

Section 1

Since the chiropractic profession's genesis in 1895, Doctors of Chiropractic have played a vital role in the diagnosis and treatment of pain. Emphasis has been placed on the etiology of pain syndromes, how the body reacts to pain, how different chiropractic treatment methodologies ameliorate or eliminate pain, and how alternative treatments can be used to relieve many painful conditions. In addition to chiropractic treatment, pain management generally benefits from a multidisciplinary approach that includes pharmacologic measures (to include analgesics such as non steroidal anti inflammatory medicines), pain modifiers (such as tricyclic antidepressants or anticonvulsants), non-pharmacologic measures (such as physical therapy and physical exercise), and psychological measures (such as biofeedback and cognitive therapy).

The purpose of this continuing education seminar is to broaden the Doctor of Chiropractic's knowledge base regarding the nature of pain, pain theories and the physiological and non physiological aspects of pain. Additionally, the course information is intended to enhance the reader's diagnostic abilities relative to pain syndromes and to provide an overview of chiropractic and alternative pain management methodologies.

Pain Perspectives

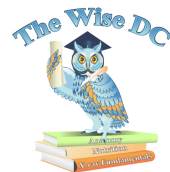
The International Association for the Study of Pain (IASP) defines pain as:

"An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage". Pain is always subjective. Each individual learns the application of the word through experiences related to injury in early life. Accordingly, pain is that experience we associate with actual or potential tissue damage. It is unquestionably a sensation in a part or parts of the body, but it is also always unpleasant and therefore also an emotional experience. Many people report pain in the absence of tissue damage or any likely pathophysiological cause. Typically, this happens for psychological reasons. There is usually no way to distinguish their experience from that due to tissue damage if we take the subjective report. If they regard their experience as pain and if they report it in the same ways as pain caused by tissue damage, it should be accepted as pain. This definition avoids tying pain to the stimulus. Activity induced in the nociceptor and nociceptive pathways by a noxious stimulus is not pain, which is always a psychological state, even though we may well appreciate that pain most often has a proximate physical cause.¹

"Clinically, pain is whatever the person says he or she is experiencing whenever he or she says it occurs"²

"Pain is a category of complex experiences, not a single sensation produced by a single stimulus" - Ronald Melzack and Patrick Wall³

"Pain is the result of nociception; activity in the nervous system that results from the stimulation of nociceptors. This nociception activity is carried to the brain via the spinal cord, and conveys information, without conscious awareness, about damage to body tissues. Pain is the conscious experience of sensorial information and a feeling of unpleasantness that can manifest as a result of nociception. As a part of the body's defense system, pain triggers mental and physical behavior that seek to end the painful experience. It is also a feedback system that promotes learning, making repetition of the painful situation less likely. The nociceptive system may transmit signals that trigger the sensation of pain, it is a critical component of the body's ability to react to damaging stimuli and it is part of a rapid-warning relay instructing diverse organs and principally the central nervous system to initiate reactions for minimizing injury."⁴



Pain Epidemiology

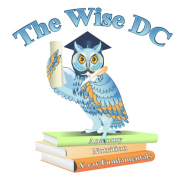
- In the USA, 23.3 million surgical procedures are performed each year, and most, if not all, result in some form of pain^{5,6,7}
- Pain in persons with cancer also remains a significant problem, with studies suggesting that as many as 30% to 40% of cancer patients and 70% to 80% of cancer patients undergoing therapy or in the end stages of life have unrelieved pain^{8,9,10,11,12,13}
- The Mayday Fund survey noted that pain is a part of life for many Americans, with 46% of respondents reporting pain at some time in their lives.¹⁴
- It has been estimated that 9% of the US adult population suffers from moderate to severe chronic nonmalignant pain.¹⁵

Epidemiology

- A complaint of pain is reported to be the main reason for visiting the emergency department in more than 50% of cases¹⁶ and is present in 30% of family practice visits.¹⁷
- Epidemiological studies of differing countries and cultures have reported widely varying prevalence rates for chronic pain, ranging from 12-80% of the population.¹⁸
- Studies suggest that the presence of pain becomes more common as people approach death. A study of 4,703 patients found that 26% had pain in the last two years of life, increasing to 46% in the last month.¹⁹
- Perquin et al. performed a survey of 6,636 children (0–18 years of age) and found that 54% of their respondents had experienced pain in the preceding three months. A quarter reported having experienced recurrent or continuous pain for three months or more, and a third of these reported frequent and intense pain. The intensity of chronic pain was higher for girls, and girls' reports of chronic pain increased markedly between ages 12 and 14.²⁰

An Australian Study of the Epidemiology of Pain By Simon L Strauss, Fiona H Guthrie*, and Fred Nicolosi* dated 1986 revealed the following **key findings** :

- the pain prevalence rate was 355 per 1000.
- as age increases the pain prevalence rate increases.
- Females have higher pain prevalence rates than males over all age 80 groups.
- the majority of respondents reported suffering from back pain.
- the majority of respondents described their pain as discomforting (the second point of a five point scale based on the McGill Pain Questionnaire).
- the cause of pain for the majority of respondents was of unknown or spontaneous origin.
- the majority of respondents had suffered from pain for three years or more.
- the pain is generally experienced either continuously or on a daily basis.
- the majority of respondents (70%) visited a health professional for treatment. This health professional was a doctor in 80% of cases.
- respondents undertaking self-treatment or no treatment did so because they considered health professionals could not help.²¹



Peter M Kent and **Jennifer L Keating** in their 2005 study entitled “The epidemiology of low back pain in primary care” reported the following findings:

- In the studies Looney and Stratford, low back pain (LBP) prevalence was estimated to be 6.8% in North America, 12% in Sweden, 13.7% in Denmark, 14% in the United Kingdom, 28.4% in Canada, and 33% in Belgium.²²
- The prevalence of LBP in children is low (1%-6%) but increases rapidly (18%–50%) in the adolescent population. The prevalence of LBP peaks around the end of the sixth decade of life.²³
- In the USA, for people aged 45 years or less, LBP is the most frequent cause of activity limitation and women were twice as likely to report severe activity limitation.²⁴
- Carey found that in a sample from North Carolina USA, 61% of recent-onset (<12 weeks) LBP sufferers sought care during their most recent episode. Those seeking care were likely to have more intense pain, leg pain, or a pain onset at work, than those who did not seek care.²⁵
- In an Australian study Walker found women more likely to seek care for LBP (adjusted odds ratio 1.7, 95%CI 1.3 to 2.2).²⁶
- The most common clinicians consulted for back pain in North America are chiropractors, general medical practitioners and orthopaedists.²⁷
- LBP is a sizeable proportion of casemix for some primary-contact disciplines. Physiotherapy LBP casemix has been estimated to be 25% .²⁸
- Chiropractic LBP casemix has been estimated to be 41%. Back pain is the ninth most common presentation in general medical practice, contributing between 3.8% and 7.1% of presenting complaints.²⁹
- The six most common types of treatment received by adults when seeking care for LBP are back exercises/stretching, massage, spinal manipulation, prescribed medication, non-prescription medication, and bed rest.³⁰
- Von Korff defined natural history as the development of a condition in the absence of treatment, and defines clinical course as its development in the presence of treatment. Recent systematic reviews of the clinical course of LBP indicate that rapid improvements occur in the first three months post-onset, but that improvements are gradual thereafter. At 6 months post-onset, 16% (range 3–40%) of patients initially off-work remain off-work, and at 12 months post-onset, 62% (range 42–75%) still have pain. Within 12 months of onset, recurrences of both pain (60%, range 44–73%), and recurrences of work absence (33%, range 26–37%) are common.^{31 32}

Pain Definitions

Allodynia

Pain due to a stimulus that does not normally provoke pain. The term allodynia was originally introduced to distinguish hyperalgesia from hyperesthesia, the conditions seen in patients with lesions of the nervous system where touch, light pressure, or moderate cold or warmth evoke pain when applied to apparently normal skin. Allo means "other" in Greek and is a common prefix for medical conditions that diverge from the expected.

Odynia is derived from the Greek word "odune" or "odyne," which is used in "coccydynia" and is similar in meaning to the root from which we derive words with -algia or -algesia in them. The term allodynia applies to conditions which may give rise to sensitization of the skin, e.g., sunburn, inflammation, trauma. Allodynia involves a change in the quality of a sensation, whether tactile, thermal, or of any other sort.³³

Analgesia

Absence of pain in response to stimulation which would normally be painful.³⁴

Anesthesia Dolorosa

Pain in an area or region which is anesthetic.³⁵

Breakthrough Pain

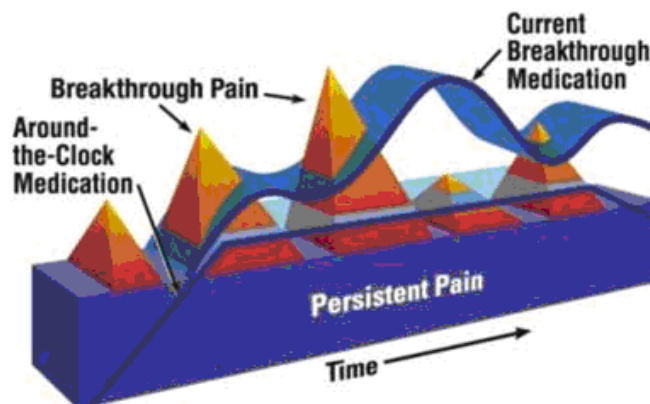
Moderate to severe pain that lasts 12 hours or more per day is referred to as persistent pain. Medical physicians will often prescribe a longer-acting medication that will help to alleviate persistent pain for up to 12 hours or more. Unfortunately, up to 86% of patients already receiving longer-acting pain medication also experience sudden flare-ups of pain that "break through" the medication they are taking.³⁶ This is called "breakthrough pain," or "BTP."

In patients who have moderate to severe pain, two components are usually present: persistent pain (lasting 12 or more hours/day) and breakthrough pain (BTP), a transitory flare up of pain of moderate to severe intensity occurring on a background of otherwise controlled pain.^{37,38,39}

In one survey, patients experienced an average of 4 episodes of BTP per day.⁴⁰ Based on the results from this same survey, the onset of BTP is often sudden, reaches maximal intensity within 3 minutes, and lasts for a median duration of 30 minutes.⁴¹

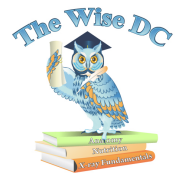
There are 3 types of breakthrough pain - spontaneous, incident, and end-of-dose failure. The etiology of BTP may be related to a disease or condition, or to its treatment.

Breakthrough pain has been further defined as a transitory exacerbation of moderate to severe pain occurring in patients against a background of persistent pain otherwise controlled with chronic opioid therapy.⁴²



Breakthrough pain strikes quickly and without warning in many cases.⁴³ Untreated breakthrough pain can decrease a patient's quality of life by negatively affecting their mood and interactions with others, and by limiting their activities of daily living.⁴⁴

Medications that are typically prescribed for persistent pain are called "longer-acting," "sustained-release," or "around-the-clock" medicine. Some examples of longer-acting medicines are pills that are taken every 8 to 12



hours or a skin patch that is worn for several days. Conversely, medicines required for breakthrough pain are called "shorter-acting," "supplemental," or "rescue" medicines.

In the assessment of pain and in the development of a diagnosis and treatment plan, it is important to understand the following characteristics regarding breakthrough pain:

- Episodes of breakthrough pain may either be spontaneous, occurring without a precipitated event, or precipitated; initiated by a volitional or non-volitional event. The etiology of breakthrough pain can be related to a disease or condition, or to its treatment. Episodic pain is also known as breakthrough pain.
- Chronic pain can be one or two types: persistent pain, which is continuous pain, and breakthrough pain, or incident pain.
- Breakthrough pain is called that because it "breaks through" a regular pain medicine schedule.
- Breakthrough pain may be different for each person and it is often unpredictable.
- Breakthrough pain typically has a rapid onset, can last up to an hour.
- The quality of breakthrough pain may feel very much like persistent pain, except that it is more severe.
- Breakthrough pain is generally a result of the same cause, or source, as persistent pain.
- In 1994, the Agency for Healthcare Research and Quality (AHCPR) defined breakthrough pain as "intermittent exacerbations of pain that can occur spontaneously or in relation to specific activity". The Agency for Healthcare Research and Quality (AHCPR) guidelines do not contain specific recommendations for assessing breakthrough pain and/or selecting and analgesic or analgesic doses of medication to treat breakthrough pain.
- Movement-related pain is often referred to as "incident" pain. Incident pain, or movement-related pain, can occur as a result of coughing, swallowing, positional changes, and activity.
- Breakthrough pain could be related to: a direct relationship between a tumor progression, such as bone metastasis, or nerve compression in the cancer patient, or as a result of a treatment modality, such as a prostatectomy or mastectomy, or a variety of disorders, such as arthritis.
- Currently, there is not an independently validated assessment tool available to evaluate breakthrough pain.⁴⁵

Causalgia

A syndrome of sustained burning pain, allodynia, and hyperpathia after a traumatic nerve lesion, often combined with vasomotor dysfunction and later trophic changes.⁴⁶

Central Pain

Pain initiated or caused by a primary lesion or dysfunction in the central nervous system.⁴⁷

Dysesthesia

An unpleasant abnormal sensation, whether spontaneous or evoked. Special cases of dysesthesia include hyperalgesia and allodynia.⁴⁸

Hyperalgesia

An increased response to a stimulus which is normally painful. Hyperalgesia reflects increased pain on suprathreshold stimulation. For pain evoked by stimuli that usually are not painful, the term allodynia is preferred, while hyperalgesia is more appropriately used for cases with an increased response at a normal threshold, or at an increased threshold, e.g., in patients with neuropathy.. Current evidence suggests that hyperalgesia is a consequence of perturbation of the nociceptive system with peripheral or central sensitization, or both.⁴⁹



Hyperesthesia

Increased sensitivity to stimulation. Hyperesthesia may refer to various modes of cutaneous sensibility including touch and thermal sensation without pain, as well as to pain. The word is used to indicate both diminished threshold to any stimulus and an increased response to stimuli that are normally recognized.⁵⁰

Hyperpathia

A painful syndrome characterized by an abnormally painful reaction to a stimulus, especially a repetitive stimulus, as well as an increased threshold. It may occur with allodynia, hyperesthesia, hyperalgesia, or dysesthesia. Faulty identification and localization of the stimulus, radiating sensation, and after-sensation may be present, and the pain is often explosive in character.

Hypoalgesia

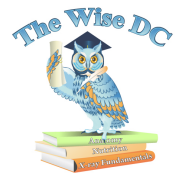
Diminished pain in response to a normally painful stimulus. Hypoalgesia refers only to the occurrence of relatively less pain in response to stimulation that produces pain.⁵¹

Hypoesthesia

Decreased sensitivity to stimulation that is normally painful.⁵²

Neuralgia

Pain in the distribution of a nerve or nerves.



