A SPORTS MEDICINE SETTING.** The purpose of this text is to provide a theoretically based but practically oriented guide to the use of therapeutic modalities for the individual who routinely treats sports-related injury. It is intended for use in advanced courses in sports medicine where various clinically oriented techniques and methods are presented.

The second edition of this text has been expanded to make the coverage of various modalities more comprehensive. In particular, the chapters on pain, basic principles of electricity, electrical stimulating currents, and massage have been expanded and updated using the latest information available. A new chapter has been added on the latest modality available to the sports therapist, the low-power laser. Also, an appendix has been added that will assist the sports therapist in the clinical decision-making process with regard to the use of the various therapeutic modalities.

This text begins with a discussion of pain, in terms of neurophysiologic mechanisms of pain and the role of therapeutic modalities in pain management. The modalities are then classified in a logical order in relation to the electromagnetic and acoustic spectra. Detailed discussions of various therapeutic modalities, including the infrared modalities, shortwave and microwave diathermies, ultraviolet therapy, ultrasound electrical stimulating currents, low-power laser, massage, and other specialized modalities are presented with emphasis on (1) the physiologic basis for use, (2) clinical applications, and (3) specific techniques of application. Although it is certainly true that therapeutic modalities are important and necessary tools that should be used in dealing with physical problems of all varieties, this text will deal specifically with why and how these modalities are best used in the treatment and rehabilitation of injuries related to sports. This text is the only one available that is oriented specifically toward the use of modalities in the treatment of sports-related injury.

**BASED ON SCIENTIFIC THEORY.** This text discusses various concepts, principles, and theories that are supported by scientific research, factual evidence, and previous experience of the authors in dealing with injuries related to sport. The material presented in this text has been carefully researched by the contributing authors to provide up-to-date information on the theoretical basis for employing a particular modality in a specific injury situation. Additionally, the manuscript for this text has been carefully reviewed by sports therapists, both athletic trainers and physical therapists, who are considered experts in their field to ensure that the material reflects factual and current concepts for modality use.

**TIMELY AND PRACTICAL.** Certainly, therapeutic modalities used in a clinical setting are important tools for the sports therapist. The availability of this text fills a void that has existed for quite some time in the educational program of the student sports therapist. Instructors have been forced to use a variety of randomly selected handouts and photocopied materials in those courses that attempt to provide the student with instruction in the theoretical basis and practical application of the various modalities.

During the preparation of this second edition, the editor received much encouragement from

sports medicine educators regarding the usability of this text in the classroom setting. It should serve as a needed guide for the sports therapist who is interested in knowing not only how to use a modality but also why that particular modality is most effective in a given situation.

The authors who have contributed to this text have a great deal of clinical experience dealing with sports-related injury. Each of these individuals has also at one time or another been involved with the formal classroom education of the student trainer or therapist. Thus this text has been directed at the student of sports-injury rehabilitation who will be asked to apply the theoretical basis of modality use to the clinical setting.

**PERTINENT TO THE SPORTS THERAPIST.** This text deals specifically with the use of therapeutic modalities in the sports medicine setting. Several other texts are available that discuss the use of the physical modalities with patient populations other than athletes. The sports medicine emphasis makes this text unique.

**PEDAGOGICAL AIDS.** The aids this text uses to facilitate its use by students and instructors include:

*Objectives* These goals are listed at the beginning of each chapter to introduce students to the points that will be emphasized.

*Figures and Tables* Essential points on each chapter are illustrated with clear visual materials.

*Summary* Each chapter has a summary that outlines the major points covered.

*Glossary of Key Terms* Each chapter contains a glossary of terms for quick reference.

*References* A list of up-to-date references is provided at the end of each chapter for the student who wishes to read further on the subject being discussed.

*Appendix A* A complete list of manufacturers of therapeutic modality equipment is provided.

**Acknowledgments**

If you have never been involved in the production of a textbook, it is difficult to understand the magnitude of such an undertaking. Dozens of individuals have been involved with this project from its inception, and all have contributed in their own way, but a few deserve special thanks.

Loren Stevenson, my developmental editor at Times Mirror/Mosby, has been responsible for coordinating the efforts between the publisher and me. She has offered much encouragement, constructive suggestions, and extreme patience in the completion of this text.

When assembling a group of contributors for a project such as this it is essential to select individuals who are both knowledgeable and well respected in their fields. It also helps if you can count them as friends, and I want to let them know that I hold each of them in the highest regard, both personally and professionally.

