A CONCEPTUAL MODEL OF PAIN

The practicing physician's guide to the causative mechanisms of pain and how to translate these basic principles into diagnoses and treatments.

By James Woessner, MD, PhD

[Editor's note: This is the first installment in a series of three articles. The second installment in the Nov/Dec 2002 issue issue will explain how to hypothesize, measure and diagnose complex pathologies involved in most pain conditions. The third installment in the Jan/Feb 2003 issue will detail the neural sites of action and give descriptions of logical and reasonable clinical treatments of nociceptive, neurogenic or central pain pathophysiology.]

Pain, as a concept and symptom, is discussed and described throughout professional and lay medical literature. "Pain" is the reason for initial contact with any physician for the vast majority of medical problems, e.g. abdominal pain, chest pain, limb pain, low back pain, etc. Understanding pain mechanisms, however, is often hampered by the complex neural interconnections involved in pain. Any one, or combination, of the following mechanisms can contribute to pain: neural "sensor" stimulation, neural "wire" misfiring, and "perceptron" dysfunction.

The general public often righteously complains about the lack of recognition and help for their pain and discomfort — aside from direct treatment of the underlying medical pathology. On the other hand, many physicians feel helpless, in the face of the onslaught of public indignation, to help with pain that is not supported by "standard" laboratory and/or radiological evidence, or even by "physiologically" consistent behavior; someone just can't hurt overall!

Medical doctors depend on knowledge of the pathophysiology or at least a diagnosis to decide on treatment. Thus, to maximize success, physicians need to understand how pain is perceived. Certainly, knowing where and what the problem is, increases the likelihood of a positive outcome. Present categories of pain mentioned in medical literature are helpful, but these concepts are not organized to provide the practicing physician with handles that can help the physician more effectively treat those patients presenting with pain — particularly chronic pain.

While the Joint Commission[1] now recognizes and mandates pain as the "fifth vital sign," the present focus of Pain Medicine is "cover-up" rather than "cure." Even with an abundance of detailed Pain Medicine literature, there appears to be a limited understanding of the basic mechanisms of pain, even within the research world. Obviously, without a reasonably detailed diagnosis reflecting the underlying pathophysiology of a given pain, treatment is no more than "hit and miss."

In the defense of physicians, we only know what we know and cannot be expected to be omniscient in our understanding and insight of all medical problems. If we cannot "cure," then it is good medicine to "cover-up" to reduce suffering. In either case, good medical care must be based on "diagnosis, diagnosis, diagnosis," and a mechanistic understanding of the underlying pain pathophysiology.

Furthermore, in light of the apparent epidemic of under-treatment of pain — which can have serious legal consequences (i.e., a recent misdemeanor conviction in California) — and the sometimes necessary prescription of socially-unacceptable narcotics in treating chronic pain, the treating physician must understand the basic pathophysiology of pain to provide both optimal and medically-justifiable treatment. For all these reasons, there is an urgent need to clarify, organize and synthesize the abundance

of information about pain, and apply these conclusions as simply as possible to the practical treatment of patients.

The Present State of Pain Theory and Thought

Pain is described in a myriad of ways:

- in temporal terms: chronic pain, subacute pain and acute pain
- in characterizations: intermittent pain, intractable pain, lancinating pain, referred pain, burning pain and dull pain
- in acceptable diagnoses (which are all basically syndromes): phantom pain, cancer pain, vascular pain, arthritic pain, nerve pain, muscle pain, fibromyalgia, myofascial pain, sympathetically maintained pain, and complex regional pain syndrome
- in mechanistic terms: neuropathic and nociceptive pain
- in anatomic perceptional terms: headache, back pain, neck pain, facial pain, limb pain, abdominal pain, etc.
- in source of origin terms: central pain as originating in the spinal cord or brain
- in psychiatric/psychogenic terms: psychosomatic "in-the-head" pain, etc.

These descriptors variously imply the chronicity, the character, the cause or the location of this type of unpleasant sensation. To add complexity, many factors, such as culture, personality, psychosocial stressors and nutritional status, can be involved to influence the degree of pain and to confound the causal factors of the pain.