Section 2

Nociception

“Pain refers to the subjective sensation that accompanies damage or near-damage to tissues. Pain can also occur in the absence of tissue damage if the systems of nociception are not functioning properly. Nociception is the physiological event that accompanies pain and refers to the system that carries signals of damage and pain from the tissues.”⁵³

Transmission of Nociception to the Central Nervous System

Nociceptive information reaches the central nervous system two ways via the neospinothalamic tract for "fast spontaneous pain"; and by way of the paleospinothalamic tract for "slow increasing pain"⁵⁴

Neospinothalamic Tract

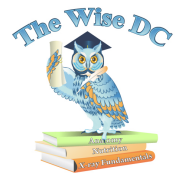
Fast spontaneous pain travels via type A-delta fibers to terminate on the dorsal horn of the spinal cord where they synapse with the dendrites of the neospinothalamic tract. The axons of these neurons travel up the spine to the brain and cross the midline through the anterior white commissure, passing upwards in the contralateral anterolateral columns. These fibers terminate on the ventrobasal complex of the thalamus and synapse with the dendrites of the somatosensory cortex. Fast pain is felt within a tenth of a second of application of the pain stimulus and is a sharp, acute, prickling pain felt in response to mechanical and thermal stimulation.⁵⁵

Paleospinothalamic Tract

Slow pain is transmitted via slower type C fibers to laminae II and III of the dorsal horns, together known as the substantia gelatinosa. Impulses are then transmitted to nerve fibers that terminate in lamina V, also in the dorsal horn, synapsing with neurons that join fibers from the fast pathway, crossing to the opposite side via the anterior white commissure, and traveling upwards through the anterolateral pathway. These neurons terminate throughout the brain stem, with one tenth of the fibers stopping in the thalamus and the rest stopping in the medulla, pons and periaqueductal grey of the midbrain tectum. Slow pain is stimulated by chemical stimulation, is poorly localized and is described as an aching, throbbing or burning pain.⁵⁶

A-Delta and C Fiber Synopsis

- “The thin (A-delta and C) peripheral sensory fibers carry information regarding the state of the body to the spinal cord.- Some of these thin fibers do not differentiate noxious from non-noxious stimuli, while others, nociceptors, respond only to painfully intense stimuli.”⁵⁷⁵⁸
- “Because the A-delta fiber is thicker than the C fiber and is thinly sheathed in an electrically insulating material (myelin), it carries its signal faster (5–30 m/s) than the unmyelinated C fiber (0.5–2 m/s).- Pain evoked by the (faster) A-delta fibers is described as sharp and is felt first. This is followed by a duller pain, often described as burning, carried by the C fibers.”⁵⁹⁶⁰



- “Spinal cord fibers dedicated to carrying A-delta fiber pain signals, and others that carry both A-delta and C fiber pain signals up the spinal cord to the thalamus in the brain have been identified. Other spinal cord fibers, known as wide dynamic range neurons, respond to A-delta and C fibers, but also to the large A-beta fibers that carry touch, pressure and vibration signals. Pain-related activity in the thalamus spreads to the insular cortex (thought to embody, among other things, the feeling that distinguishes pain from other homeostatic emotions such as itch and nausea) and anterior cingulate cortex (thought to embody, among other things, the motivational element of pain.”⁶¹

Effects in the Central Nervous System

When nociceptors are stimulated they transmit signals through sensory neurons in the spinal cord. These neurons release the excitatory neurotransmitter glutamate at their synapses. If the signals are sent to the reticular formation and thalamus, the sensation of pain enters consciousness in a dull, poorly localized manner. From the thalamus, the signal can travel to the somatosensory cortex in the cerebrum when the pain is experienced as localized and having more specific qualities.

Nociception can also cause generalized autonomic responses before or without reaching consciousness to cause pallor, bradycardia, hypotension, lightheadedness, nausea and fainting.⁶²

Nociceptors

Nociceptors are free nerve endings whose cell bodies are found outside of the spinal column in the dorsal root ganglia. Nociceptors can detect mechanical, thermal, and chemical stimuli, and are found in the skin and on internal structures such as the periosteum or joint surfaces.

The body's pain receptors are:

- **Mechanical:** capable of detecting a stretch gone too far.
- **Thermal:** receptors capable of detecting extreme heat or cold.
- **Chemical:** receptors that can detect body products released during trauma or inflammation. (Lactic acid, for example, causes muscle pain after heavy exercise.)

Deep internal surfaces are only weakly supplied with pain receptors and will propagate sensations of chronic, aching pain if tissue damage in these areas occurs. Nociceptors do not adapt to stimuli. In some conditions, excitation of pain fibers becomes greater as the pain stimulus continues, leading to hyperalgesia.

Noxious Stimulus

A noxious stimulus is one which is damaging to normal tissues.

Pain Threshold

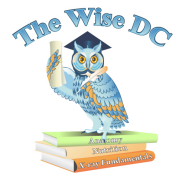
Is the least stimulus intensity at which a subject perceives pain. Pain threshold is really the experience of the patient, whereas the intensity measured is an external event. In psychophysics, thresholds are defined as the



level at which 50% of stimuli are recognized. In that case, the pain threshold would be the level at which 50% of stimuli would be recognized as painful. The stimulus is not pain and cannot be a measure of pain.⁶³

Peripheral Neuropathic Pain

Pain initiated or caused by a primary lesion or dysfunction in the peripheral nervous system.



Section 3

Pain Theories

Intensive Theory

In 1794 Erasmus Darwin postulated the idea in Plato's *Timaeus*, that pain is not a unique sensory modality, but an emotional state produced by stronger than normal stimuli such as intense light, pressure or temperature.^{64 65}

Following up on Darwin's findings, in 1874 Wilhelm Erb opined that pain can be generated by any sensory stimulus, provided it is intense enough, and his formulation of the hypothesis became known as the intensive theory.⁶⁶ Wilhelm Erb's intensive theory, that a pain signal can be generated by intense enough stimulation of any sensory receptor, has been soundly disproved.

Subsequent research in 1884 by Alfred Goldscheider confirmed the existence of distinct heat and cold sensors, by evoking heat and cold sensations using a fine needle to penetrate to and electrically stimulate different nerve trunks, bypassing their receptors. Though he failed to find specific pain sensitive spots on the skin, Goldscheider concluded in 1895 that the available evidence supported pain specificity.

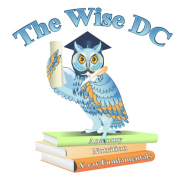
Bernhard Naunyn performed pain experimentation in 1889 in which he rapidly (60–600 times/second) prodded the skin of tabes dorsalis patients, below their touch threshold (e.g., with a hair), and in 6–20 seconds produced unbearable pain. He obtained similar results using other stimuli including electricity to produce rapid, sub-threshold stimulation, and concluded pain is the product of summation.

“ In 1894 Goldscheider extended the intensive theory, proposing that each tactile nerve fiber can evoke three distinct qualities of sensation – tickle, touch and pain – the quality depending on the intensity of stimulation; and extended Naunyn's summation idea, proposing that, over time, activity from peripheral fibers may accumulate in the dorsal horn of the spinal cord, and "spill over" from the peripheral fiber to a pain-signalling spinal cord fiber once a threshold of activity has been crossed.^{67 68}

“William Kenneth Livingston advanced a summation theory in 1943, proposing that high intensity signals, arriving at the spinal cord from damage to nerve or tissue, set up a reverberating, self-exciting loop of activity in a pool of interneurons, and once a threshold of activity is crossed, these interneurons then activate "transmission" cells which carry the signal to the brain's pain mechanism; that the reverberating interneuron activity also spreads to other spinal cord cells that trigger a sympathetic nervous system and somatic motor system response; and these responses, as well as fear and other emotions elicited by pain, feed into and perpetuate the reverberating interneuron activity. A similar proposal was made by RW Gerard in 1951, who proposed also that intense peripheral nerve signalling may cause temporary failure of inhibition in spinal cord neurons, allowing them to fire as synchronized pools, with signal volleys strong enough to activate the pain mechanism.”⁶⁹

The Specificity Theory Of Pain

In 1644, French philosopher, mathematician and scientist, René Descartes proposed a theory of pain that survived until the mid-1960s. Descartes postulated that the human body was a form of machine and therefore, could be studied like other mechanical entities. According to Descartes' classic thoughts regarding pain, if a



person cut their finger a half-inch in length, it would hurt twice as much as if they had cut it a quarter of an inch in length. In Descartes' model, pain traveled in a single direction. Under Descartes' influence, the scientific search for how pain was conducted culminated in what has come to be known as the specificity theory. This theory posits that pain is a simple system where an input of one kind travels along special nerves for that kind of input, terminating in specific areas of the brain which are receptive to that input. The input which went in was thought to be what was felt by the brain. This belief resulted in the use of various inappropriate kinds of therapy, including the cutting of nerve pathways to try and abolish chronic pain.⁷⁰

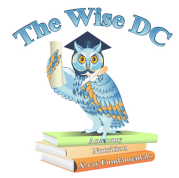
“Descartes theory proposed that the intensity of pain is directly related to the amount of associated tissue injury. For instance, pricking one's finger with a needle would produce minimal pain, whereas cutting one's hand with a knife would cause more tissue injury and be more painful. This theory - the “specificity theory” - is generally accurate when applied to certain types of injuries and the acute pain associated with them. But chronic pain is often quite different, though no less severe, and a more extensive and up to date scientific understanding of pain is required to treat it. Unfortunately, many practicing doctors still try to extend the specificity theory to chronic pain cases. This approach is probably not valid when studying or treating chronic back pain. The theory assumes that if surgery or medication can eliminate the alleged "cause" of the pain, then the chronic pain will disappear. This is very often not true for chronic pain. If doctors continue to apply the specificity theory to a patient's chronic pain problem, the patient is at risk for receiving unnecessary and ineffective diagnostic procedures, drugs and surgical treatment as the search for the patient's "source of chronic back pain" presses on.”⁷¹

Problems With The Specificity Theory And Chronic Pain

Up until the introduction of the gate control theory of pain in 1965, the specificity theory had been the dominant idea in the study of pain. One of the first doctors to question its validity was Dr. Henry Beecher. Dr. Beecher began his investigation into relationships between subjective psychological states and objective drug responses during his work with severely wounded soldiers in World War II.

Beecher's clinical observations proved that the specificity theory was inadequate to explain chronic pain. He observed that only one out of five soldiers carried into a combat hospital complained of enough pain to require morphine. When Dr. Beecher returned to his practice in the United States after the war, he noticed that trauma patients with wounds similar to those of the soldiers he had treated were much more likely to require morphine to control their pain. In fact, one out of three civilian patients required morphine for pain from these wounds. Dr. Beecher concluded that there was no direct relationship between the severity of the wound and the intensity of pain. Dr. Beecher believed the meaning attached to the injuries in the two groups explained the different levels of pain. To the soldier, the wound meant surviving the battlefield and returning home. Alternatively, the injured civilian often faced major surgery and a resulting loss of income, diminishment of activities, and many other negative consequences.⁷²

Another finding that discredited the specificity theory was that of phantom limb pain. Patients who undergo the amputation of a limb may continue to report sensations or chronic pain that seems to come from the limb that has been amputated. This may include feeling that the limb is still there, or it may be a sensation of chronic pain. Clearly, these sensations cannot actually come from the limb since it has been removed. The specificity theory cannot account for these findings since there is no ongoing tissue injury in the amputated limb, which would mean that there should be no chronic pain.⁷³



"The specificity theory cannot explain how hypnosis can be used for anesthesia during surgery. Under hypnosis, certain people can evidently undergo significant tissue damage from surgery without experiencing intense pain. This would support the notion that one's mental state or frame of mind can override the specificity theory. Similar examples of severe pain or chronic pain following relatively minor injuries can also be furnished."⁷⁴

Upon final analysis, the specificity theory was clearly erroneous because it implied that if there was no specific, identifiable injury to account for the pain then it could not exist. Therefore, patients who did complain of pain in those circumstances were often mistakenly diagnosed as being mentally ill.

Peripheral Pattern Theory

In 1955, DC Sinclair and G Weddell proposed the "peripheral pattern theory". This theory ignored a large body of strong evidence for receptor fiber specificity and contended that all skin fiber endings (with the exception of those innervating hair cells) are identical, and that pain is produced by intense stimulation of these fibers.⁷⁵

In 1953, Willem Noordenbos observed that a signal carried from the area of injury along large diameter "touch, pressure or vibration" fibers may inhibit the signal carried by the thinner "pain" fibers - the ratio of large fiber signal to thin fiber signal determining pain intensity; hence, we rub a smack. This observation was taken as a demonstration that pattern of stimulation (of large and thin fibers in this instance) modulates pain intensity.⁷⁶



Section 4

The Gate Control Theory Of Pain

Ronald Melzack and Patrick Wall introduced the gate control theory of pain in 1965. The gate control theory is based on several propositions:

- The transmission of impulses from the body into the central nervous system is “gated” (altered, changed, modulated) in the spinal cord.
- Gating is affected by the degree of activity in the large diameter and the small diameter nerve fibers. Impulses along the larger fibers tends to block pain transmission (close the gates) and more activity in the smaller fibers tends to facilitate transmission (open the gates).
- This gating mechanism in the spinal cord is affected by descending impulses from the brain.
- Large fibers may activate specific cognitive processes in the brain, which then influence the gate by descending impulse transmission.
- When the pain output to the brain reaches a certain level it activates the action system and the individual in pain takes an action to alleviate the pain.⁷⁷

Melzack and Wall theorized that the perception of physical pain is not a direct result of activation of pain receptor neurons, but instead is modulated by the interaction between different neurons. Melzack utilized these scientific and clinical observations to explain how pain is multidimensional and not just something that happens to the body, as Descartes’ mechanistic view of pain suggested. Melzack and Wall further proposed that pain is ‘gated’ or modulated by past experiences. They suggested that once the noxious agent that triggered the pain had ‘opened the pain gate’; other factors may contribute to the perpetuation of pain. Although their work drew on Descartes’ theory, they went further to claim that the brain and central nervous system played integral and active parts in receiving, modulating and transmitting pain impulses. As a result of their research, a bodily function for pain was proposed and chemical and surgical treatment methodologies were developed to help alleviate, or even completely remove patient complaints of pain.⁷⁸

The Physiology of the Gate Control Theory

Afferent pain-receptive nerves are responsible for bringing pain signals to the brain. These nerves are comprised of two kinds of fibers. The first fiber is a fast, thick, and myelinated "A-delta" fiber that carries messages quickly with intense pain. The second is a small, unmyelinated, slow "C" fiber that carries the longer-term throbbing and chronic pain signals. Large-diameter A-beta fibers are nonnociceptive (do not transmit pain stimuli) and inhibit the effects of firing by A-delta and C fibers. The peripheral nervous system has centers which regulate pain stimuli. Areas in the dorsal horn of the spinal cord that are involved in receiving pain stimuli from A-delta and C fibers, called laminae, also receive input from A-beta fibers. The nonnociceptive fibers indirectly inhibit the effects of the pain fibers, 'closing a gate' to the transmission of their stimuli. In other parts of the laminae, pain fibers also inhibit the effects of nonnociceptive fibers, 'opening the gate'.⁷⁹