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# Pain and Mechanisms of Pain Relief

1

Phillip B. Donley and Craig Denegar

**OBJECTIVES**

*Following completion of this chapter, the student will be able to:*

- Define pain, its types, and its positive and negative effects.
- Describe the characteristics of sensory receptors.
- Describe an appropriate neurophysiologic mechanism for pain control for the therapeutic modalities used by the sports therapist.
- Describe how pain perception can be modified by cognitive factors.

The International Association for the Study of Pain defines **pain** as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage."[14] Pain is a subjective sensation with more than one dimension and an abundance of descriptors of its qualities and characteristics. In spite of its universality, pain is composed of a variety of human discomforts, rather than being a single entity.[13] The perception of pain can be subjectively modified by past experiences and expectations. Much of what we do to treat athletes' pain is to change their perceptions of pain.[4]

Pain does have a purpose. It warns us that there is something wrong and can provoke a withdrawal response to avoid further injury. It also results in muscle spasm and guarding or protection of the injured part. Pain, however, can persist after it is no longer useful. It can become a means of enhancing disability and inhibiting efforts to rehabilitate the injury. Prolonged spasm, which leads to circulatory deficiency, muscle atrophy, disuse habits, and conscious or unconscious guarding, may lead to a severe loss of athletic ability.[10] Chronic pain may become a disease state in itself. Often lacking an identifiable cause, chronic pain can totally disable a patient.

Research in recent years has led to a better understanding of pain and pain relief. This research also has raised new questions, while leaving many unanswered. We now have better explanations for the analgesic properties of the physical agents we use, as well as a better understanding of the psychology of pain. However, new physical agents, such as the laser and microamperage elec-

trical stimulators, and new approaches to older agents such as transcutaneous electrical nerve stimulators, challenge our understanding of injury and pain. Not even the mechanisms for the analgesic response to heat and cold have been fully described.

The control of pain is an essential aspect of caring for the injured athlete. The sports therapist has several therapeutic agents with analgesic properties from which to choose. The selection of a therapeutic agent should be based on a sound understanding of its physical properties and physiologic effects. This chapter will not provide a complete explanation of neurophysiology, pain and pain relief. Instead, it presents an overview of some theories of pain control, intended to provide a stimulus for the sports therapist to develop his or her own rationale for using modalities in the treatment of injured athletes. Ideally, it also will interest some in research to establish the physiologic and psychologic soundness of the use of agents for pain relief and to expand our understanding of pain. Several physiology textbooks provide extensive discussions of human neurophysiology and neurobiology to supplement this chapter.

Many of the modalities discussed in later chapters have analgesic properties. Often, they are employed to reduce pain and permit the athlete to perform therapeutic exercises. Some understanding of what pain is, how it affects us, and how it is perceived is essential for the sports therapist who uses these modalities.

## TYPES OF PAIN

Pain has been categorized as either **acute** or **chronic.** Pain lasting for more than 6 weeks is generally classified as chronic. There is more research devoted to chronic pain and its treatment, but acute pain, or pain of sudden onset lasting less than 6 weeks, is a more likely problem for the sports therapist.

**Referred pain,** which also may be either acute or chronic, is pain that is perceived to be in an area that seems to have little relation to the existing pathology. For example, injury to the spleen often results in pain in the left shoulder. This pattern, known as **Kehr's sign,** is useful for identifying this serious injury and arranging prompt emergency care. Referred pain can outlast the causative events because of altered reflex patterns, continuing mechanical stress on muscles, learned habits of guarding, or the development of hypersensitive areas, called **trigger points.**

Irritation of nerves and nerve roots can cause **radiating pain.** Pressure on the lumbar nerve roots associated with a herniated disc or a contusion of the sciatic nerve can result in pain radiating down the lower extremity to the foot.

Deep somatic pain is a type that seems to be **sclerotomic** (associated with a **sclerotome,** a segment of bone innervated by a spinal segment). There is often a discrepancy between the site of the disorder and the site of the pain.

## TISSUE SENSITIVITY

The structures most sensitive to damaging (noxious) stimuli are, first, the periosteum and joint capsule; second, subchondral bone, tendons, and ligaments; third, muscle and cortical bone; and finally, the synovium and articular carti-

lage. A variety of "silent" fractures produce little or no pain. Different anatomic tissues exhibit varying degrees of sensitivity to pain. Avulsion fractures tend to be quite painful, because they tear away the periosteum. Musculoskeletal pain is usually spread over a large area unless it is close to the surface. For example, a hamstring strain usually results in pain over the posterior thigh, whereas an acromioclavicular sprain usually localizes over the joint.