Caudill[2] analyzed pain from different angles to emphasize its complexity, where pain:

- biologically — serves as a signal that the body has been harmed,
- psychologically — is experienced as emotional suffering,
- behaviorally — alters the way a person moves and acts,
- cognitively — calls for thinking about its meaning, its cause, and possible remedies,
- spiritually — serves as a reminder of mortality,
- culturally — tests a people's fortitude or forces their submission.

Pain disorders are categorized in the DSM-IV-TR[3] (coded for the medical condition) as follows:

- 307.80 Pain Disorder Associated with Psychological Factors
- 307.89 Pain Disorder Associated with both Psychological Factors and a General Pain Condition

Elsewhere, the DSM-IV-TR[3] attributes neural dysfunction to pain. Again, these are only descriptive categories.

The simplest traditional categorization of pain has been "acute" and "chronic." Acute pain is really just a result of the stimulation of a normally-functioning pain detection system and serves to alert us to avoid or minimize tissue damage. Chronic pain merely means that pain is perceived over a long period of time, which has been arbitrarily set at 6 months.

Descartes showed a basic understanding of the pain pathways in 1664.[4] However, this schematic (see Figure 1) suggests that he only appreciated nociceptive pain, which implies normally functioning pain pathways. A more recent rendering of the details of pain pathways is presented in Figure 2.[5]

Formal classification systems (see Table 1) do exist and provide some insight into what we are trying to accomplish in this article.

The most advanced concepts are expressed by Craig,[6] who states that pain is just one manifestation of the mind-body's homeostasis system. From the patient's point of view, the spectrum of pain control spans temporary treatments (usually pharmaceutical) in suppressing pain to permanent remission or cure of the underlying pathology/disease.

While these are all very useful concepts, they are generally academic in nature and do not provide much practical help to a physician. Concepts of pain pathophysiology, and thus classification, are abundantly available in the scientific and medical literatures. There is a need to refine and clarify all of this information and apply it as simply as possible to the treatment of pain in the physician's office.

Dallel and Voisin recognized the need for a clear roadmap: "once pain-generating mechanisms are known, it becomes possible to establish the appropriate treatment of pain."[7] We suggest that refining these concepts are a giant step in the right direction and propose to present a simple, clear pathophysiologically-based classification model. We contend that pain treatment should primarily focus on reversing pathologic mechanisms that cause the pain in the first place.

Neuroanatomy and Neurophysiology

Nerves, or neurons, are long tubes of protoplasm (rather than a series of "sausage links") and which may, or may not, be surrounded by poor conducting myelin (insulation). Nerves generally present themselves in various sizes and characteristics and have numerous branches to other neurons. Neurons interact/communicate via numerous electrical (gap junction) and chemical synapses. There are motor

FIGURE 1: *Descartes[4] depicted the passage of pain signals from the source of the insult to the brain.*