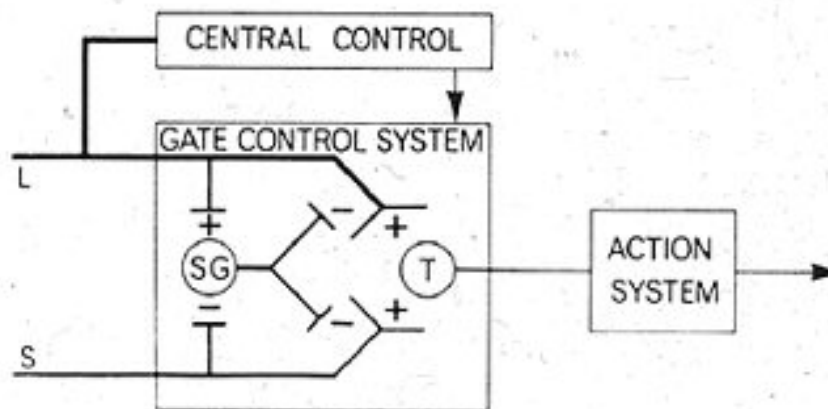
An inhibitory connection may exist with A-beta and C fibers, which may form a synapse on the same projection neuron. The same neurons may also form synapses with an inhibitory interneuron that also synapses on the projection neuron, reducing the chance that the latter will fire and transmit pain stimuli to the brain. The C fiber's synapse would inhibit the inhibitory interneuron, indirectly increasing the projection neuron's chance of firing. The A-beta fiber, on the other hand, forms an excitatory connection with the inhibitory interneuron, thus decreasing the projection neuron's chance of firing (like the C fiber, the A-beta fiber also has an excitatory

connection on the projection neuron itself). Therefore, depending on the relative rates of firing of C and A-beta fibers, the firing of the nonnociceptive fiber may inhibit the firing of the projection neuron and the transmission of pain stimuli.⁸⁰

The gate control theory explains how stimulus that activates only nonnociceptive nerves can inhibit pain. The pain seems to be lessened when the area is rubbed because activation of nonnociceptive fibers inhibits the firing of nociceptive ones in the laminae. In transcutaneous electrical stimulation (TENS), nonnociceptive fibers are selectively stimulated with electrodes in order to produce this effect and thereby lessen pain.⁸¹

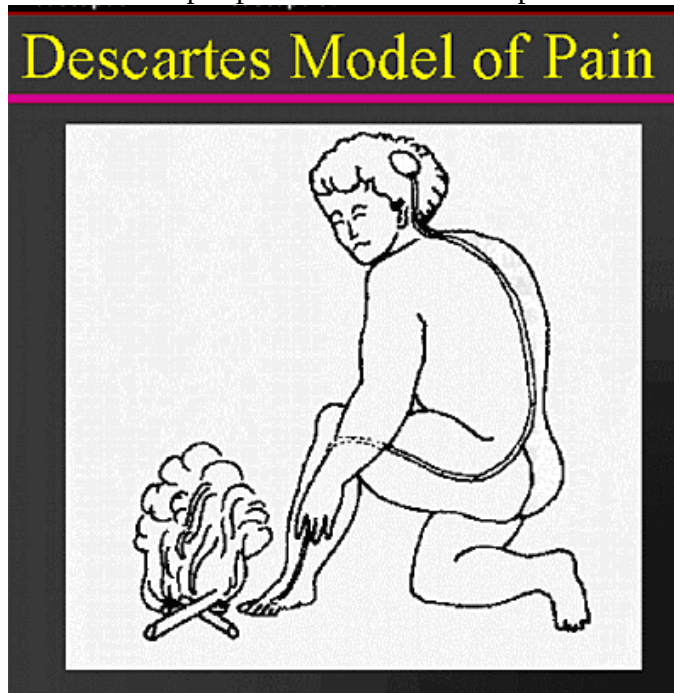
One area of the brain involved in the reduction of pain sensation is the periaqueductal gray matter that surrounds the third ventricle and the cerebral aqueduct of the ventricular system. Stimulation of this area produces analgesia by activating descending pathways that directly and indirectly inhibit nociceptors in the laminae of the spinal cord. It also activates opioid receptors, which are contained in parts of the spinal cord.⁸²

Afferent pathways interfere with each other constructively, so that the brain can control the degree of pain that is perceived, based on which pain stimuli are to be ignored to pursue potential gains. The brain determines which stimuli are best to ignore over time. Thus, the brain controls the perception of pain directly, and can be trained to turn off or lessen pain signals. This understanding led Melzack to point out that pain is in the brain and he opined that the brain should be the focus of the future of pain research.



The Gate Control Model. Large-diameter afferent fibers (L) stimulate the substantia gelatinosa (SG) and the transmission cells (T) in lamina 4. The SG cells reduce the membrane potential of afferent terminals, thus producing presynaptic inhibition. Small-diameter afferent fibers (S) also stimulate the transmission cells but inhibit SG cells and thus turn off the existing presynaptic inhibition.⁸³

Melzack illustrated his theory with Descartes' well-known picture "Boy with Foot in Fire" seen below. The treatment of pain by surgical transection of peripheral nerves and/or spinal cord was derived from this theory.



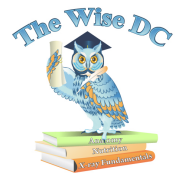
Descartes' *Boy with Foot in Fire*.

According to Melzack and Wall's gate control theory, large fiber afferents modulate the pain transmission of the small nociceptive fibers through a gateway. The substantia gelatinosa in the dorsal horn of the spinal cord was the proposed location of the gate that modulated the synaptic transmission of nerve impulses from peripheral fibers to central cells (refer to the figure below). This theory predated the advent of transcutaneous electrical nerve stimulation and spinal cord stimulation to modulate the transmission of pain in the spinal cord. Other methods of modulation include psychology and hypnosis.⁸⁴

Melzack's Neuromatrix Theory

In the 1990s, Melzack introduced a theory involving the integration of three separate pathways in the brain, with a concept called the "neurosignature". He proposed that a large number of interconnected neurons, called a "neuromatrix", exist in everyone. This neuromatrix analyzes the sensory information and gives perception of sensation. The neurosignature then comes into play and tells the brain that the perceptions of sensation are from the "self", in other words the neurosignature tells the brain that your arm is YOUR arm and not someone else's. Although his work is continually updated and revised, it continues to influence the research and management of pain.⁸⁵

Melzack's neuromatrix theory of pain posits that pain is a multidimensional experience produced by characteristic "neurosignature" patterns of nerve impulses that are generated by a widely distributed neural network -- the "body-self neuromatrix" -- in the brain. It is a parallel and serial process that can be thought of as



an expansion of the central control processes in the original gate control theory. The central control processes would encompass cognitive-evaluative, motivational-affective, and sensory-discriminative systems.⁸⁶

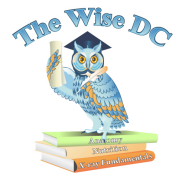
Multiple factors act on the neuromatrix and contribute to the output "neurosignature." These factors include sensory inputs; visual and other sensory inputs that influence cognitive interpretation; phasic and tonic cognitive and emotional inputs; intrinsic neural inhibitory modulation; and the activity of the stress-regulation system (endocrine, autonomic, immune, and opioid systems). Pain may be triggered by sensory inputs, but may also be generated independently of them. Therefore, pain could be produced by the output of a widely distributed neural network in the brain, rather than directly by nociceptive stimuli.⁸⁷

An example Dr. Melzack gave of the central process of pain is phantom limb pain in patients with spinal cord transection. In this scenario, the pathway for pain transmission has been severed, yet the patient feels pain in the insensate limb. He also presented the case of a woman who had the congenital absence of both legs and an arm, and felt phantom limb pain in the missing limbs. Phantom limb pain is commonly experienced by quadriplegics who have lost large portions of the spinal cord and cannot possibly be receiving any messages from it. This phenomenon is explained by Melzack's notion of the body-self neuro-matrix.. The sensation of a foot that is burning, for instance, has become separated from the foot that is still attached to a useless leg dangling into space - the disconnection actually frees the neurological part of the matrix to "invent" a body. Different parts of the brain then perceive the whole body in different ways, and these are more free to clash if real signals from a real body are not disciplining them all with a common external experience.⁸⁸

At present, pain theorists continue to explore the genetic, endocrine, and immune systems, all of which may contribute to the neuromatrix. Pain theorists have offered explanations of pain that entail both physiological and psychological components. The neuromatrix theory is still evolving and the brain functions and mechanisms in this theory need to continue to be further explored and scrutinized.⁸⁹

Pain Theory Experimentation

Research records indicate that pain experimentation was performed on dogs that were raised in confined environments. When the dogs were released, they were excited, constantly ran around, and required several attempts to learn to avoid pain. When a pain stimulus such as a pinch was introduced, the animals did not take action to avoid the stimulus immediately. This finding seemed to demonstrate that pain is understood and avoided only by experience. Consequently, these experimental observations led to the thought that aversion to pain is not inherent or automatic, and the organism has no way to know what will cause repeated pain without a repeated experience.⁹⁰



Advantages of the Gate Control Theory

Prior to Melzack and Wall's gate control theory, neurochemistry theorists had not taken into account the brain in the discussion of pain. Based upon the classic theory of pain proposed by René Descartes, pain was thought to be simply a direct response to a stimulus. This one-way 'alarm system' pain-pleasure theory was incapable of offering a scientific explanation for phantom limb pain where the pain signal is in fact impossible to receive in the absence of a neurological pain pathway. Conversely, the gate control theory in general, and the neuromatrix theory in particular, provide an explanation for the phantom limb pain phenomenon. Unlike the specificity theory of pain, the gate control theory allows for the dynamic role of the brain in the pain process. Psychological aspects of pain are now seen as integral parts of the process and not just reactions to pain. This has opened the way for psychological approaches to be used for pain treatment and management. Simplistic treatments such as cutting nerve pathways (which often made the pain worse) have been replaced by more realistic and efficacious treatment methodologies.

Consequences of Melzack's Work

In his paper *The Tragedy of Needless Pain*, Melzack further asserts that "pain is a fundamental human experience, and requires an integrative understanding of that whole experience, and every choice we have made, that has formed our own 'gates'. He frames the choice to deal with pain or ignore it as moral: if the brain *can* control pain, we who know that *must* make use of that capacity, and in turn take control of pain on a species level - only by doing so can we achieve control of the larger causes of all of the pain that humans cause each other by carelessness, hatred, and failures of empathy - which might extend beyond humans."⁹¹

Melzack's work has led to advancements in the assessment of pain. Melzack was a professor at McGill University and thus the name of his McGill Pain Questionnaire. According to Melzack pain is an entirely personal experience, and therefore it is difficult to accurately measure. Melzack's McGill Pain Questionnaire asks a number of directed questions to assess and categorize the experience of pain. Melzack collected approximately 200 words used by his patients to describe pain. A "burning pain" for instance, can be described as "hot" or even "searing". "Throbbing" becomes "palpitating". Some describe sensory aspects of pain, and others, such as "excruciating", describe emotional experiences. More intense pain generally requires more words to describe. A patient, in the questionnaire, picks one of 20 words to describe their pain, and assign it a point on a scale. Melzack's work was the first to actually qualify and define types of pain - something impossible prior to the gate control theory. An advantage of this was to make it easier to determine the difference between organic pain and non-organic pain - the latter being entirely treatable by psychological means.⁹²

Section 5

Anatomy and Physiology of Pain Pathways

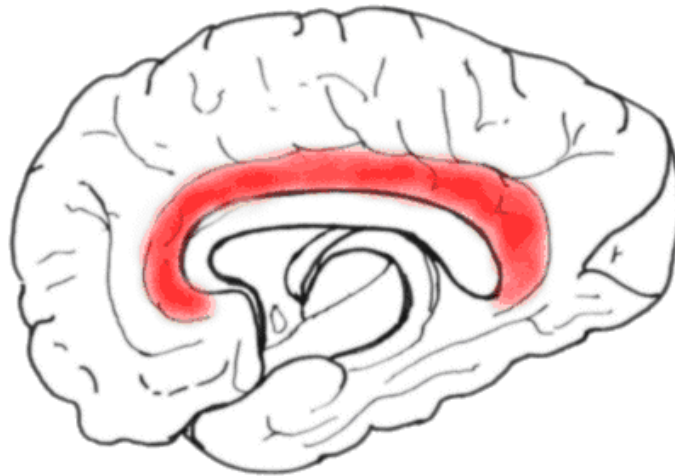
The Cortex of the Brain

The brain is devoid of nociceptive tissue, and therefore cannot experience pain. Thus, a headache is not due to stimulation of pain fibers in the brain itself. Rather, the dura mater membrane surrounding the brain and spinal cord is innervated with pain receptors, and stimulation of these dural nociceptors is thought to be involved to some extent in producing headache pain. Vasoconstriction of peripheral vessels is also another common cause of headache.

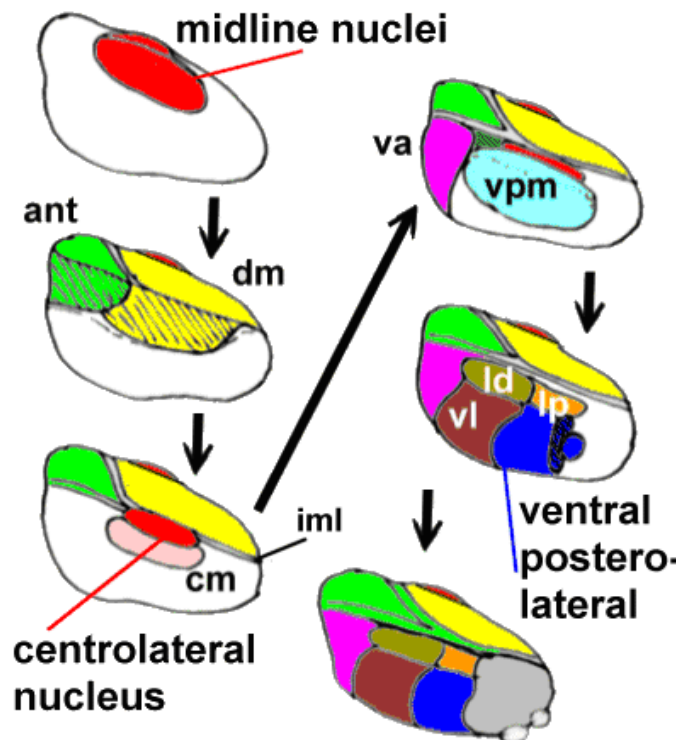
Research suggests that cortical structures are involved in the perception of pain. Connections that link higher cortical structures with pain-centered nuclei in the thalamus and brainstem include the following:

- The primary sensory cortex, S I .
- The secondary sensory cortex, S II.
- The anterior part of the insula .
- The cingulate gyrus.