## GOALS IN DEALING WITH PAIN

Regardless of the cause of pain, its reduction is an essential part of treatment. Pain signals the athlete to seek assistance and often is useful in establishing a diagnosis. Once the injury or illness is diagnosed, pain serves little purpose. Medical or surgical treatment or immobilization is necessary to treat some conditions, but physical therapy and an early return to activity are appropriate following many athletic injuries. The sports therapist's objectives are to encourage the body to heal through exercise designed to progressively increase the capacity for athletic work and to return the athlete to competition as swiftly and safely as possible. Pain will inhibit therapeutic exercise. The challenge for the sports therapist is to control acute pain and protect the athlete from further injury, while encouraging progressive exercise in a supervised environment.

## PERCEPTION AND TRANSMISSION OF PAIN

### Sensory Receptors

There are several types of sensory receptors in the body, and the sports therapist should be aware of their existence and the types of stimuli that activate them. Activation of some of these sense organs with therapeutic agents will decrease the athlete's perception of pain.

Six different types of receptor nerve endings are encapsulated in connective tissue and are found in the skin:

1. Meissner's corpuscles are activated by light touch.
2. Pacinian corpuscles respond to deep pressure.
3. Merkel's corpuscles respond to deep pressure, but more slowly than pacinian corpuscles, and also are activated by hair follicle deflection.
4. Ruffini corpuscles in the skin are sensitive to touch, tension, and possibly heat, and those in the joint capsules and ligaments are sensitive to change in position.
5. Krause's end bulbs are thermoreceptors that react to a decrease in temperature and touch.[18]
6. Pain receptors, called **nociceptors** or **free nerve endings,** are sensitive to extreme mechanical, thermal, or chemical energy.[3] They respond to noxious stimuli, in other words, to impending or actual tissue damage (for example, cuts, burns, sprains, and so on). The term *nociceptive* is from the Latin *nocere,* to damage, and is used to imply pain information.

These organs respond to superficial forms of heat and cold, analgesic balms, and massage.

Proprioceptors found in muscles, joint capsules, ligaments, and tendons provide information regarding muscle tone. The muscle spindles react to

changes in length and tension when the muscle is stretched or contracted. The Golgi tendon organs also react to changes in length and tension within the muscle. See Table 1-1 for a more complete listing.

Some sensory receptors respond to phasic activity and produce an impulse when the stimulus is increasing or decreasing, but not during a sustained stimulus. They adapt to a constant stimulus. Meissner's corpuscles and pacinian corpuscles are examples of such receptors.

Tonic receptors produce impulses as long as the stimulus is present. Examples of tonic receptors are muscle spindles, free nerve endings, and Krause's end bulbs. The initial impulse is at a higher frequency than later impulses that occur during sustained stimulation.

**Adaptation** is the decline in generator potential and the reduction of frequency that occurs with a prolonged stimulus or with frequently repeated stimuli. If some physical agents are used too often or for too long, the receptors may adapt to or accommodate the stimulus and reduce their impulses. The accommodation phenomenon can be observed with the use of superficial hot and cold agents, such as ice packs and hydrocollator packs.

As a stimulus becomes stronger, the number of receptors excited increases, and the frequency of the impulses increases. This provides more electrical activity at the spinal cord level, which may facilitate the effects of some physcal agents.

**TABLE 1-1 Some Characteristics of Selected Sensory Receptors**

| Type of Sensory Receptors | Stimulus | | Receptor | |
|---|---|---|---|---|
| | General Term | Specific Nature | Term | Location |
| Mechanoreceptors | Pressure | Movement of hair in a hair follicle | Afferent nerve fiber | Base of hair follicles |
| | | Light pressure | Meissner's corpuscle | Skin |
| | | Deep pressure | Pacinian corpuscle | Skin |
| | | Touch | Merkel's touch corpuscle | Skin |
| Nociceptors | Pain | Distension (stretch) | Free nerve endings | Wall of gastrointestinal tract, pharynx, skin |
| Proprioceptors | Tension | Distension | Corpuscles of Ruffini | Skin and capsules in joints and ligaments |
| | | Length changes | Muscle spindles | Skeletal muscle |
| | | Tension changes | Golgi tendon organs | Between muscles and tendons |
| Thermoreceptors | Temperature change | Cold | Krause's end bulbs | Skin |
| | | Heat | Corpuscles of Ruffini | Skin and capsules in joints and ligaments |

Modified from Previte, J.J.: Human physiology, New York, 1983, McGraw-Hill, Inc.