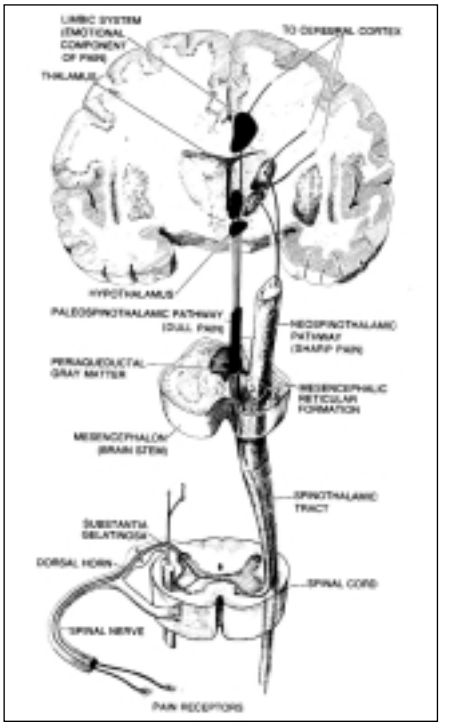

Figure 2: *A 1998 presentation of details of pain pathways.[5]*

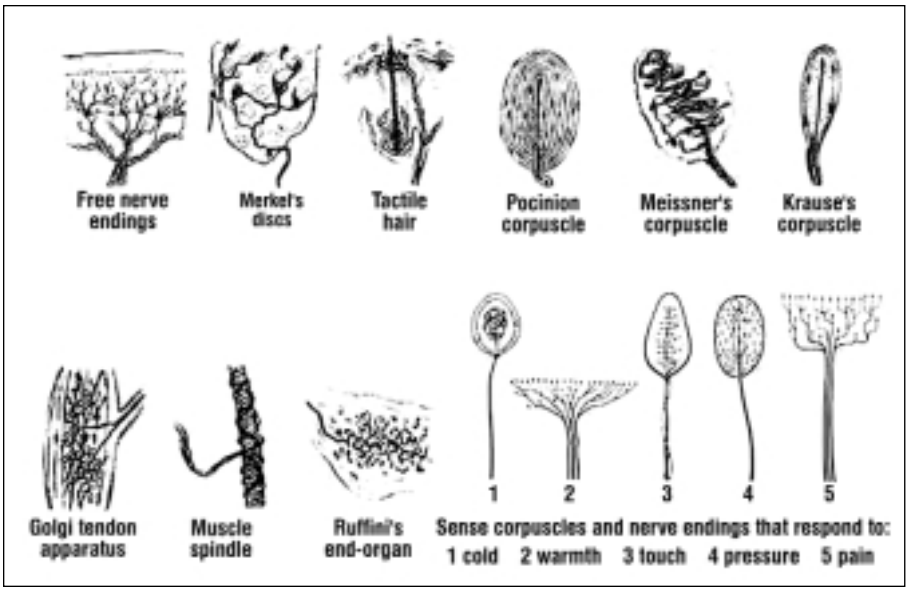

FIGURE 3: *Variety of nerve endings. In this context, we are primarily interested in those that sense stimuli that are transmitted as pain, i.e. mainly free nerve endings.*[5,8]

(efferent) neurons, which primarily carry signals from the brain to muscles, and sensory (afferent) neurons, which primarily carry signals from the periphery to the brain.[8]

The primary focus in Pain Medicine are the small sensory nerves, which carry unpleasant signals to the brain and may or may not be perceived by the brain. Descartes[4] depicted exactly that: a noxious stimulus causes information to be conveyed to the brain which is then perceived as pain (see Figure 1).

Neural signals are conveyed by potassium and sodium ions moving into and out of neurons via voltage-gated channels, in specific patterns, to form a relatively slow moving wave of information to, from, and within the central nervous system. These voltage-gated channels are concentrated in "holes" in the myelin (nodes of Ranvier), but are more evenly distributed in the more primitive unmyelinated nerve fibers (C-fibers).

In the absence of neural wire damage, there is a continuum across various numbers of synapses (switching stations) from the source or place of stimulation to the site of perception. At the distal end of sensory nerves, there are various types of nerve endings. When it comes to pain nerves, those endings are so-called "free"

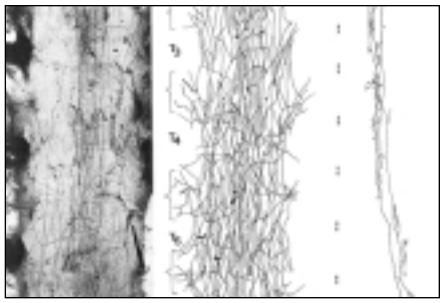

FIGURE 4: *Stained nerve fibers that coat the skin around the spinal cord.*[11] *We expect that a similar distribution occurs on other tissue surfaces throughout the body.*

nerve endings (see Figure 3). At the proximal end is the "perceptor" areas of the brain.[8]

There are three types of fibers that carry pain signals to the brain — A-beta, A-delta and C-fibers. The first two are evolutionarily modern fibers that are myelinated (insulated) and carry nerve impulses rapidly to the cortical regions of the brain (refer to a basic neurophysiology textbook).