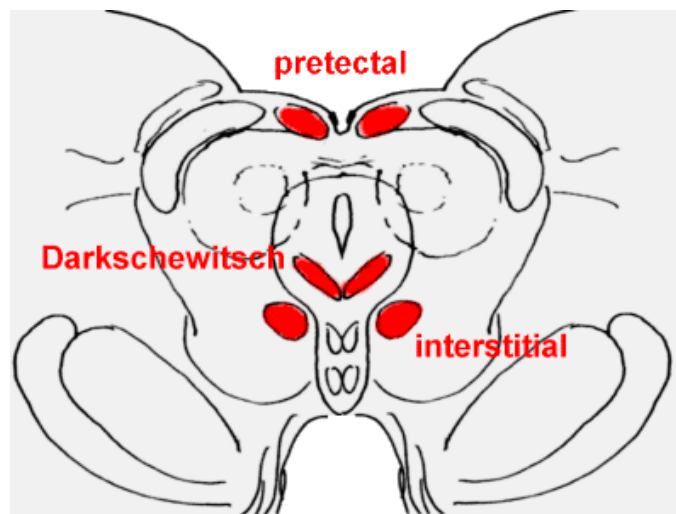
S I is concerned with the localization of pain, while the other three structures are thought to be concerned with the motivational-affective aspects of pain.



The human brain as seen from the side, showing the SI and SII sensory cortex. SI is a thin strip made up of Brodman areas 3,1 and 2 posterior to the central sulcus, while SII is found just above the lateral sulcus.⁹³

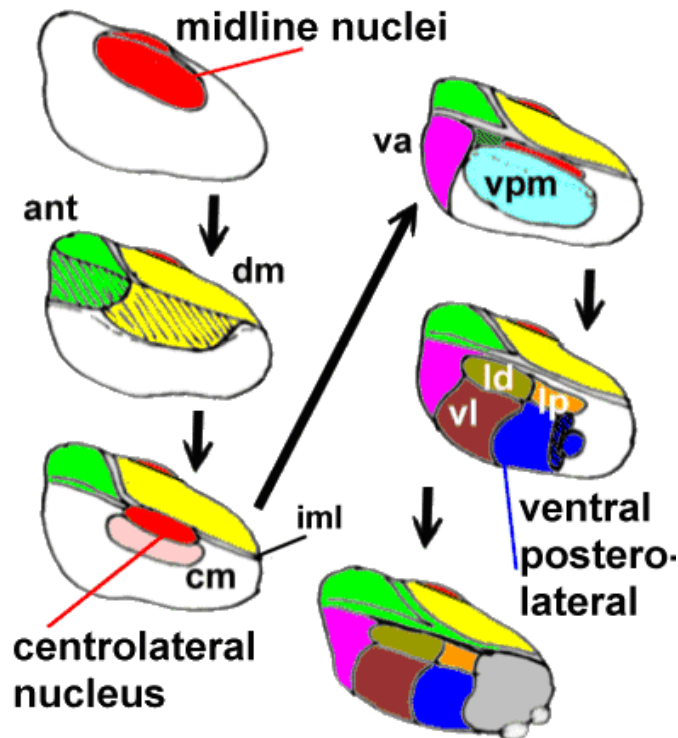


Retracted temporal lobe revealing the insula.
(the anterior portion is thought to be concerned with pain perception.)⁹⁴



The picture above illustrates the medial aspect of a hemisphere, showing the cingulate gyrus.
The anterior part of the cingulate gyrus is important in the perception of 'affective' pain.

Thalamic Nuclei



The Thalamus

The thalamus has multiple nuclei that are involved with the perception of pain. The lateral nuclei deal mainly with sensory/discriminative aspects, while the medial nuclei deal with 'affective' pain. Medially is the midline nuclei. Lateral to the midline nuclei is the dorsomedial nucleus(dm) and, more anteriorly the anterior nuclei(ant) . Even further laterally we have the lateral dorsal(ld), lateral posterior(lp), ventral lateral(vl) and ventral posterolateral nuclei. The pulvinar (grey) is situated posteriorly

The internal medullary lamina bounds the dorsomedial nucleus laterally, and separates it from the anterior nuclei. Within the internal medullary lamina are the intralaminar nuclei, including the centromedian (cm) and centrolateral nuclei. Lateral to the internal medullary lamina (iml) lies the ventral posteromedial nucleus (vpm) and anterior to this is the ventral anterior nucleus (va).

A Diagram of the Thalamic nuclei. The lateral thalamus is thought to be mainly concerned with 'discriminative' pain, the medial with "affect/motivation".⁹⁵

The midline nuclei include the:

- nucleus reuniens
- rhomboidal nucleus
- submedius nucleus

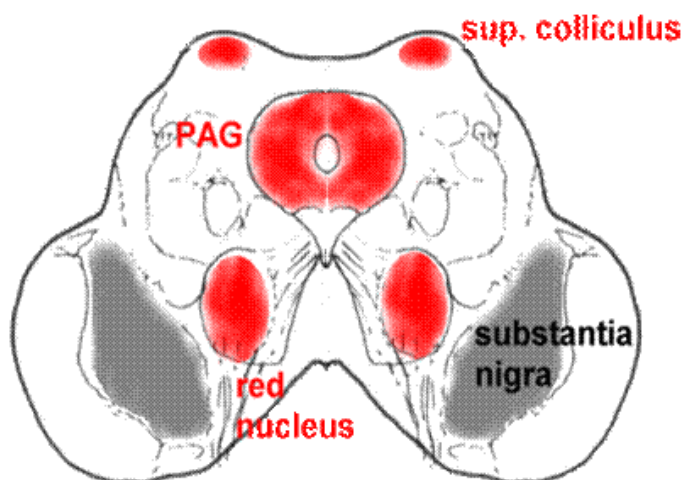
The submedius nucleus has been found to play a significant role in pain perception.

Midbrain

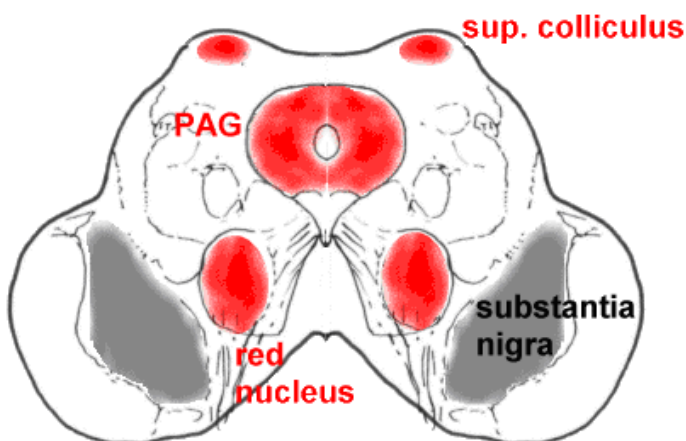
There is a host of pain-related structures in the midbrain. The midbrain is involved in 'affective' pain, with extensive connections to the reticular system of the brainstem.

Important components are:

- The peri-aqueductal grey matter (PAG)
- Deep layers of the superior colliculus
- The red nucleus
- The pre-tectal nuclei (anterior and posterior)
- The nucleus of Darkschewitsch
- the interstitial nucleus of Cajal
- The intercolliculus nucleus, nucleus cuneiformis and the Edinger-Westphal nucleus.



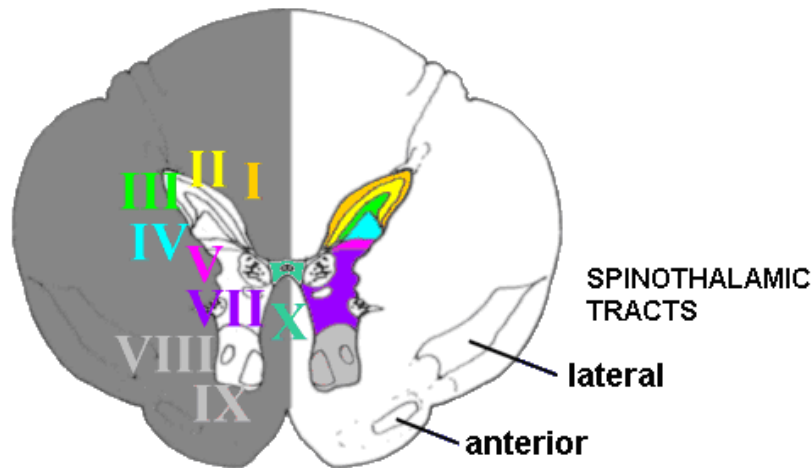
Rostral midbrain, with the anterior pretectal nucleus, interstitial nucleus of Cajal, and nucleus of Darkschewitsch in red.



A caudal section through the midbrain, showing the superior colliculus (SC), periaqueductal grey matter (PAG), and red nucleus (RN).⁹⁶

The Pons

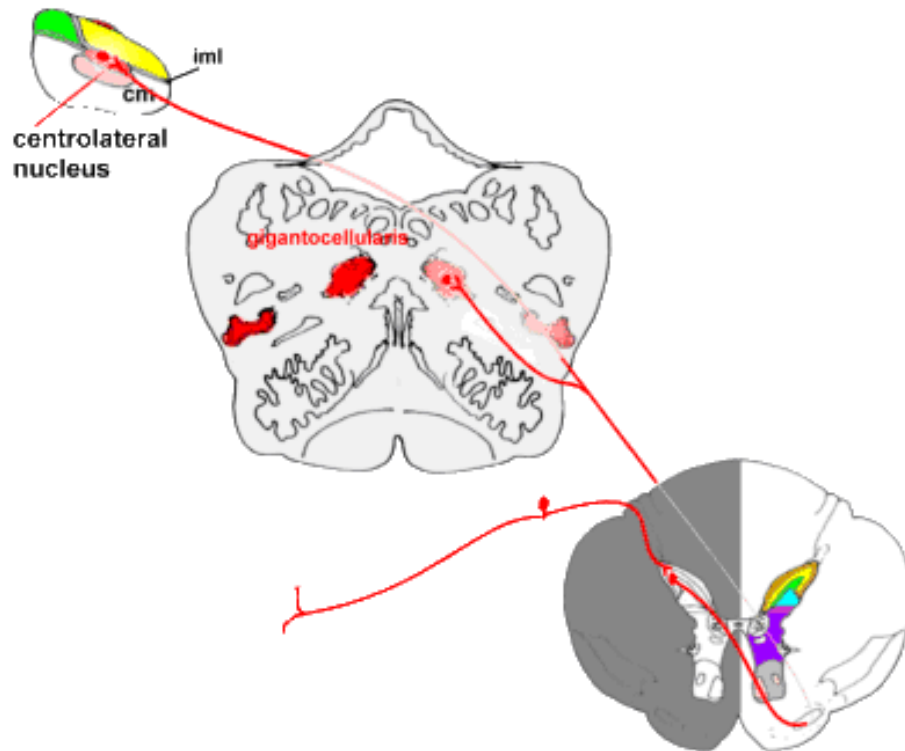
The most important pain-related nucleus in the pons is the locus coeruleus. This structure has noradrenaline-containing neurons, and projects to a variety of brainstem structures that modulate pain through pathways that descend to the spinal cord. Also, the parabrachial nuclei receive a vast number of ascending spinothalamic fibers. Both are shown in the following cross-section through the pons.



A cross-section through the pons, showing the locus coeruleus (LC) and the lateral parabrachial nucleus (PB) ⁹⁷

The Medulla

The medulla is involved in the motivational/affective aspects of pain. Important cell groups are the nucleus gigantocellularis and related nuclei, the lateral reticular nucleus, and a variety of other nuclei. The raphe nuclei near the midline are important in the descending pathways that suppress pain.



The rostral medulla, with the nucleus gigantocellularis and lateral reticular nucleus ⁹⁸

Section 6

An Overview of How The Brain Nuclei Function Together

'Discriminative' fibers ascend from the spinal cord to the lateral thalamus and then to the primary sensory cortex (S I). These are called the neospinothalamic fibers.

'Affective' fibers that follow a similar course from the spinal cord, but end up in the reticular formation of the hindbrain. These spino-reticulodiencephalic fibers have extensive connections throughout the brainstem, and from there project to the medial thalamus and the cortex (S II).

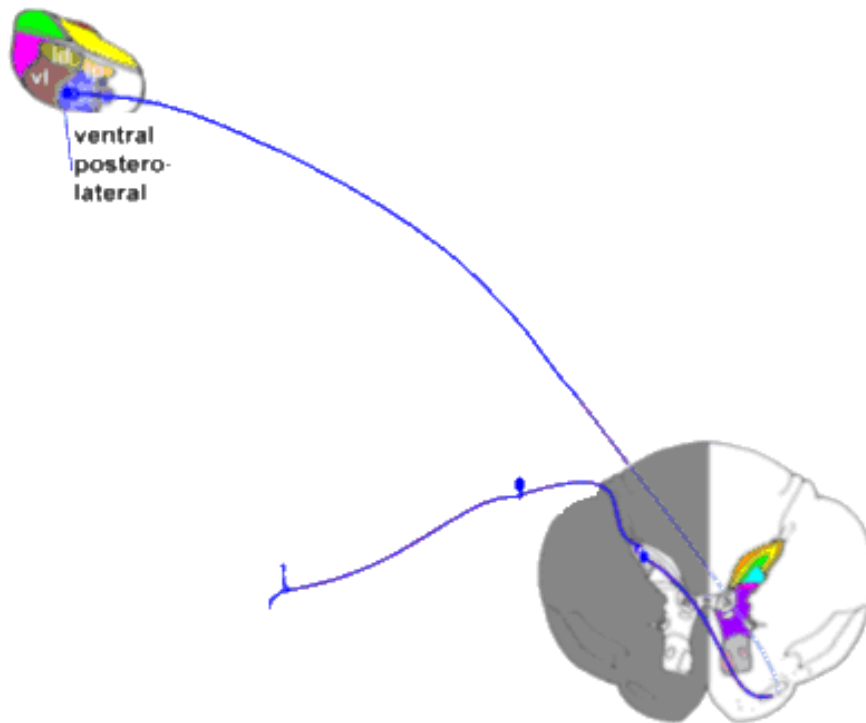
Descending fibers pass down from the brainstem to the spinal cord inhibiting incoming sensations of pain.

Many of these descending fibers originate in the locus coeruleus, others in the raphe nuclei.

The Spinal Cord

Traditionally it was thought that most pain fibers entered the dorsal root of the spinal cord (the "sensory" root) and then synapsed in the dorsal part of the spinal grey matter, before passing the message up through the spinothalamic tract. However, research suggests that up to 40% of sensory fibers enter in the ventral root.⁹⁹

Histologically the grey matter of the spinal cord is divided into ten 'laminae'. The dorsal part is divided into five laminae (I to V), components of which deal with most incoming pain fibers. VII is in between these laminae and the more ventral laminae VIII and IX, and X refers to the grey matter around the central canal of the spinal cord. And what about lamina VI? This is only discernible in the bulges in the cord related to where the innervation of the limbs originates.



