Knowledge is an important tool in treating pain. With the knowledge of pain etiology, the pathways that carry the message of pain to the brain, the social and psychological aspects of pain perception, and the ways in which pharmaceutical agents block pain, athletic trainers and therapists can communicate pain-management options to their athletes. In the daily course of the job, we must deal with our athletes’ pain in order to treat and manage their sports-related injuries. Disregarding an athlete’s pain in the injury-management process can retard rehabilitation.

Knowledge is an important tool in treating pain. With the knowledge of pain etiology, the pathways that carry the message of pain to the brain, the social and psychological aspects of pain perception, and the ways in which pharmaceutical agents block pain, athletic trainers and therapists can communicate pain-management options to their athletes. In the daily course of the job, we must deal with our athletes’ pain in order to treat and manage their sports-related injuries. Disregarding an athlete’s pain in the injury-management process can retard rehabilitation.

The objectives of this article are to review three current and well-accepted pain theories, provide ideas to clinicians who are teaching injured athletes about pain, and apply the theories to athletic training and therapy practice in traditional and nontraditional patient treatment.

Endogenous opiates and enkephalins are natural peptides produced and released by the body to relieve pain.

Key Words: gate-control theory, central-biasing theory, pain tolerance, endogenous opiates, enkephalins

Pain has been defined as an unpleasant sensory and emotional experience arising from actual or potential tissue damage or described in terms of such damage. Pain includes not only the perception of an uncomfortable stimulus but also the response to that perception. (Thomas, 1997, p. 1387)

Pain perception is the body’s way of signaling danger but also can often hinder rehabilitation, activities of daily living, and athletic activities.

Gate-Control Pain Theory

Melzack and Wall’s gate-control theory of pain is the first of the three pain theories reviewed. Within the spinal cord lies a group of nerve cells known as the substantia gelatinosa, also referred to as Laminae II and III of the dorsal horn. Using a gate as an analogy, the substantia gelatinosa is a “gate keeper” that determines whether a sensory message will reach the brain or be blocked. The gate plays an important role in pain management of the central nervous system (CNS). Pain messages that pass through the gate stimulate T cells (transport cells) of the dorsal horn in the spinal cord and facilitate message transport to the brain. T cells send messages up the spinal cord, much as an elevator transports people to the top floor of a building.

The gate-control theory is predicated on transporting messages to the brain via impulses that are received on a “first come, first served” basis, and the strength of the incoming impulses also seems to dictate their influence on the gate.
For example, if a 20-lb dumbbell is dropped on the thumb, the weight contacts the thumb and a pain stimulus is created at the pain receptors (nociceptors) in the skin. The pain-carrying A-delta nerve fibers in the peripheral nervous system are activated first and start the message (bright, local pain) moving toward the spinal cord, at a rate of approximately 15 m/s. Activation of C nerve fibers sends a slow pain message (slow, dull, diffuse pain; conducting speed no faster than 2 m/s) that follows the same pathway. The pain messages proceed to the substantia gelatinosa, where substance P is released. The gate is opened and the message is sent to the T cell, which transports the message in the “elevatorlike” spinothalamic or spinoreticular tract in the spinal cord. The message ascends to the reticular formation, which is the part of the brainstem that influences alertness, waking, sleeping, and reflexes (Gilman & Newman, 1992). When the reticular formation is stimulated, autonomic motor and sensory responses are quickly produced (Buxton, 1999).

This explains why athletic trainers and therapists are taught to pull an athlete’s arm hair, press on the nail bed, or grind their knuckles into the sternum to assess the condition of a potentially brain-injured athlete. If the reticular formation is unharmed, the pain reflex results in a motor action (pulling the arm or finger away or becoming alert) in response to the painful stimulus. The pain threshold determines the first pain felt and is a function of the reticular formation. Once the reticular formation relays the message, the stimulus travels on to synapse with other structures of the midbrain and central brain (Buxton, 1999).

The central brain, or cortex, perceives the sensory message of sharp pain (caused by the dumbbell’s effect) on the left thumb and sends an afferent message back down the spinal cord in the “elevator,” which is now descending. Multiple messages to the descending tract can either exaggerate or inhibit the perception of pain. Muscle movement is the result of message facilitation resulting in shaking the left hand or rubbing or massaging the left thumb. Consequently the receptors in the thumb have a new message; rubbing the thumb now sends a message of pressure. The new message is sent along a different pathway, the afferent A-beta nerve—a large, myelinated nerve with a low threshold for stimulation. The A-beta nerve moves the message so fast (up to 70 m/s) that it will reach the gate in the substantia gelatinosa more quickly than the next message of pain from the thumb (Gilman & Newman, 1992). The new message of pressure occupies the gate, and the next message of pain cannot get through the gate to the T cell. As long as the thumb is being rubbed, pressure rather than pain is perceived. When the rubbing stops, the message of pressure no longer occupies the gate and the message of pain is allowed to move to the brain, where pain will be perceived and analyzed. This is, of course, a simplification of a complex series of events. There are many gates in the spinal cord, and all would have to be blocked to cancel the pain. At best, rubbing the thumb decreases the pain sensation rather than eliminating it. In short, the gate-control theory of pain states that nonpainful stimuli can block painful stimuli (Buxton, 1999). The perception of pain depends on whether the dominant message is the one ascending to deliver the message of pain or the descending one, which will act to inhibit the painful stimuli (Watt-Watson, 1999).

Acute pain serves an important function because it often protects the body from further harm. When pain continues past the protection and warning phase, it becomes problematic. Chronic pain, pain that lasts longer than 6 months postinjury, can also be explained by the gate-control theory (Prentice, 1999). Through a negative-feedback system in the spinal cord, the gate remains open, with less input from the large A-beta afferent nerves and more input from the small pain-carrying sensory nerves (Buxton, 1999). Overriding of small pain-carrying nerves by large afferent nerves is the foundational theory behind the use of transcutaneous electrical nerve stimulators (TENS) as a pain-modulating tool (Figure 1). TENS stimulates the afferent nerve fibers that travel along large myelinated nerves to the substantia gelatinosa that occupy the gate, thus blocking the painful stimuli traveling along the smaller unmyelinated nerve fibers to the gate. Ice application and electrical stimulation work by stimulating sensory nerves in the skin to produce pain relief. The applications of the gate theory account for the effectiveness of analgesic balms and other counterirritants in changing the perception of pain (Prentice). Some joint-mobilization techniques are believed to stimulate mechanoreceptors and block nociceptive pathways (Kisner & Colby, 1996). In practical experience, the gate theory seems to be valid.