The A-beta are probably reserved for deep, lancinating pain; certainly these carry vibratory signals. The A-delta fibers are somatic, myelinated fibers that have primary connections to the cortical regions of the brain. These fibers convey sharp, lancinating, easily localized pain signals; this pain sensation usually quickly passes. The C-fibers are relatively primitive, are unmyelinated and conduct rather slowly to the subcortical part of the brain. The brain perceives a more generalized burning, aching pain sensation,

Pain Classification System Table

Categories	I	II	III	IV	V
Traditional	Acute	Subacute	Chronic		
Pathogenetic	Primary	Secondary	Tx. Effect (chemotherapy, tissue trauma, edema, etc.)		
ICD-9	Disease process	Pain location	Secondary		
IASP[9]	Region	System	Chronology	Intensity	Etiology
Biopsychosocial	Acute	Recurrent acute	Condition related		
Dickerson	Neuropathic	Inflammatory	Long-term		

(special case adapted by Brookoff,[10] *who elaborates the various subtypes)*

TABLE 1. *The "traditional" classification scheme merely addresses chronology. The "pathogenetic" system grossly indicates the cause. "ICD-9" coding is as listed above. The IASP system provides a detailed description of the pain, but fails to approach the cause, except generally in Etiology; the IASP definition of pain avoids linking pain to a specific stimulus or cause. "Biopsychosocial" considerations is one step up from the "traditional." Dickerson starts in the right direction, but still misses a direct approach to pathophysiology.*

and this pain takes longer to pass. Thus, when one experiences a paper cut, you quickly appreciate a "zing" followed by a "burning" pain. You know exactly where the "zing" comes from, but the brain sees the burning pain through "fogged glass."

Different nerves, when it comes to function, have different characteristics. These characteristics may overlap between function (see Table 2). There is no absolute scientific agreement as to how features and function are related, but to facilitate discussion and understanding, generalization is desirable (see Table 3).

Now that we know generally how these small nerves work, we need to know where these nerve endings and small pain nerves occur. Our standard anatomy books, such as Netter,[12] do not depict or describe these networks of nerves. Dr. Fishman,[13] an insightful pain doctor, has described in his book entitled, "The War on Pain," that these nerve fibers cover and line most of the tissue plane surfaces throughout the body. Figure 4 presents stained nerve fibers coating the skin around the spinal cord and provides a general impression of how the nerves cover other tissue planes/surfaces.

Proposed Model of Pain Neurophysiology

Nociceptive pain is merely normal functioning of the neural sensor/wire/perceptron system. This system serves useful purposes in alerting the brain to bodily injury. Neuropathic and central pain, however, are manifestations of true dysfunction, and can be the "disease" itself.

If one considers a bundle of axons (see Glossary), neuropraxia, axonotmesis and neurotmesis represent points along a complex continuum of damage to axons and nerves. The three possibilities for individual axons are normal function, hyperfunction (hyperesthesia, hyperalgesia, hyperpathia, and allodynia) and hypofunction (hypoesthesia, hypoalgesia, conduction block, and death). Hyperfunction can also be thought of as sensitization or irritation. The ultimate hypofunction is axon death without regrowth. Free nerve endings can also be sensitized or irritated, but is considered here to be in the neuropathic category.

Understanding neurophysiology of pain pathways is helpful. Further, we propose that all pain can be understood by consid-