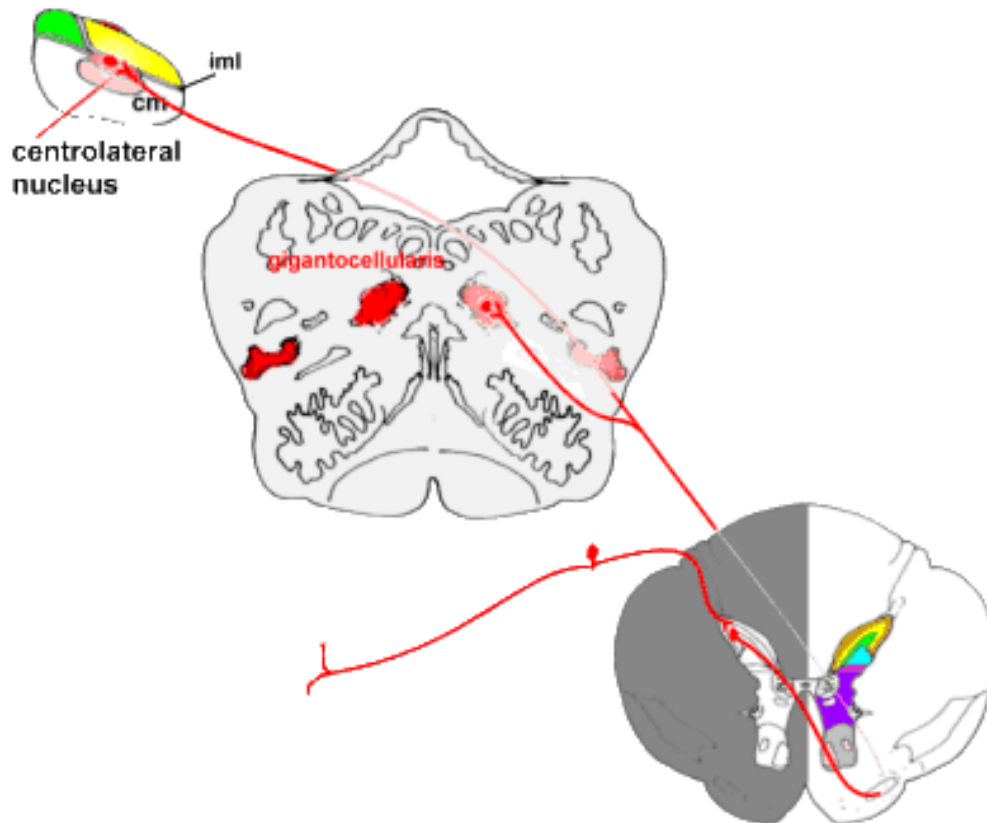
A transverse section through the thoracic spinal cord illustrating the grey matter and various laminae. Important fibers coming from the periphery into the dorsal horn include:

- Tiny unmyelinated 'C' fibers that are important carriers of long-lasting burning pain. These fibers terminate in lamina II.
- Thin myelinated 'A delta' fibers, concerned with more accurate localization of pain, and terminating mostly laterally in laminae I and V.
- Thick 'A beta' fibers that carry information about vibration and position sense from the periphery to the cord.

Unpleasant stimuli entering via the C fibers can be suppressed by concurrent stimulation of A delta fibers (high amplitude low frequency stimulation, for example by acupuncture) or by impulses passing through A beta fibers. Examples of the latter include TENS (transcutaneous electrical nerve stimulation) or by rubbing the skin.

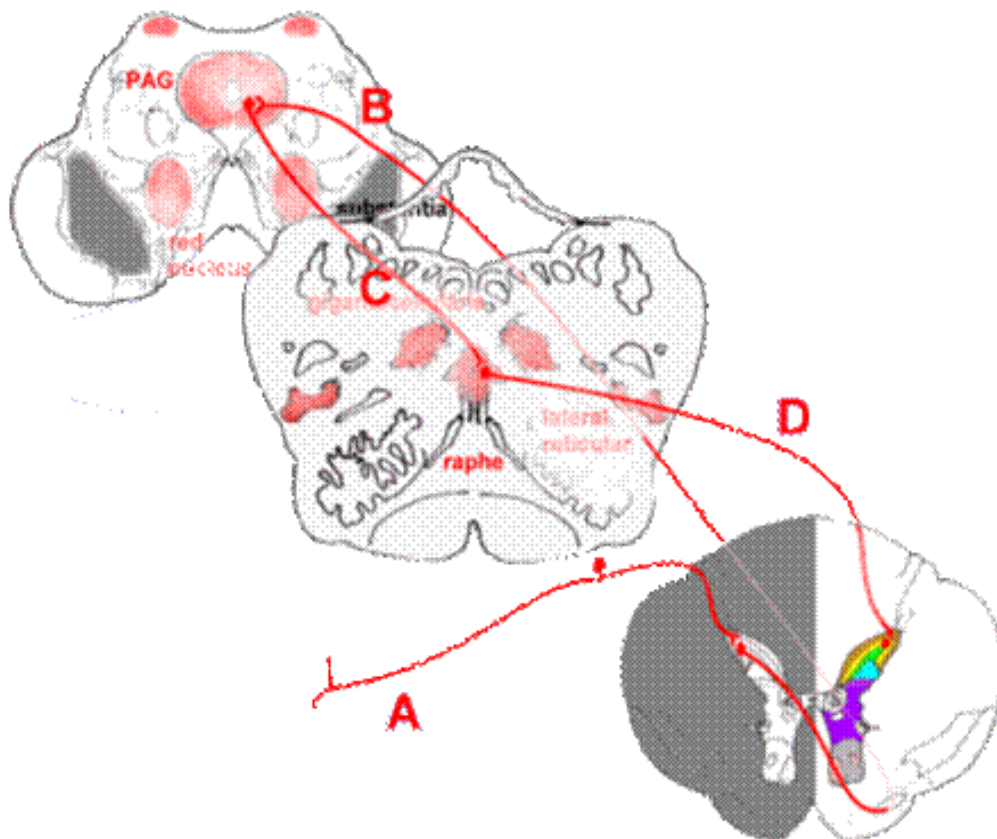
Ascending Pain Connections

Spino-reticulo-diencephalic connections:



Pathways from the spinal cord to the brainstem, and from there to the thalamus (diencephalon). Some fibers pass directly to the medial thalamus, while others end in (or send collaterals to) a variety of nuclei in the brainstem.

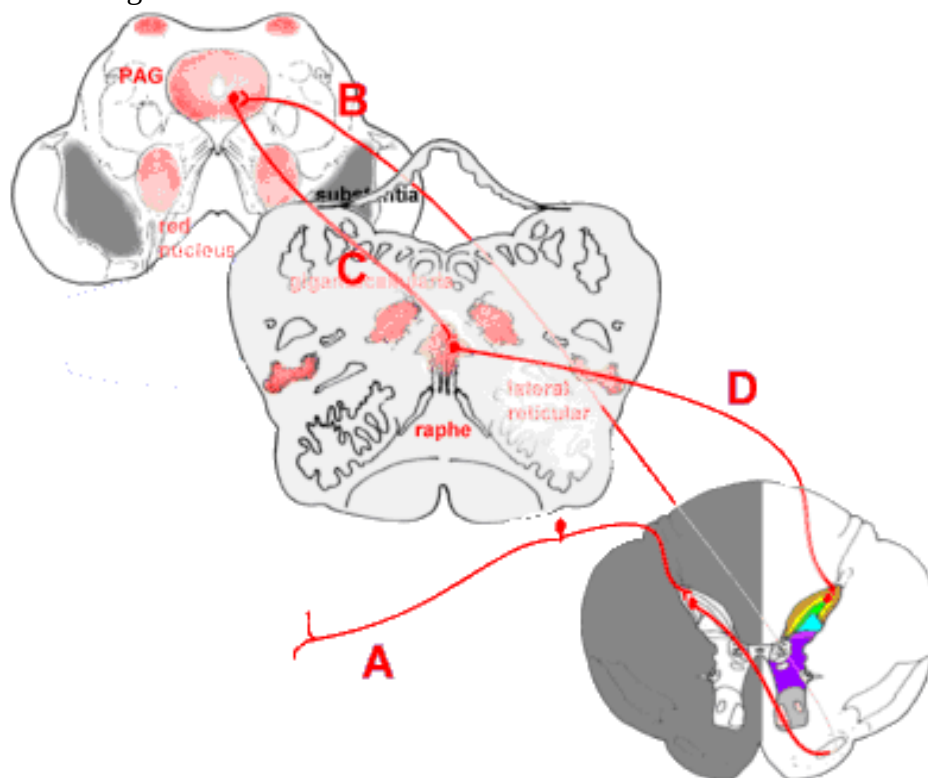
Pathways from cord to lateral thalamus and then on to the primary sensory cortex(SI) are discriminative pain pathways, and have little to do with perception of pain as a 'sore' stimulus. These pathways have few or no opioid receptors. Consequently, the use of morphine medication therapy would have no effect on such pathways.



The neospinothalamic tract.

Descending Pain Connections

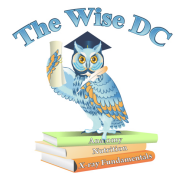
Fibers that descend from the brainstem to the spinal cord act to modulate incoming pain signals. Notable neurotransmitters mediating this anti-nociceptive effect include noradrenaline (norepinephrine), especially in the locus coeruleus, and serotonin in the raphe nuclei. Opioid receptors are abundant in the descending pain connection. Some descending connections are:



Descending connections that modulate incoming pain impulses.

Incoming pain stimuli are transmitted (A) to the dorsal horn, and from there (B) to the periaqueductal grey (PAG). Descending impulses pass (C) to the raphe nuclei, especially the nucleus raphe magnus, in the upper medulla, and thence back to the dorsal horn via reticulospinal fibers (D).

The above shows only the serotonergic descending fibers. Other pain-suppressing impulses pass from the PAG to the locus coeruleus, and from there to the dorsal horn.¹⁰⁰



Section 7

An Overview of Neurotransmitters

Numerous neurotransmitters mediate the transmission of the sensations of pain in both the brain and the spinal cord. Neurotransmitters can be categorized into the following groups:

Excitatory neurotransmitters to include:

- Glutamate
- Tachykinins
- Agents that act at the various neurokinin receptors, such as substance P ('P is for pain'), neurokinin A and neurokinin B.
- Calcitonin gene-related peptide
- Vasoactive intestinal polypeptide
- Somatostatin

Inhibitory neurotransmitters to include: Gamma Amino Butyric Acid (GABA). Over forty percent of inhibition in the human central nervous system is GABAergic.

Neurotransmitters involved in descending pain regulation include:

- Noradrenaline (norepinephrine)
- Serotonin.

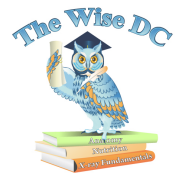
Further Analysis of the Major Neurotransmitters

Glutamate

The three major glutamate receptors are "NMDA", "AMPA" and the metabotropic receptor. "The NMDA receptor mediates a host of spinal responses to severe painful stimulation, but there are several catches to understanding how it works. Normally, the receptor is inactive as it is physiologically choked by a magnesium ion sitting in its ion channel. In order for this ion to be removed, adjacent peptide receptors have to be stimulated - the Mg^{++} then pops off, and an emphatic painful response occurs. Neurophysiologists have known about this phenomenon for ages, gracing it with the label "wind-up" - as the frequency of C-fibre stimulation increases there is a dramatic and long-lasting central response, with some populations of spinal neurones becoming more and more sensitive to stimulation. Consequences of glutamate receptor activation include production of c-fos and spinal production of prostanoids and the ubiquitous Dr NO, nitric oxide. Unfortunately all this knowledge benefits clinicians surprisingly little, as drugs that antagonise the effect of glutamate at the NMDA receptor tend to induce psychosis in humans, but the combination of low dose NMDA antagonists with opioids may be supra-additive with fewer side effects." ¹⁰¹

GABA

GABA is found throughout the brain and spinal cord and, it has major pain inhibitory effects. Interneurons in laminae I, II and III are GABA-rich, and mediate gate control in the dorsal horn by synapsing on neurones that contain substance P. ¹⁰²



Tachykinins

Neurokinin receptors mediate pain in the spinal cord. Substance P binds to the NK-1 receptor while neurokinins A and B bind respectively to the NK-2 and NK-3 receptors. Collectively these substances are known as 'tachykinins'. The tachykinin receptors are G-protein coupled, and increase intracellular calcium levels, triggering gene transcription.”¹⁰³

Analysis Of Pain At The Cellular Level

Arguably, the most significant discovery *ever* in the field of pain has been the gene c-fos. This gene and its cellular product, the protein called *Fos* are crucial to the central nervous system changes that occur when a person feels pain. Central nervous system c-fos expression correlates extremely well with painful stimulation. Fos is one of the **inducible transcription factors** (ITFs) that controls mammalian gene expression.

Since c-fos is a proto-oncogene promote vast intracellular changes including cellular restructuring and proliferation and is thought to be involved in the long-term neurological consequences of noxious stimulation. Genetic research suggests that noxious peripheral stimulation causes Fos to appear in the spinal cord. Brief stimulation for as little as 10 minutes causes ITFs to appear within 30 minutes, peak at one to two hours, and disappear within about eight hours. Prolonged stimulation causes a many-fold increase in ITF expression, and substantially prolongs expression. Nociceptive C-fiber stimulation seems to be the main stimulus for ITF production in the spinal cord(c-fos appears within an hour in laminae I and II, the parasympathetic column, the dorsal grey commissure, as well as in the hindbrain). The reuniens, rhomboid and submedius nuclei all express c-fos, as do a number of areas in the cortex, thalamus, hypothalamus, amygdale and the parabrachial nucleus.

With prolonged stimulation, c-fos disappears from spinal neurons after two to seven days. This disappearance is despite increased neuronal excitability and a marked increase in expression of neurokinin and glutamate receptors, and may be simply because the neuronal changes are fixed, so the ITF is no longer needed. However, chronic lesions of sensory nerves, such as a partial sciatic nerve ligation can induce chronic c-fos expression even in nerves which don't normally express the ITF.

In conclusion, c-fos plays a vital role in cell replication and differentiation.¹⁰⁴



Section 8

Types of Physiological Pain

- **Cutanenous pain:** Caused by injury to the skin or superficial tissues. The pain-detecting neurons are just below the skin where there is a high concentration of nerve endings. The pain is localized and short-term. Examples: paper cut, first-degree burn, exterior wound.
- **Somatic pain:** Comes from ligaments, tendons, bones, blood vessels and nerves. There are few somatic pain receptors in these areas, so the pain is a dull pain of longer duration. Examples: sprains, and fractured bones.
- **Visceral pain:** Visceral neurons are within body organs and cavities. Pain receptors in these areas are very diffuse, so pain is felt as an ache over a longer period of time. Visceral pain is difficult to localize, and is often called "referred pain." This means the sensation is unrelated to the injury site. For example, myocardial ischemia can be felt in the upper chest, or as an ache in the left shoulder, arm or hand.
- **Neuropathic Pain:** Caused by injury or disease of the nerve tissue. This can disrupt the sensory nerves from transmitting correct information to the thalamus.