## Neural Transmission

A nociceptive neuron is one that transmits pain signals. Its cell body is in the dorsal root ganglion near the spinal cord. Afferent neurons or nerve fibers conduct impulses from the periphery toward the brain, while efferent fibers, such as motor neurons, conduct impulses from the brain toward the periphery. Approximately 25% of the myelinated Aδ and 50% of the unmyelinated C fibers contact nociceptors and are considered nociceptive, afferent neurons (Table 1-2).

Once a nociceptor is stimulated, it releases a neuropeptide **(substance P)** that initiates the electrical impulses along the afferent fiber toward the spinal cord. Substance P also serves as a transmitter substance between the first-order afferent fiber and a second-order afferent fiber (Fig. 1-1) at the dorsal horn of the spinal column. Many nervous system transmitters conduct the excitation across the synapse and initiate an electrical impulse in the second-order nerve fiber. Such electrical impulses carry sensory messages (pain, warmth, touch) to sensory centers in the brain where they are integrated, interpreted, and acted upon.

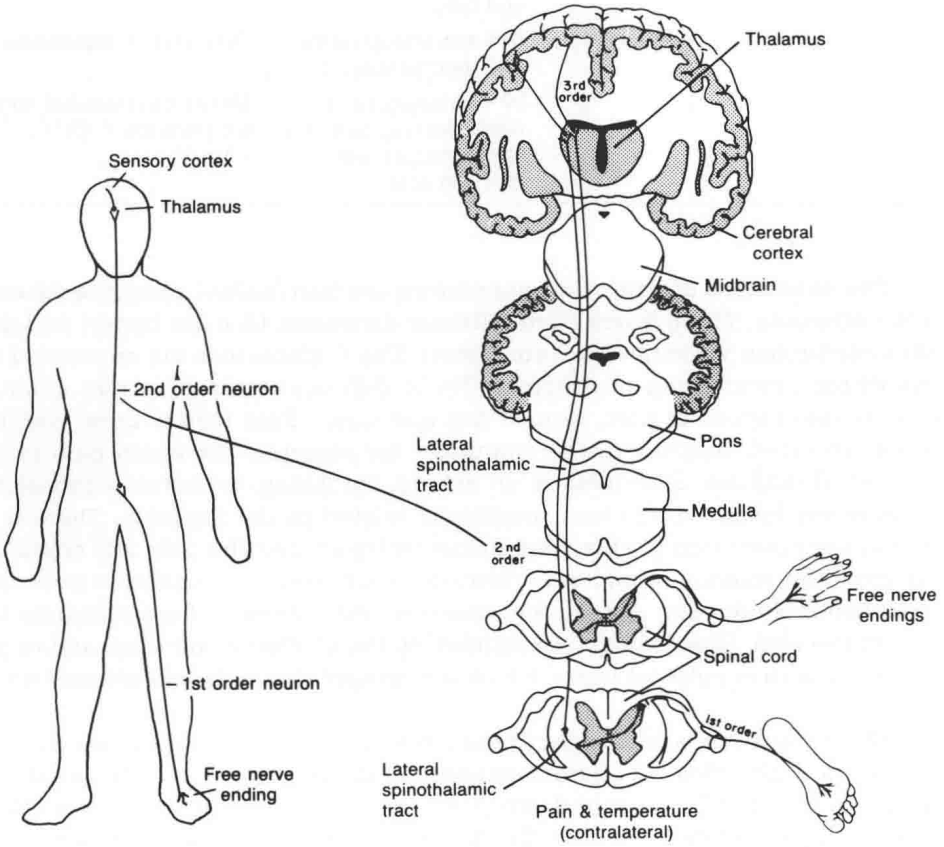


**Figure 1-1.** The lateral spinothalamic tract carries impulses of pain and temperature from the sensory receptors to the cortex.

**TABLE 1-2 Classification of Afferent Neurons**

| Size | Type | Group | Subgroup | Diameter (Micrometers) | Conduction Velocity | Receptor | Stimulus |
|---|---|---|---|---|---|---|---|
| Large | A α | I | 1a | 12-20 (22) | 70-120 | Proprioceptive mechanoreceptor | Muscle velocity and length change, muscle shortening of rapid speed |
| | A α | I | 1b | | | | |
| | A β | II | Muscle | 6-12 | 36-72 | Proprioceptive mechanoreceptor | Muscle length information from touch and pacinian corpuscles |
| | A β | II | Skin | | | Cutaneous receptors | Touch, vibration, hair receptors |
| | A δ | III | Muscle | 1-5 (6) | 6(12)-36(80) | 75% mechanoreceptors and thermoreceptors | Temperature change |
| Small | A δ | III | Skin | | | 25% nociceptors, mechanoreceptors and thermoreceptors (hot and cold) | Noxious mechanical and temperature (>45° C, <10° C) |
| | C | IV | Muscle | 0.3-1.0 | 0.4-1.0 | 50% mechanoreceptors and thermoreceptors | Touch and temperature |
| | C | IV | Skin | | | 50% nociceptors, 20% mechanoreceptors, and 30% thermoreceptors (hot and cold) | Noxious mechanical and temperature (>45° C, <10° C) |