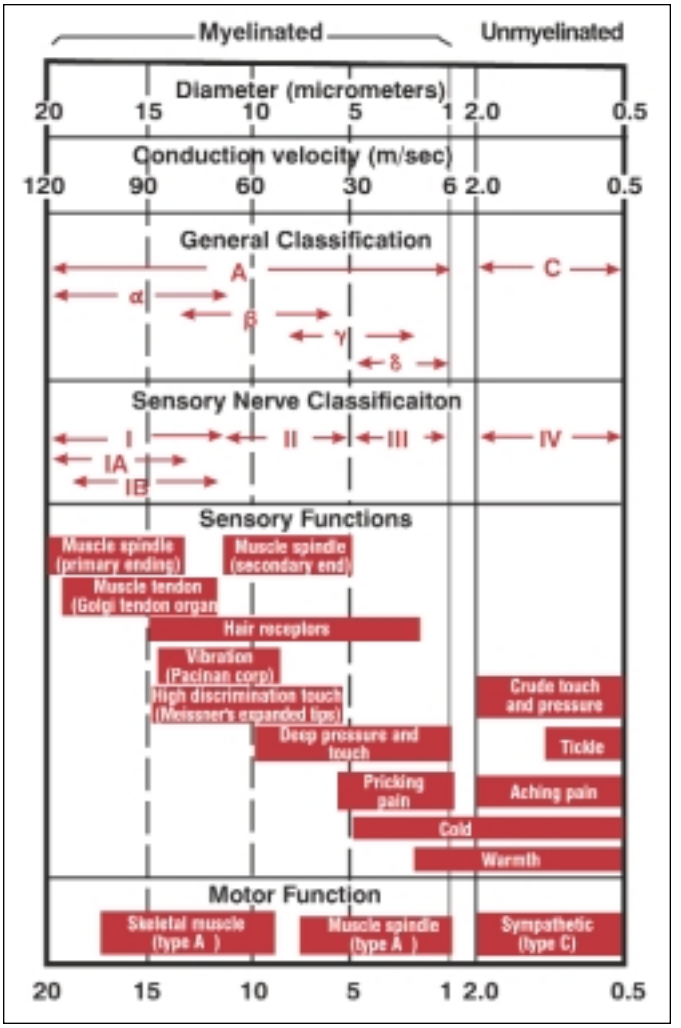

TABLE 2. *Physiological classifications and functions of nerve fibers, which shows the complexity of functional overlap and associated nomenclature.*[8]

Peripheral Nerve Fiber Types/Characteristics

class\units	stimuli/function	perception	conduct velocity m/sec	diameter microns	myelination
A-alpha fibers	motor contraction efferent transmission	none direct	30-85	12-22	+++
A-beta fibers	vibration and pressure afferent transmission	vibration and pressure	30-70	5-12	+++
A-delta fibers	cold sensation and pain fast pain and localized touch afferent transmission	cold sensation and pain localized touch	5-25	1-4	++
C fibers	hot sensation and pain slow pain and generalized touch afferent transmission	hot sensation and pain generalized touch	0.7-2.0	0.3-1.3	-*

** C-fibers can still be clumped and embedded in other non-conducting tissue.*

TABLE 3. *Summary of primary nerves under discussion here; simplified from above and other sources (notably Haines,[14] Cousins,[15] and Raj[16]).*