Phantom Pain

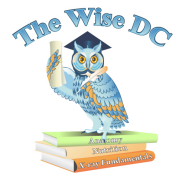
Phantom pain is pain from a part of the body that has been lost or from which the brain no longer receives signals. It is a type of neuropathic pain. Phantom limb pain is a common experience of amputees.

“The prevalence of phantom pain in upper limb amputees is nearly 82%, and in lower limb amputees is 54%.¹⁰⁵ Phantom pain and phantom sensations in upper limb amputees: an epidemiological study.¹⁰⁶ One study found that eight days after amputation, 72 percent of patients had phantom limb pain, and six months later, 65 percent reported it.^{107,108}

Some amputees experience continuous pain that varies in intensity or quality; others experience several bouts a day, or it may occur only once every week or two. It is often described as shooting, crushing, burning or cramping. If the pain is continuous for a long period, parts of the intact body may become sensitized, so that touching them evokes pain in the phantom limb, or phantom limb pain may accompany urination or defecation.”¹⁰⁹

Treatment of phantom pain includes local anesthetic injections into the nerves or sensitive areas of the stump which may relieve pain for days, weeks or, sometimes permanently, despite the drug wearing off in a matter of hours; vigorous vibration or electrical stimulation of the stump, or current from electrodes surgically implanted onto the spinal cord; and movement and touch in a phantom limb which in turn cause a reduction in pain.¹¹⁰

“Paraplegia, the loss of sensation and voluntary motor control after serious spinal cord damage, may be accompanied by girdle pain at the level of the spinal cord damage, visceral pain evoked by a filling bladder or bowel, or, in five to ten per cent of paraplegics, phantom body pain in areas of complete sensory loss. This phantom body pain is initially described as burning or tingling but may evolve into severe crushing or pinching pain, fire running down the legs, or a knife twisting in the flesh. Onset may be immediate or may not occur until years after the disabling injury. Surgical treatment rarely provides lasting relief.”¹¹¹



Psychogenic Pain

“Psychogenic pain, also called *psychalgia* or *somatoform pain*, is pain caused, increased, or prolonged by mental, emotional, or behavioral factors. Headache, back pain, and stomach pain are sometimes diagnosed as psychogenic. Sufferers are often stigmatized, because both medical professionals and the general public tend to think that pain from a psychological source is not "real". However, specialists consider that it is no less actual or hurtful than pain from any other source.”¹¹²

Studies suggest that chronic pain sufferers frequently display psychological disturbance, with elevated scores on the Minnesota Multiphasic Personality Inventory scales of hysteria, depression and hypochondriasis (the "neurotic triad"). Additionally, it was noted that when long term pain is relieved by therapeutic intervention, scores on the neurotic triad and anxiety fall, often to normal levels. Self-esteem, often low in chronic pain patients, also shows improvement once pain has resolved.¹¹³

“The term 'psychogenic' assumes that medical diagnosis is so perfect that all organic causes of pain can be detected; regrettably, we are far from such infallibility... All too often, the diagnosis of neurosis as the cause of pain hides our ignorance of many aspects of pain medicine.”¹¹⁴

Types of Psychogenic Pain

Headaches, muscle pains, back pain, and stomach pains are some of the most common types of psychogenic pain.

Psychogenic Pain Diagnosis

The diagnosis of psychogenic pain is made when other causes of pain are ruled out. A person with a pain disorder will complain of pain that does not match his or her symptoms. Medical doctors and mental health specialists working together are often most helpful to those with this disorder.

Psychogenic Pain Treatment

Treatment for psychogenic pain may include:

- Psychotherapy
- Individuals with more social support experience less cancer pain, take less pain medication, report less labor pain and are less likely to use epidural anesthesia during childbirth or suffer from chest pain after coronary artery bypass surgery.¹¹⁵
- Suggestion can significantly affect pain intensity. About 35% of people report marked relief after receiving a saline injection they believe to have been morphine. This "placebo" effect is more pronounced in people who are prone to anxiety, so anxiety reduction may account for some of the effect, but it does not account for all of the effect. Placebos are more effective in intense pain than mild pain; and they produce progressively weaker effects with repeated administration.¹¹⁶

“**Cognitive behavioral therapy (CBT)** is a psychotherapeutic approach: a talking therapy. CBT aims to solve problems concerning dysfunctional emotions, behaviors and cognitions through a goal-oriented, systematic procedure in the present. The title is used in diverse ways to designate behavior therapy, cognitive therapy, and to refer to therapy based upon a combination of basic behavioral and cognitive research.

CBT has been identified as a treatment of choice for a number of mental health difficulties, including post-traumatic stress disorder, OCD, bulimia nervosa, and clinical depression. There is empirical evidence that CBT



is effective for the treatment of a variety of problems, including mood, anxiety, personality, eating, substance abuse, and psychotic disorders.

- Antidepressants
- Non-narcotic painkillers ¹¹⁷



Section 9

Comparison of Nociceptive and Neuropathic Pain

Nociceptive Pain

Nociceptive pain results from tissue damage. Intact neurons report damage, and pain is experienced. Nociceptive pain can be subdivided into somatic and visceral pain. Nociceptive pain can be experienced as sharp, dull, or aching. There may be radiation of the pain, especially visceral pain, but it will not be in a direct nerve distribution. For example, gallbladder pain can radiate to the scapula. Nociceptive pain is generally responsive to NSAIDs (nonsteroidal anti-inflammatory drugs) and opioids. Conditions associated with inflammation, bone pain, and joint disease are particularly responsive to NSAIDs.

Neuropathic Pain

Neuropathic pain may occur when there is either damage to or dysfunction of nerves in the peripheral or central nervous system. Examples of neuropathic pain include diabetic neuropathy and trigeminal neuralgia. Neuropathic pain frequently coexists with nociceptive pain. Examples include trauma that damages both tissue and nerves, such as severe burns that burn skin as well as damage nerve endings and a lumbar disc herniation that results in tissue pain along the distribution of the sciatic nerve..

Neuropathic pain is often described as having a burning or electrical quality. It may feel like a shock or lightning bolt. Sometimes stimuli that usually do not cause pain, such as light touch, may elicit a paroxysm of pain. A light stroke of the cheek that results in the sudden pain of trigeminal neuralgia is an example of this type of pain. Sometimes patients do not describe the sensation as being "painful" but rather as feeling unpleasant, strange or tingly. This feeling is called a dysesthesia.

Neuropathic pain in the peripheral nervous system frequently follows a nerve distribution. This distribution may replicate a particular nerve, as in sciatic pain or trigeminal neuralgia, or may represent the distribution of terminal nerve endings, as in the stocking-glove distribution of peripheral neuropathies.

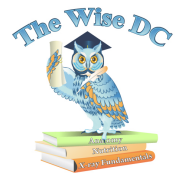
Neuropathic pain is relatively resistant to NSAIDs and opioids, although they may be helpful in certain cases. The other major classes of medications that are prescribed for neuropathic pain include tricyclic antidepressants, anticonvulsants, and sodium channel blockers.¹¹⁸

Analysis of Somatic, Visceral and Neuropathic Pain

Somatic, visceral and neuropathic pain can all be either acute or chronic in nature. Somatic, visceral, and neuropathic pain can all be felt at the same time or singly and at different times. Most cancer patients experience both somatic and visceral pain. Only about 15-20% of all cancer patients report neuropathic pain. The different types of pain respond differently to the various pain management therapies. Somatic and visceral pain are both easier to manage than neuropathic pain.¹¹⁹

Somatic Pain

Somatic pain is caused by the activation of pain receptors in either the cutaneous musculoskeletal tissues. When it occurs in the musculoskeletal tissues, it is called deep somatic pain. Common causes of somatic cancer pain include metastasis in the bone (an example of deep somatic pain) and postsurgical pain from a surgical incision



(an example of surface pain). Deep somatic pain is usually described as dull or aching but localized. Surface somatic pain is usually sharper and may have a burning or pricking quality.¹²⁰

Visceral Pain

Visceral pain is caused by activation of pain receptors resulting from infiltration, compression, extension, or stretching of the thoracic, abdominal, or pelvic viscera. Common causes of visceral pain include pancreatic cancer and metastases in the abdomen. Visceral pain is not well localized and is usually described as pressure-like, deep squeezing.¹²¹

Neuropathic Pain

Neuropathic pain is caused by injury to the nervous system either as a result of a tumor compressing nerves or the spinal cord, or cancer actually infiltrating the nerves or spinal cord. It also results from chemical damage to the nervous system that may be caused by cancer treatment (chemotherapy, radiation, surgery). This type of pain is severe and usually described as burning or tingling. Tumors that lie close to neural structures are believed to cause the most severe pain that cancer patients feel.

Acute versus Chronic Pain Signs and Symptoms

Acute Pain

Acute pain begins suddenly and is usually sharp in quality. It serves as a warning of disease or a threat to the body. Acute pain might be caused by a traumatic event or its etiology and onset can be insidious in nature. Acute pain might be mild and last just a moment, or several days, increasing in intensity over time (subacute pain), or it can occur intermittently. Acute pain can also be severe and last for weeks or months. In most cases, acute pain does not last longer than three months, and it disappears when the underlying cause of pain has been treated or has healed.

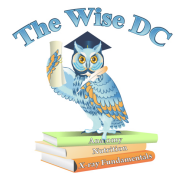
The longer acute pain persists the more susceptible it is to other influences and developing into a chronic pain problem. These influences include such things as the ongoing pain signal input to the nervous system even without tissue damage, the increased potential for physical deconditioning, and psychological manifestations of depression and anxiety.

Sub Acute Pain: Subacute pain is a subset of acute pain: it is pain that has been present for at least six weeks but less than three months (van Tulder et al. 1997).

Chronic Pain

Chronic pain is defined as pain lasting for more than 3 months. It is much more subjective and not as easily described as acute pain. Chronic pain persists despite the fact that the injury has healed. Pain signals remain active in the nervous system for weeks, months, or years. Physical effects include increased muscle tone, decreased ranges of motion, lethargy, and changes in appetite. Emotional effects include depression, anger, anxiety, and fear of re-injury. Common chronic pain complaints include:

- Headache
- Low back pain
- Cancer pain
- Arthritis pain
- Neurogenic pain
- Psychogenic pain



Chronic pain might have originated with an initial trauma/injury or infection, or there might be an ongoing cause of pain. However, some people suffer chronic pain in the absence of any past injury or evidence of body damage.

Effectively treating chronic pain poses a great challenge for physicians. This kind of pain usually affects a person's life in many ways. It can change someone's personality, ability to function, and quality of life.¹²²

Types of Chronic Pain Scenarios

There are two different types of chronic pain scenarios - chronic pain due to an identifiable pain generator (e.g. an injury), and chronic pain with no identifiable pain generator (e.g. the injury has healed).

Chronic pain due to an identifiable pain generator

This type of chronic pain is due to a clearly identifiable cause. Certain structural spine conditions (for example, degenerative disc disease, spinal stenosis and spondylolisthesis) can cause ongoing pain until successfully treated. These conditions are due to a diagnosable anatomical problem.

Chronic pain with no identifiable pain generator

This type of pain continues beyond the point of tissue healing and there is no clearly identifiable pain generator that explains the pain. It is often termed “chronic benign pain”.

Pain can set up a pathway in the nervous system and, in some cases, this becomes the problem in and of itself. In chronic pain the dysfunctioning nervous system sends a pain signal even though there is no ongoing tissue damage.

The term “chronic pain” is generally used to describe pain that lasts more than three months, or beyond the point of tissue healing. Chronic pain is usually less directly related to identifiable tissue damage and structural problems. Examples of chronic pain are: chronic back pain without a clearly determined cause, failed back surgery syndrome, and fibromyalgia.

Causes of Pain By Region

Visceral pain sensation is often referred by the CNS to a dermatome region which is be far away from the originating organ. These correlate to the position of the organ in the embryo. Examples of this include the heart which originates in the neck, thus producing the classical neck and arm pain experienced during acute cardiac pain.

Head and Neck

Jaw - Temporal arteritis, trauma .

Ear - otitis media, otitis externa, trauma.

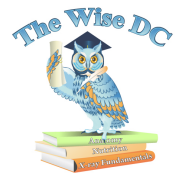
Eye - glaucoma, trauma.

Head - migraine, tension headache, cluster headache, cancer, cerebral aneurysm, sinusitis, meningitis.

Neck pain – myocardial infarction, trauma.

Thorax

Back - cancer, trauma.



Breast - premenstrual, cancer, trauma.

Chest – Myocardial infarction, pancreatitis, hiatal hernia, aortic dissection, pulmonary embolism, Costochondritis.

Shoulder - cholecystitis.

Abdomen

Left and right upper quadrant - peptic ulcer disease, gastroenteritis, hepatitis, pancreatitis, cholecystitis, abdominal aortic aneurysm, gastric cancer.

Left and right lower quadrant - appendicitis, ulcerative colitis, Crohn's disease, ectopic pregnancy, endometriosis, pelvic inflammatory disease, diverticulitis, urolithiasis, pyelonephritis, cancer.

Back

Back - Muscle strain, cancer, spinal disc herniation, degenerative disc disease, coccydynia.

Limbs

Arm - myocardial infarction (classically the left arm, sometimes bilateral).

Leg - deep vein thrombosis, peripheral vascular disease (claudication), musculoskeletal, spinal disc herniation, sciatica.

Joints

Classically small joints - osteoarthritis (common in the elderly), rheumatoid arthritis, systemic lupus erythematosus, gout, pseudogout/carpal tunnel syndrome.

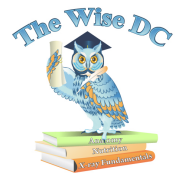
Classically large joints (hip, knee) - osteoarthritis (common in the elderly), septic arthritis, hemarthrosis, trauma.

Other - psoriatic arthritis, Reiter's syndrome.

Genotype and Pain

Pain may be experienced differently depending on genotype (i.e, genetic background). A study by Liem *et al.* suggests that redheads are more susceptible to thermal pain.¹²³ However, another study suggests that redheads--who have a non-functional melanocortin-1 receptor (MC1R) gene--are less sensitive to pain from electric shock.¹²⁴

Gene SCN9A has been identified as a major factor in the development of the pain-perception systems within the body. A rare genetic mutation in this area causes non-functional development of certain sodium channels in the nervous system, which prevents the brain from receiving messages of physical damage. People having this disorder are completely ignorant to pain, and can perform without pain various kinds of self mutilation or damage. In the families studied, this has ranged from biting of the person's own tongue leading to damage, to death from injuries due to a failure to have learned limits on injury through experience of pain. The same gene also appears to mediate a form of hyper-sensitivity to pain, with other mutations seeming to be "at the root of paroxysmal extreme pain disorder"¹²⁵



Section 10

Pain Assessment

Assessment of the patient experiencing pain is the cornerstone to optimal pain management. Pain assessment should include a history, physical examination, and a review of the results of pertinent laboratory and other ancillary diagnostic testing procedures. Initial evaluation of the pain complaint should include characteristics such as: intensity; character; frequency (or pattern, or both); location; duration; and precipitating and relieving factors. The mnemonic PPQRST may be helpful to follow: palliative, provocative, quality, region (or radiation), severity and temporal pattern of pain.