The sensations of pain and temperature are transmitted along the Aδ and C fiber afferents. These fibers have different diameters (A δ are larger) and different conduction velocities (A δ are faster). The C fibers also are connected to more of the nonadapting nociceptors. These differences result in two qualitatively different types of pain, termed *fast* and *slow*.[3] Fast pain is brief, well-localized, and well-matched to the stimulus—for example, the initial pain of an unexpected pinprick. Slow pain is an aching, throbbing, or burning sensation that is poorly localized and less specifically related to the stimulus. There is a delay in the perception of slow pain following injury, but the pain will continue long after the noxious stimulus is removed. Fast pain is transmitted over the larger, faster-conducting A δ afferent neurons and originates from receptors located in the skin. Slow pain is transmitted by the C afferent neurons and originates from both superficial tissue (skin) and deeper tissue (ligaments and muscle).[3]

The various types of afferent fibers follow different courses as they ascend toward the brain. Most C afferent neurons enter the spinal cord through the dorsolateral tract of Lissauer and synapse in an area called the **substantia gelatinosa** with a second-order neuron. The second-order neuron crosses contralaterally to the lateral spinothalamic tract, where it travels up the spinal cord to the thalamus. Here it synapses with a third-order afferent neuron that sends its axon to the postcentral gyrus or the sensory cortex. Most analgesic physical

agents used in sports medicine are believed to slow or block the impulses ascending along the C afferent neuron pathways.

## Synapse Transmission

For information to pass between neurons, a transmitter substance must be released from one neuron terminal (presynaptic membrane), enter the synaptic cleft, and attach to a receptor site on the next neuron (postsynaptic membrane). In the past, all the activity within the synapse was attributed to **neurotransmitters,** such as acetylcholine. It is now apparent that several compounds that are not true neurotransmitters can facilitate or inhibit synaptic activity. These compounds are classified as biogenic amine transmitters or neuroactive peptides. Serotonin and norepinephrine are examples of biogenic amine transmitters. About two dozen neuroactive peptides have been identified, including substance P, enkephalins, and β-endorphin.[3]

Serotonin and enkephalins may be active in descending (efferent) pathways thought to block the pain message.[5] Enkephalin is an endogenous (made by the body) opiate that inhibits the release of substance P. It is released from **interneurons,** enkephalin neurons with short axons. The enkephalins are stored in nerve-ending vesicles found in the substantia gelatinosa and several areas of the brain. When released, enkephalin may bind to presynaptic or postsynaptic membranes.[3]

**Norepinephrine** is a biogenic amine transmitter that is released by the depolarization of some neurons and that binds to the postsynaptic membranes. Analgesia increases with the inhibition of norepinephrine and decreases with its stimulation. An increased level of norepinephrine in the central nervous system usually is associated with decreased analgesia.[1]

Other endogenous opiates may be active analgesic agents. These neuroactive peptides are released into the central nervous system and have an action similar to that of morphine, an opiate analgesic. There are specific receptors located at strategic sites, called *binding sites,* to receive these compounds. **β-Endorphin** is a 31–amino acid peptide with potent analgesic effects. It is released by the anterior pituitary gland and elsewhere within the central nervous system.

## NEUROPHYSIOLOGIC EXPLANATIONS OF PAIN CONTROL

The neurophysiologic mechanisms of pain control through stimulation of cutaneous receptors have not been fully explained. Much of what is known and current theory are the result of work involving transcutaneous electrical nerve stimulation. However, this information often provides an explanation for the analgesic response to other modalities, such as massage, analgesic balms, and moist heat.

The models of the analgesic response to cutaneous receptor stimulation presented here were first proposed by Melzack and Wall[12] and Castel.[5] These models essentially present three analgesic mechanisms:

1. Stimulation from ascending A β afferents results in the blocking of impulses (pain messages) carried along A δ and C afferent fibers.
2. Stimulation along descending pathways in the dorsal horn of the spinal cord