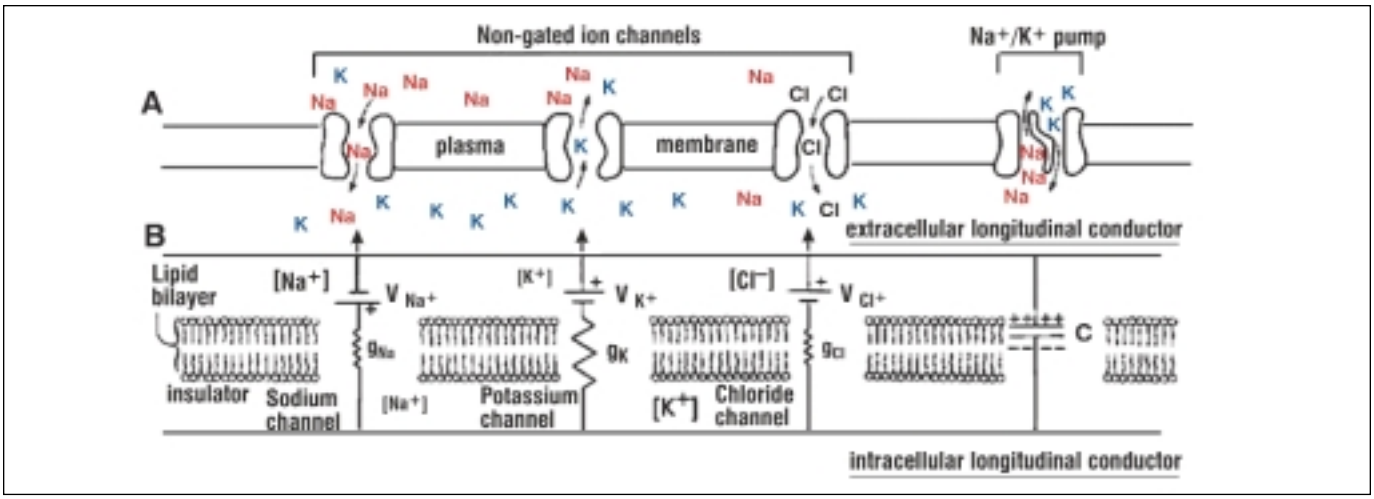

FIGURE 5: *The Haines[14] electrical equivalent circuit of nerve cell membrane. The lipid bilayer is an insulator separating the conducting extracellular and intracellular fluids. Na^+, K^+, and Cl^- channels penetrate the membrane, permitting flux of ions (A). Na^+ and Cl^- have highest concentrations outside, and K^+ has highest concentration inside the cell (B). These potential gradients determines the magnitudes and polarities of the three-ion "batteries." In the resting membrane, the K^+ channel has the highest permeability, so the K^+ potential dominates the membrane otential. The capacitance C, is composed of the insulating membrane and the conductive extracellular and intracellular electrolytes.*

ering problems of stimulation of sensors, conduction along nerves and/or perception in the spinal cord and brain. The perception then may involve feedback, either positive or negative (i.e. release — or not — of native painkillers, e.g. endorphins). If negative, the result is, by and large, a dysfunction that conceptually could stand alone.

Haines[14] depicts an electronic schematic of the nerve cell membrane (see Figure 5) and forms the basis of concepts discussed below. A key concept is that the neural pain system follows basic electrical principles.

The analogy of the neural net in complex electrical circuitry seems to be an accurate one. The pain sensors (free nerve endings) are relatively simple. The wires (peripheral nerves) are even simpler. The central nervous system, however, is incredibly complex. We are discovering that the spinal cord is not just a transmission device; complex interactions can occur here also. Finally, the complexity of the brain is difficult to imagine with billions of neurons and millions of synapses.[8]

Essentially no pain condition is unifactorial. For the actual pain conditions that the practicing physician encounters, it is useful to assess the pain mechanisms afflicting a patient using a conceptual framework of the relative mechanistic contributions of pain perception. A sample pain mechanistic conceptualization matrix is presented in Table 4. This approach has been found to be a useful tool in assessing an individual patient's pain and deciding on treatment within the conceptual pain model. The physician is encouraged to generate an individualized pain mechanistic conceptual matrix for each pain patient as a tool in deciding on diagnoses, and thus treatments. (A blank matrix suitable for reproduction is included as an appendix to this article.)

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Pain Perception within the Conceptual Framework

Stimulation of Sensors (nociceptive pain)

Normal stimulation of pain sensors is manifested in "good" pain (eudynia; "Pain: The Gift Nobody Wants"[17]) in that the free nerve endings of pain pathways are working perfectly and normally — giving good information to the body and brain that tissue is being damaged — or about to be damaged — and that the body needs to do something. Impact on mechano(noci)-ceptors, heat or cold stimulation of thermo(noci)ceptors or caustic chemicals on chemo(noci)ceptors start the process of perception of pain. In other words, this type of pain is based on mechanical, thermal, and/or chemical stimulation of normally functioning pain nerves; nerves that detect pain as a signal and indicating impending or active tissue damage.

Misfiring of Wires (neurogenic or neuropathic pain)

During the normal transmission of neural signals to the central nervous system, any damage to the neural pathway itself may manifest itself analogous to "static" in radio transmissions. This neural "static" alters the neural signal and is then perceived as pain. Nerves can be damaged just as any soft tissue — in which these nerves occur — can be damaged. Neuropathic pain therefore is merely damaged and malfunctioning wires/nerve fibers. One can also conceive of similar damage to nerve fibers in the central nervous system. As