The WILDA Approach To Pain Assessment

Pain assessment should be ongoing (occurring at regular intervals), individualized, and documented so that all involved in the patient's care understand the pain problem. Using the WILDA approach ensures that the 5 key components to a pain assessment are incorporated into the process.

W-Words to describe the pain

Pain assessment usually begins with an open-ended inquiry: "Tell me about your pain." This allows the patient to tell his or her story, including the aspects of the pain experience that are most problematic. It is imperative that the clinician listens closely to the patient's words to describe their pain. It has been said that upwards of 90% of a diagnosis is derived from the patient's history.

A patient's statement, "I have pain," is not descriptive enough to inform a health care professional about pain type. Asking patients to describe their pain using words will guide clinicians to the appropriate interventions for specific pain types. Patients may have more than one type of pain. The following questions should be asked of patients:

What does your pain feel like?

Because various pain types are described using different words, what words would you use to describe the pain you are having?

Neuropathic pain. This type of pain can be described as burning, shooting, tingling, radiating, lancinating, or numbness. Sometimes patients say that their pain is like a fire or an electrical jolt. This type of pain can be due to nerve disorders; nerve involvement by a tumor pressing on cervical, brachial, or lumbosacral plexi; postherpetic neuralgia; or peripheral neuropathies secondary to treatment (chemotherapy, radiation fibrosis).

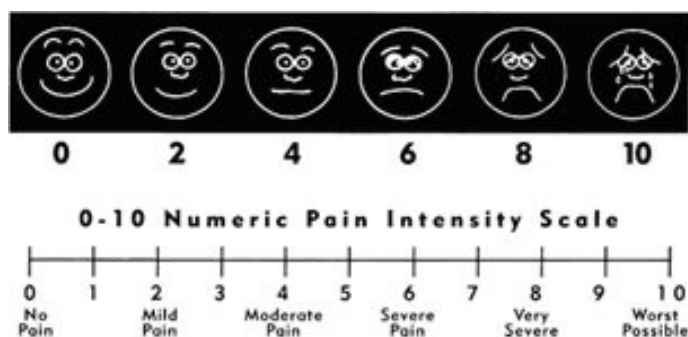
Somatic pain. Described as achy, throbbing, or dull, somatic pain is typically well localized. Somatic pain accompanies arthritis, bone or spine metastases, low back pain, and orthopedic procedures.

Visceral pain. Pain described as squeezing, pressure, cramping, distention, dull, deep, and stretching is visceral in origin. Visceral pain is manifested in patients after abdominal or thoracic surgery. It also occurs secondary to liver metastases or bowel or venous obstruction.

I- Intensity of the pain

The ability to quantify the intensity of pain is essential when caring for persons with acute and chronic pain. Though no scale is suitable for all patients, Many physicians use a 0 to 10 scale for clinical assessment of pain intensity in adult patients. Standardization may promote collaboration and consistency among caregivers in multiple settings—inpatient, outpatient, and home care environments. Using a pain scale with 0 being no pain and 10 being the worst pain imaginable, a numerical value can be assigned to the patient's perceived intensity of pain. Asking patients to rate their present pain, their pain after an intervention, and their pain over the past 24 hours will enable health care providers to see if the pain is worsening or improving. Also, inquiring about the pain level acceptable to the patient will help clinicians understand the patient's goal of therapy.

The Wong/Baker Faces Rating Scale is a visual representation of the numerical scale. Although the faces scale was developed for use in pediatric patients, it has also proven useful with elderly patients and patients with language barriers. Patients will be asked to rate their pain on a scale of zero - 10, where zero = no pain, 5 = moderate pain and 10 = the worst pain possible. Other patients, such as young children, those who do not speak English and those who are cognitively impaired may use the "faces" scale to describe their level of pain.



Verbal Description Scale	0 No pain	1,2 Mild pain	3,4 Moderate pain	5,6 Moderate pain	7,8 Severe Pain	9,10 Worst Possible
Wong-Baker Facial Grimace Scale	 Alert, smiling	 No humor, serious, flat	 Furrowed brow, pursed lips, breath holding	 Wrinkled nose, rapid breathing	 Slow blink, open mouth	 Eyes closed, moaning, crying
Activity Tolerance Scale	No pain	Pain can be ignored	Interferes with tasks	Interferes with concentration	Interferes with basic needs	Bed rest required

L- Location of the pain

Most patients have two or more sites of pain. Therefore, it is important to ask patients, “Where is your pain?” or “Do you have pain in more than one area?” Having the patient point to the painful area can be more specific and help to determine interventions.

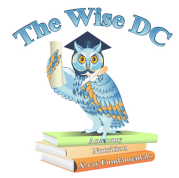
Localization is not always accurate in defining the problematic area. Some pain sensations may be diffuse or referred. Referred pain, usually happening in visceral disease, occurs when sensory fibers from the viscera enter the same segment of the spinal cord as somatic nerves i.e. those from superficial tissues. The sensory nerve from the viscus stimulates the closely associated nerve in the spinal cord and the pain perceived at the sensorial area of the brain is perceived as originating in the area supplied by the somatic nerve. An example is the left shoulder pain associated with heart damage.¹²⁶

D- Duration of the pain

The duration of a patient’s complaints of pain is often clinically significant. For example, fleeting or short duration of pain is rarely serious. Conversely, pain over a long duration of time can negatively impact a patient’s functional status thereby perpetuating the pain.

A- Aggravating/Alleviating Factors

Asking the patient to describe the factors that aggravate or alleviate the pain will help plan interventions. A typical question might be, “What makes the pain better or worse?” Other factors (movement, physical therapy,



activity, intravenous sticks or blood draws, mental anguish, depression, sadness, bad news) may intensify the pain.

Other things to include in the pain assessment are the presence of contributing symptoms or side effects associated with pain and its treatment. These include nausea, vomiting, constipation, sleepiness, confusion, urinary retention, and weakness. Inquiring about the presence or absence of changes in appetite, activity, relationships, sexual functioning, irritability, sleep, anxiety, anger, and ability to concentrate will help the clinician understand the pain experience in each individual.

Qualifying Pain Utilizing the PQRST Method

P = provocation / palliation : What were you doing when the pain started? What caused it? What makes it better? worse? What seems to trigger it? Does it seem to be getting better, or getting worse, or does it remain the same? What relieves it? What makes the problem worse?

Q = quality / quantity : What does it feel like? Is it sharp? Dull? Stabbing? Burning? Throbbing?

R = region / radiation : Where is the pain located? Does the pain radiate? Where does it radiate?

S = severity scale : How severe is the pain on a scale of 0 - 10, zero being no pain at all and 10 being the worst pain ever? Does it interfere with activities? How bad is it when it's at its worst? Does it force you to sit down, lie down? How long does an episode last?

T = timing : When did the pain start? How long did it last? How often does it occur? Is it sudden or gradual? What were you doing when you first experienced or noticed it? How often do you experience it: hourly? daily? weekly? monthly? When do you usually experience it: daytime? night? in the early morning? Are you ever awakened by it? Does it lead to anything else? Is it accompanied by other signs and symptoms? Does it ever occur before, during or after meals? Does it occur seasonally?

Mc Gill Pain Questionnaire

The **McGill Pain Questionnaire**, also known as McGill pain index, is a scale of rating pain developed at McGill University by Melzack and Torgerson in 1971.

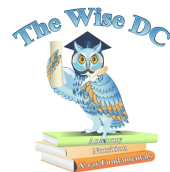
Instructions for use include the following:

1. Circle the words that describe your pain but do not circle more than one word in a group.
2. Then go back and circle the three words in groups 1-10 that most convey your pain response.
3. Pick the two words in groups 11-15 that do the same thing. Then pick one word in group 16. Finally, pick 1 word in groups 17-20.
4. At the end you should have seven words that you can take to your doctor that will help describe both the quality of your pain and the intensity of it.



Sample Questionnaire

Group	Words
1	Flickering, Pulsing, Quivering, Throbbing, Beating, Pounding
2	Jumping, Flashing, Shooting
3	Pricking, Boring, Drilling, Stabbing
4	Sharp, Cutting, Lacerating
5	Pinching, Pressing, Gnawing, Cramping, Crushing
6	Tugging, Pulling, Wrenching
7	Hot, Burning, Scalding, Searing
8	Tingling, Itchy, Smarting, Stinging
9	Dull, Sore, Hurting, Aching, Heavy
10	Tender, Taut (tight), Rasping, Splitting
11	Tiring, Exhausting
12	Sickening, Suffocating
13	Fearful, Frightful, Terrifying
14	Punishing, Grueling, Cruel, Vicious, Killing
15	Wretched, Blinding
16	Annoying, Troublesome, Miserable, Intense, Unbearable
17	Spreading, Radiating, Penetrating, Piercing
18	Tight, Numb, Squeezing, Drawing, Tearing
19	Cool, Cold, Freezing
20	Nagging, Nauseating, Agonizing, Dreadful, Torturing

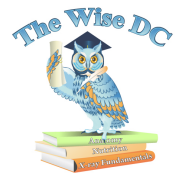


Section 11

Diagnostic Testing Procedures For Pain

There is no way to tell how much pain a person has. No test can measure the intensity of pain, no imaging device can show pain, and no instrument can locate pain precisely. Sometimes, as in the case of headaches, physicians find that the best aid to diagnosis is the patient's own description of the type, duration, and location of pain. Defining pain as sharp or dull, constant or intermittent, burning or aching may give the best clues to the cause of pain. These descriptions are part of what is called the pain history, taken by the physician during the preliminary examination of a patient with pain. However, as Doctors of Chiropractic we do have a number of diagnostic testing procedures at our disposal to include:

- Plain film radiographs
- Full spine, plain film radiographs
- Plain film radiographs: Stress films
- Videofluoroscopy / Cineradiography
- Plain film radiographs with contrast: myelography and discography
- Tomography
- Computerized tomography
- Magnetic resonance imaging
- Ultrasound/sonograms/echography
- Radionuclide scanning: bone scan
- Thermography
- Electrocardiogram
- Angiography
- Current Perception Threshold (CPT)
- Electroencephalography (EEG)
- Surface Electromyography (SEMG)
- Electromyography (EMG)
- Nerve Conduction Velocity (NCV)
- Somatosensory Evoked Potentials (SSEP)
- Brain Stem Auditory Evoked Potential
- Visual Evoked Potential



Overview of Frequently Utilized Diagnostic Procedures

Plain Film Radiographs

- Provide information regarding skeletal integrity, misalignment, components of vertebral subluxation and the dynamics of spinal motion.
- Conventional plain film radiography is the most widely utilized skeletal imaging method.
- X-ray image contrast is derived from the five radiographic densities - air, fat, water, bone, and metal.
- There is an absolute necessity of having a minimum of two views perpendicular to each other. These should be supplemented with additional projections, such as oblique, angulated, or stress studies, as clinically indicated.
- A routine cervical series of X-rays involves three views: A-P lower cervical, A-P open mouth cervical, and lateral cervical. A Davis series also includes obliques, flexion, and extension views and is considered the gold standard in cases of cervical whiplash.
- A routine thoracic series of X-rays involves two views: A-P thoracic and lateral thoracic.
- A routine lumbar or lumbosacral series of X-rays involves two to three views: A-P lumbar and lateral lumbar. A lateral L5-S1 spot shot is often elected.

Applications:

- Useful in the detection of skeletal abnormalities such as neoplasms, traumatic injury, infection, and anomalies.
- Provides information as to what additional imaging modality may be indicated or contraindicated.
- Allows for comparison of changes of a disease process over time.

Advantages:

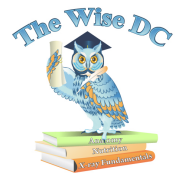
- Inexpensive and accessible.
- Provides excellent anatomical detail.

Disadvantages:

- Exposure of radiosensitive tissues such as the thyroid gland and gonads to ionizing radiation.
- Diagnostic sensitivity is limited.
- 30% to 50% loss of bone density and a lesion size of 1 to 5 cm is often necessary before a lesion is visible on X-ray. A patient may have extensive histologic disease and have a normal appearing radiograph. The time interval from when a disease process manifests clinically until it becomes visible radiographically is the radiographic latent period. This period can be quite long.
- Soft tissue depiction is limited.

Magnetic Resonance Imaging

Magnetic resonance imaging provides clear images of disc deterioration, pathologies of the spinal cord, spinal stenosis, herniated discs, spinal tumors, and abnormalities in nerves and ligaments. Contrast dye may be injected to highlight problematic areas.



Applications:

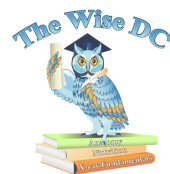
- Widely utilized for the differential diagnosis of both pathological and acute lesions.
- In recent times, MRI has replaced CT as the gold standard in the evaluation of cervical, thoracic, and lumbar disc disease.
- No other imaging modality defines the anatomical relationship of the intervertebral disc and the content of the spinal canal as accurately MRI.
- Unparalleled evaluation of spinal cord injury has been obtained with MRI.
- Only imaging modality which can visualize ligamentous tears, bone contusions, spinal hematomas, and spinal contusions.
- Used to detect vertebral fractures; however, CT is considered better at identifying neural arch fracture and the presence of bone fragments within the spinal canal.
- Spinal cord or nerve root injury because of its superior spatial and contrast resolution.
- Evaluation of bone hemorrhage and bone marrow edema.
- Infection of soft tissue and bone.
- MRI provides the most sensitive imaging modality in the detection of osseous metastatic disease in the spine. The initial MRI findings of vertebral metastasis usually affect the marrow adjacent to the posterior cortical margin. The remaining portion of the vertebral body, pedicles, and the rest of the neural arch may be involved secondarily as the neoplastic process spreads throughout the intravertebral venous system. The replacement of normal fatty marrow within the vertebral body is often seen with MRI in the absence of gross morphological alterations.
- Definitive diagnosis of multiple sclerosis. MRI is the first and only imaging modality that allows direct visualization of the central nervous system plaques that characterize MS. Recently, the use of contrast has made it possible to distinguish between acute inflammation and fresh plaques in areas of chronic involvement.
- Soft tissue injury to virtually every joint of the body.

Computerized Tomography

Computerized tomography is an x-ray that utilizes computer technology and can be enhanced with contrast dye. It is used to show abnormalities in bones and soft tissue. CT scan can be used for patients who are unable to tolerate MRI.

Applications:

- Most commonly used in conjunction with plain film X-ray findings.
- Traumatic lesions of the musculoskeletal system, particularly of complex anatomical structures, flat bones, vertebrae, pelvis, ankle, wrist, and shoulder.
- The use of CT is indicated when radiographs are equivocal or when suspected clinical findings are not substantiated by the present plain film study.
- In instances of comminuted fractures, CT provides information concerning the location of bony fragments.
- CT is the imaging modality of choice for visualization of calcified lesions of periosteum or soft tissues.
- Infection of discitis and osteomyelitis, are well visualized.
- In the evaluation of soft tissue neoplasm, CT best evaluates the osseous characteristics and calcified regions of a neoplasm. Able to detect bony changes of neoplasm earlier than X-ray; however, nuclear medicine scans are far more superior concerning early detection.
- Best suited for detailed assessment of bony cortex, bony expansion, periosteal reaction, or subtle fracture.