Weighted Contributions in Acute and Chronic Pain Conditions

		nociceptive		neuropathic		central	
	pain etiology	acute	chronic	acute	chronic	acute	chronic
1	acute trauma	+++	-	-	-	-	-
2	bone fractures	+++	+++	-	-	-	-
3	radiculopathy	+++	+++	++	++	-	+
4	peripheral neuropathy	+	++	++	+++	-	-
5	arthritis	+	+++	-	+	-	-
6	headaches	+	+++	-	-	-	-
7	CRPS	+++	+++	++	+++	-	+++
8	myofascial	+++	+++	-	-	-	-
9	fibromyalgia	+	+++	+++	+++	-	+++
10	phantom pain	+++	+	-	-	-	+++
11	psychiatric problems	-	-	-	-	+++	+++

Nociceptive - normal functioning of pain free nerve endings
Neuropathic - actual nerve damage resulting in hypoactivity or hyperactivity
Central - pathway malfunction in the central nervous system

TABLE 4. *Author's sample illustration of the potential role of each of the above proposed mechanistic categories in these major pain conditions and how each of these pain conditions may be described acutely and chronically depending on the mechanism of pain generation. Each individual patient's condition will be different in detail and over time.*

long as those fibers are not the end of the pathway, the phenomenon is similar. Damaged nerve fibers follow a course of anatomic and physiologic change involving irritation (hyperactivity) and dysfunction/death (hypoactivity). Upon nerve death, of course, signals can no longer be transmitted along the neural pathway.

Perceptron Dysfunction of Perception (central pain)

The most complex part of the pain pathway(s) is in the central nervous system and occurs at the end of the neural pathway, where these signals are interpreted — the spinal cord and/or the brain. Perception and consequences can occur in the dorsal horn of the spinal cord. If central neurons malfunction in any part of the pain perception pathway, one possible consequence is that the brain perceives "pain." The biochemical environment of the central nervous system can also play a part. This complex system can be considered together to be a "perceptron." This word has been chosen to convey the truly complex, computer-like nature of these central nervous system phenomena. This pain pathology can also be called central neurogenic pain.

Perceptron Dysfunction of Response (antinociceptive pain)

The human body possesses anti-pain (anti-nociceptor) systems producing endorphins, enkephalins, etc. that are utilized as natural painkillers. In normal function, the human body releases these painkillers to modulate or mollify pain. At the very least, if these chemicals are not released or do not arrive at the affected receptors, the perceptrons will appreciate pain or greater pain in the presence of pain signals.[10]

Combined Pain Perception (most chronic pain conditions)

Over time and with the presence of widespread and/or severe causal factors, more than one aspect of the pain perception system may be malfunctioning at the same time. For example, it is common for patients to develop pain in a limb due to trauma that injures small pain fibers in addition to the other soft tissue. One can have stump pain along with phantom pain, possibly not coincidentally. Central sensitization can develop over time in a patient with ongoing peripheral disease. Dysfunctional efferent reflexes or reactions can change the physical and chemical environment of pain sensors, which, in turn, then cause nociceptive pain.

Complex Regional Pain Syndrome (CRPS)

Possibly the ultimate combined pain condition is Complex Regional Pain Syndrome. CRPS probably starts with concomitant damage to small pain fibers (C-fibers and A-delta fibers) with relatively minor trauma to local tissues of the body. There is probably functional and dysfunctional cross-talk between these fiber types and the damage can be equal or predominantly one or the other. The surrounding tissue damage causes physical pressure/impact and caustic chemical stimuli to the nociceptive sensors. Normal pain signals are probably, to varying degrees, amplified or otherwise altered on their way to the central nervous system (afferent pathways). It is likely that efferent nerves are also either damaged or experiencing dysfunctional feedback and thereby changing, or completely blocking, signals to receptors on muscles and other tissues. Long-term efferent signals seem to change the physical structure of the initially-damaged tissue (a classic pathology in CRPS/RSD patients), while long-term afferent pain signals (nociceptive and neuropathic) can alter central nervous system perceptive function.[18] Because CRPS can spread to other parts of the body from the original site, as well as due to other reasons, central (spinal and/or brain) pathology is probable.[19]

Summary

All chronic pain conditions are patchy or widespread combinations of the three basic pain dysfunctions discussed: nociceptive,

neurogenic, and central. The complex mechanical, biochemical, and thermal causes of dysfunction in information transmissions, as well as structural/micro-anatomic changes, are being intensively and extensively studied all over the world.

While the details of pain pathophysiology is incredibly complex, pain can be categorized pathophysiologically so that a physician can make an educated shot at providing focused and logical pain relief to their patients. The better the physician understands the pathophysiology of the patient's pain, the more effective the treatment can be.

For nociceptive pain, the primary goal is to resolve ("cure") or remove the stimulant (the causative pathology), while covering up the pain. For neurogenic pain, the goal is to stop the irritation and promote rebuilding of the damaged nerves or normalization of their function. For central pain, the goal is to employ techniques to change the central nervous system neural environment. For antinociceptive pain, the goal is to normalize pain perception and reestablish natural painkiller production and function.

The ultimate "trick" for effectively treating pain is individualizing and balancing the various approaches for optimal results in complex chronic pain cases. By understanding the cause, the physician clearly has a better chance of effectively serving their patients with better pain relief. ■

Dr. James Woessner holds a doctorate in bioscience in conjuction wih a medical degree. His professional training includes neurology and physiatry. Dr. Woessner records data on a daily basis in his solo practice and frequently participates in active academic discussions about pain and other subjects in Physical Medicine. Dr. Woessner may be contacted at Advanced Phys Med, 3628 50th Street, Lubbock, TX 79413; 806-780-2080.

Glossary

Allodynia — pain due to a stimulus that does not normally provoke pain; dynamic, static and thermal.[20]

Analgesia — absence of pain in response to stimulation that would normally be painful.[20]

Axon — a single tube of protoplasm extending from peripheral receptor to ganglion, ganglion to ganglion or ganglion to preceptor. The longest can be on the order of a meter long. The numerous primitive small axons have no myelin (isolation). The larger, more advanced axons are variously myelinated.

Axonotmesis — interruption of the axons of a nerve followed by complete degeneration of the peripheral segment, without severance of the supporting structures of the nerve; such a lesion may result from pinching, crushing, or prolonged pressure.[21]

Conduction block — failure of impulse transmission at some point along a nerve, although conduction along the segments proximal and distal to it are unaffected; clinically, most often the result of an area of focal demyelination, when caused by focal trauma, called neurapraxia, neurotmesis and axonotmesis.

Eudynia — good pain, basically acute nociceptive pain.

Hyperalgesia — an increased response to a stimulus that is normally painful.[20]

Hyperesthesia — increased sensitivity to stimulation, excluding the special senses.[20]

Hyperpathia — abnormally painful reaction to a stimulus, especially a repetitive stimulus, as well as an increased threshold.[20]

Hypoalgesia — diminished pain in response to a normally painful stimulus.[20]

Hypoesthesia — decreased sensitivity to stimulation, excluding the special senses.[20]

Maldynia — bad pain, basically chronic non-functional pain.

Neurapraxia — the mildest type of focal nerve lesion that produces clinical deficits; localized loss of conduction along a nerve without axon degeneration; caused by a focal lesion, usually demyelinating, and followed by a complete recovery.[21]

Neurotmesis — a type of axon loss lesion resulting from focal peripheral nerve injury in which, at the lesion site, the nerve stroma is damaged to varying degrees, as well as the axon and myelin, which degenerate from that point distally; with the most severe nerve lesions, the gross continuity of the nerve is disrupted.[21]

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Weighted Contributions in Acute and Chronic Pain Conditions

		nociceptive		neuropathic		central	
	pain etiology	**acute**	**chronic**	**acute**	**chronic**	**acute**	**chronic**
1	acute trauma						
2	bone fractures						
3	radiculopathy						
4	peripheral neuropathy						
5	arthritis						
6	headaches						
7	CRPS						
8	myofascial						
9	fibromyalgia						
10	phantom pain						
11	psychiatric problems						

Nociceptive - normal functioning of pain free nerve endings
Neuropathic - actual nerve damage resulting in hypoactivity or hyperactivity
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APPENDIX. *Woessner Pain Mechanism Conceptualization Matrix Form*