- Used in the assessment of congenital malformations of neural arch and facet asymmetry.
- Accurate assessment of the extent of degenerative joint disease, particularly in cases of spinal canal and lateral recess stenosis secondary to degenerative bony hypertrophy of endplates and facets and ligamentous hypertrophy of the posterior longitudinal ligament and ligamentum flavum.
- Evaluation for disc herniation or injury in communities where MRI is not available or when a patient is not candidate for MRI evaluation.
- MRI has replaced CT scanning as the imaging modality of choice for disc herniations in any region of the spine

Comparison of CT Scanning and MRI

	CT	MRI
1.	Accurate for evaluation of bone cortex, bony expansion, periosteal reaction, fractures, and subtle bone fractures particularly of the neural arch.	Accurate for evaluation of soft tissue injury, spinal cord injury, trauma of bone marrow, and bone fractures.
2.	Used to evaluate neoplasms of bone. Best demonstrates the osseous characteristics and calcified regions of soft tissue neoplastic processes.	Used to evaluate neoplasms of soft tissue, spinal cord, bone marrow, and bone. Provides early detection of vertebral metastatic disease.
3.	Accurate in determining spinal canal and lateral recess stenosis as a result of degenerative hypertrophy of vertebral endplates and facets and congenitally short pedicles.	The gold standard in the evaluation of intervertebral disc disease. No other imaging modality defines the anatomical relationship of the intervertebral disc and the content of the spinal canal as accurately.
4.	Utilized to evaluate osseous infections.	Utilized to evaluate soft tissue and osseous infections. Provides early detection of osseous infections.
5.	Does not adequately visualize the spinal cord for assessment of pathology.	Visualizes plaques and demyelination of the central nervous system.
6.	Uses ionizing radiation.	Does not use ionizing radiation.

Electrodiagnostic Tests

Electrodiagnostic procedures include electromyography (EMG), nerve conduction studies, and evoked potential (EP) studies. Information from EMG can help physicians tell precisely which muscles or nerves are



affected by weakness or pain. Thin needles are inserted in muscles and a physician can see or listen to electrical signals displayed on an EMG machine. With nerve conduction studies the doctor uses two sets of electrodes (similar to those used during an electrocardiogram) that are placed on the skin over the muscles. The first set gives the patient a mild shock that stimulates the nerve that runs to that muscle. The second set of electrodes is used to make a recording of the nerve's electrical signals, and from this information the doctor can determine if there is nerve damage.

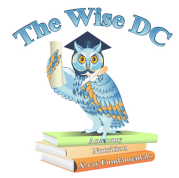
Myelography

Myelography is used to examine the spinal canal and cord. Contrast dye is injected into the cerebrospinal fluid to outline the spinal cord and nerve roots, thus allowing abnormal disc conditions or bone spurs to be visualized with x-ray or CT scan.

Spinal tap involves drawing a sample of cerebrospinal fluid and analyzing it for elevated pressure, infection, bleeding, or tumor.

Bone Scan

Bone scan locates problems (e.g., fracture, osteoporosis) in the vertebrae. A radioactive tracer is injected into the patient and after several hours, x-ray will reveal bone undergoing rapid changes where large amounts of tracer accumulate.



Section 12

Analysis of Back Pain

Incidence and Prevalence

“In the United States, back pain is reported to occur at least once in 85% of adults below the age of 50. Nearly all of them will have at least one recurrence. It is the second most common illness-related reason given for a missed workday and the most common cause of disability. Work-related back injury is the number one occupational hazard.”¹²⁷

Risk Factors Include:

- **Aging** produces wear and tear on the spine that may result in conditions (e.g., disc degeneration, spinal stenosis) that produce neck and back pain.
- A **previous back injury** puts one at risk for another injury.
- Physically demanding **occupations** that require repetitive bending and lifting have a high incidence of back injury (e.g., construction worker, caregiver).
- **Sedentary lifestyle** (i.e., not exercising regularly or engaging in physical recreation).
- **Being overweight** can increase stress on the lower back.
- **Poor posture and** poor body mechanics when lifting and carrying heavy loads are risk factors.
- **Sports** that involve twisting the back, such as baseball and golf, can result in an acute back injury or worsen an existing back injury.
- **Joint and/or bone disease** (e.g., osteoporosis, arthritis) and **infectious disease** (e.g., spinal meningitis) can lead to degeneration, inflammation, and compression.

Causes of Back Pain include:

- Vertebral subluxations.
- **Overuse or underuse of the back** is by far the most common cause of back pain that manifests as tightening or spasm of the muscles that connect to the spine. Inflammation and swelling often occur in the joints and ligaments, especially in the cervical and lumbar regions, as people age.
- A **herniated disc** which results from a tearing of the annulus fibrosus of the disc and extrusion of the nucleus pulposus causing nerve root compression.
- “Ninety percent of disc herniations occur in the lower two lumbar vertebrae.” ¹²⁸



- **Spinal stenosis**, narrowing of the spine, can cause spinal cord irritation and injury. Conditions that cause spinal stenosis include infection, tumors, trauma, herniated disc, arthritis, thickening of ligaments, growth of bone spurs, and disc degeneration. Spinal stenosis most commonly occurs in older individuals as a result of vertebral degeneration.
- Radiculopathy occurs when something rubs or presses against a nerve, creating irritation or inflammation. Radiculopathy can result from a herniated disc, bone spur, tumor growing into the nerves, and vertebral fracture, and many other conditions. **Sciatica** is an example of an extremely painful radiculopathy that involves inflammation of the largest nerve in the body, the sciatic nerve. Pain is experienced along the large sciatic nerve, from the lower back down through the buttocks and along the back of the leg.
- A **spinal tumor** that originates in the spine (primary tumor) or spreads to the spine from another part of the body (metastatic tumor) can compress the spine or nerve roots and cause significant pain.
- An **infection** that develops in the vertebrae (e.g., vertebral osteomyelitis), the discs, the meninges (e.g., spinal meningitis), or the cerebrospinal fluid can compress the spinal cord and result in serious neurological deterioration, if it is not diagnosed and treated immediately.
- **Facet joint degeneration can cause substantial low back pain..** As facet joints degenerate, they may not align correctly, and the cartilage and fluid that lubricates the joints may deteriorate.
- **Bone and joint diseases** (e.g., osteoporosis, ankylosing spondylitis, osteoarthritis) can cause degeneration, inflammation, and spinal nerve compression.

Multidisciplinary Approaches To Pain Management

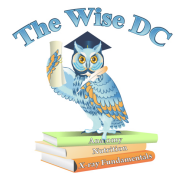
Medical Approach

Medications

- Acetaminophen and ibuprofen are most commonly recommended for pain relief.
- To relieve acute back pain, anti-inflammatory drugs (e.g., Celebrex), non-narcotic pain relievers (e.g., Tramadol), muscle relaxants (e.g., Flexeril), and narcotic pain relievers may be prescribed.
- An oral steroid (e.g., prednisone) is sometimes prescribed for acute episodes of low back pain. Patients are started on a high dose that is gradually reduced over 5 or 6 days. Serious side effects associated with steroid use include bone loss, impaired wound healing, and headache.
- Chronic back pain caused by nerve root damage is sometimes treated with tricyclic antidepressants, such as amitriptyline (e.g., Elavil) and nortriptyline (e.g., Pamelor), for numbness, burning, aching, throbbing, or stabbing pains that shoot down the limbs. Side effects include drowsiness, dry mouth, and constipation.
- Anticonvulsant drugs, such as gabapentin (Neurontin), may alleviate pain caused by nerve degeneration and persistent leg pain after surgery. Side effects include drowsiness, dizziness, fatigue, and impaired motor coordination.

Injections

- Steroid injections can significantly decrease inflammation and pain caused by spinal stenosis, disc herniation, and degenerative disc disease. A steroid is injected directly into the membrane that surrounds the nerve roots (dura). Selective nerve root block (SNRB) uses a steroid with anesthetic.



Surgery

- Surgery may be indicated for progressive or severe neurological dysfunction—such as muscle weakness, spinal cord compression, or bowel, bladder, or sexual dysfunction—and for cases of pain that is not easily relieved. Also, implanted pumps deliver a constant rate of pain-relieving medication to the spinal area. Surgically implanted spinal cord stimulators modulate the pain response, so the patient experiences less pain.

Physical Therapy Approach

- Physical Therapy

The goals of physical therapy are to decrease pain, increase function, restore normal movement, and prevent recurrences.

Physical Therapy Modalities

Cryotherapy

Is commonly used to alleviate the pain of acute musculoskeletal injuries.

Effects of Cryotherapy

- anesthesia (numbness)
- antispasmodic
- vasoconstriction reduces edema formation
- increased blood pressure, decreased pulse rate and respiration.

Heat Modalities

Effects:

Analgesic, Antispasmodic, increases connective tissue elasticity.

Vasodilator: Lowers blood pressure and increases blood and lymph flow which increases:

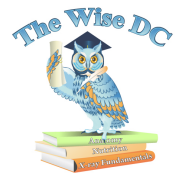
- phagocyte, leukocyte production
- edema formation (in acute phase/up to 72 hours)
- Pulse, perspiration, respiration and metabolic rate all increase

Indication: Sub-acute and chronic musculoskeletal conditions; sprains, strains, muscle spasms, back aches, myositis, tendinitis, bursitis, arthritis.

Contraindications include: Acute injuries and/or musculoskeletal conditions, cardiovascular disease, active tuberculosis, encapsulated swellings, diabetes mellitus.

Electrotherapy

Melzack and Wall's gate control theory of pain control suggests that nerve fibers carrying pain messages (C fibers) pass through the same segment of the spinal cord as those stimulated by electricity (known as A fibers).



It has been shown that the transmission of impulses along C fibers is slower than along A fibers. Therefore, overloading A fibers with electrotherapy would block the “gate” so the pain transmitted through the C fibers could not reach the brain.

Low frequency electromagnetic energy (<1000 Hz) is used to produce electrical stimulating currents which are used in electrotherapy. Two basic types of electrical current are used: alternating current/A.C. and direct current/D.C.

I. A/C. comes in many forms, the two types most commonly used are:

Sinusoidal (symmetrical)

Faradic (asymmetrical)

A/C can be used to stimulate:

1. sensory nerves for pain modulation *and/or*
2. neuromuscular elements in innervated muscles to produce electrokinetic effects: muscle contraction, muscle fatigue/relaxation, stretching fibrotic tissue, increased blood and lymph flow, decongestion and, detoxification.

II. Direct current (unidirectional) comes in two basic forms: galvanic and pulsed D.C.

Galvanic (waveless)

Pulsed (interrupted, shaped)

Direct current produces a mixture of electrokinetic and electrochemical effects. Galvanic current is used primarily for its electrochemical effects and is the only form of current suitable for iontophoresis. Pulsed square wave D.C. can be used to stimulate innervated and denervated muscle to produce electrokinetic effects (and is useful in electrodiagnosis). Hi-volt D.C. has little electrochemical effects and is primarily used to stimulate sensory nerves for pain modulation and to stimulate neuromuscular elements of innervated muscle for electrokinetic effects.

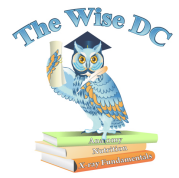
TENS (Transcutaneous Electrical Nerve Stimulation)

A procedure where an electrical current is passed across the skin. Generally, the term is reserved for small portable electrical units that patients wear to control pain. The portable unit is designed to provide sensory stimulation without motor stimulation. Afferent nerve fibers differ from efferent nerve fibers in length of refractory accommodation to stimuli, threshold of firing, and response to different wave forms. The wave forms of a TENS unit are interrupted or pulsed. Most units have a wave that alternates and is a variation of the faradic or square wave.

Electrode placement should be on the same dermatome as the patient's perception of pain, preferably over or proximal to the site of pain. In the presence of radiation, electrodes may be placed over the major nerve pathways.

Application

TENS is intended for symptomatic relief of a large number of painful syndromes. This would include the relief of chronic and intractable pain syndromes or cases where analgesic drugs would be contraindicated. Like all electrical stimulation, this procedure should be used with caution in undiagnosed pain syndromes where etiology has not been firmly established.



Massage

Massage therapy increases circulation to the affected area. There are several techniques and devices used in massage therapy.

Exercise

Exercise can correct current back problems, help prevent new ones, and relieve back pain, particularly after an injury. Proper exercise strengthens back muscles that support the spine and strengthens the abdomen, arms, and legs, reducing strain on the back. Exercise also strengthens bones and reduces the risk of falls and injuries.

Chiropractic Approach

Chiropractic management should be an integral part of care plans for most patients suffering from acute and chronic musculoskeletal pain syndromes. Chiropractic pain management strategies encompass a wide range of spinal manipulative treatment procedures as well as the use of physiotherapy modalities. Relatively speaking, chiropractic treatment methodologies carry fewer adverse effects compared to drug therapy and hospitalization.

According to Steven G. Yeoman's, "a single chiropractic adjustment produces both sensory and motor effects as well as sympathetic nervous system effects."

The sensory and motor effects of a chiropractic manipulation include:

- increased joint ROM in all 3 planes and reduction of pain.
- increased skin pain tolerance level.
- increased paraspinal muscle pressure pain tolerance.
- reduced muscle electrical activity and tension.

Sympathetic nervous system effects of a chiropractic manipulation include:

- increased blood flow and distal skin temperature.
- blood pressure reduction.

Blood chemistry changes after a chiropractic manipulation include:

- increased secretion of melatonin.
- increased plasma beta endorphin levels.
- elevation of Substance P and enhanced neutrophil respiratory burst.
- pupillary diameter changes.¹²⁹



Non-Medication Treatments for Pain

The following methods have been shown to help enhance the effect of pain medication.

Acupuncture

An acupuncturist inserts hair-thin needles under the skin, which remain in place for 15 to 30 minutes. The needles cause little or no pain. Pain relief may result from the release of endorphins, the body's intrinsic painkillers.

Deep Breathing - A common reaction to pain is to tense the muscles. Slow, deep breathing can be used to relax muscles and relieve pain.

Relaxation - Relaxation videos may be borrowed from the Education Department (ext. 6195) during your stay.

Progressive Muscle Relaxation - Alternatively tensing and relaxing muscle groups.

Imagery - Imagining peaceful places. See, touch, feel, smell and taste everything in your imagination.

Distraction - Any activity that takes your mind off of pain. Examples include reading a book, listening to music, watching television or doing crafts.

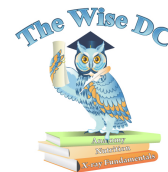


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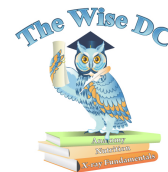
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