The Integration of Pain Sciences into Clinical Practice

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ABSTRACT: In the past three decades, a scientific revolution has occurred in the understanding of the experience of pain. However, a clinical revolution based on the new science is yet to occur. Pain is a multidimensional experience with many contributing and interacting biological/pathobiological mechanisms. These mechanisms may be nociceptive, peripheral neurogenic, central, affective/cognitive or relate to output systems such as the motor and autonomic nervous system. With a better understanding of pain-related neurobiology and some clinical decision making skills, reasoned attempts at a diagnosis of pain can be made. The essential question and first step related to clinical integration is to ask, "what is (are) the predominant mechanism(s) in a given patient's pain state?" This paper provides the underlying clinical biology of pain mechanisms and proposes pain patterns related to the mechanisms.
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Pain has become a trendy topic. The number of pain journals has increased, journals such as this one now include special pain editions, and membership of the International Association for the Study of Pain (IASP) is growing rapidly. All these things parallel an undoubted scientific revolution in the understanding of the phenomenon of pain. Witness the new knowledge about neurotransmitters, dorsal horn and brain circuitry, for example, much of it now filtering down to popular science magazines. The major catalyst was surely Melzack and Wall's gate control theory in 1965,[1] the beginning of a serious challenge to prevailing peripheralistic and mechanistic concepts of pain.

However, this revolution began more than 30 years ago and despite the years, there is little evidence to show that patients are benefiting. The scientific revolution has not turned into a much-needed clinical revolution. For example, approximately one person in every ten in Western society suffers a persistently painful problem[2]—one in seven according to Magni.[3] The numbers of patients with pain problems may be getting larger, especially those with so-called musculoskeletal pain. Despite the revolution, information may not be getting through to the clinician at the battlefront in an easily integrative framework, or the clinician is ill equipped to change, or the clinician refuses to change. After all, to take on pain in its entirety may mean admitting that previously comfortable practices may have been wrong or that responses to well-meaning therapeutic intervention may have occurred for completely different reasons than that held by the clinician.

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In our view, to integrate pain sciences into a clinical science that benefits patients, professionals who deal with pain must understand the biology and pathobiology of the whole pain phenomenon. They must have the ability to diagnose pain, or at least categorize pain and make clinical decisions related to the categories. Clinical integration skills are as essential as knowledge of neural circuits, transmitters, and receptors. To begin this process, the key question to consider in the patient with a painful hand is, "What class of pain is the patient suffering?" This simple question, if answered using the most recent developments in pain-related sciences, will provide a powerful feel for prognosis, an awareness of new therapies, and perhaps render some currently used tests and therapies obsolete.

PAIN—SPECIAL ISSUES FOR HAND THERAPISTS

To take on and integrate pain into clinical practice, broad key issues, some of which may be confronting, need to be appreciated. First is the definition of pain. The IASP definition has survived some years of argument. The association defines pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage."[4] In our opinion, most therapists do not take the time to think and analyze the real meaning of this definition. The key words are experience, emotional and the concept of potential tissue damage. By broad definition, inputs such as anxiety, fear, and frustration affect the same clusters of neurones in the central nervous system (CNS) to create a perceptual experience, as do inputs from damaged tissues. Another key issue that arises from the definition is the frequent lack of close correlation between injury and pain.[5] The potential for a close

correlation between injury and pain always exists, but other factors (such as anxiety, fear, and frustration) powerfully influence the central processing of incoming information from the injured tissues. For every tissue injury, or for every nociceptive input, however major or minor, our clinical thinking needs to incorporate a hypothetical "brain/mind scrutinizing module" that has the power to decide (subconsciously and/or consciously) if the input is worth contemplating and reacting upon. Much of this is determined by the brain modules' past experiences, knowledge, beliefs, and culture.

Lastly, note that there are cases where noxious stimuli can always produce an injury but never evoke pain—for example, inhalation of carbon monoxide or the exposure to high doses of radiation.[6] The fact that we do not appear to have the physiologic apparatus to detect this type of stimulus is probably a reflection of our ancestral environment; the need to evolve the means to detect carbon monoxide or high dosages of radiation has only recently emerged.[7]

Acute Pain Differs from Chronic Pain

Appreciating the difference between acute and chronic pain is the second key issue. It is clinically beneficial to decide whether the pain has a useful or "adaptive" purpose, or is of no value—i.e., "maladaptive." Classically, pain is divided into acute adaptive pain and chronic maladaptive pain, but some caution is warranted. For instance, many chronic pains may be advantageous in that they protect weakened or diseased tissues that are incapable of complete recovery. A good example of this is osteoarthritis or rheumatoid arthritis affecting the metacarpal joints or the carpometacarpal joints at the thumb. A degree of pain, especially the more intense type of pain that is produced when a chronically weakened tissue is mechanically threatened, may be necessary for the protection and health of the tissues concerned. Problems arise for therapists and patients when all ongoing pains, whatever their quality, "irritability," and behavior, are viewed as the result of tissue damage. This is especially the case where no apparent disease process or injury seems to be involved.[8]

Most acute pains are seen as being the result of physiologic events that serve a very clear biologic purpose: to call on bodily adaptive measures to stop the pain and protect the injured tissues. It is useful for healing as the inputs derived from damaged tissues help to mobilize the bodies' protective reactions as well as the bodies' healing systems. It may be conceptually better to view nociceptive messages from damaged tissues as having two roles—first, to inform subconscious brain systems in order to promote a coordinated physiologic healing response, and, second, to inform consciousness via the medium of "pain" in order to change behavior. If the conscious and unconscious parts of the brain are otherwise occupied, for example, with survival or are focused on something very enjoyable, they are likely to take little notice until later, when it is safe or the focus of attention changes to less valuable things.

TABLE 1. **Pathobiologic Pain Mechanisms**

Nociceptive
Peripheral neurogenic
Central
Affective
Motor/autonomic

Most chronic pain can be regarded as a neurologic disease state[9] that as yet cannot be quantified in terms of hard anatomic data. Its primary mechanism is currently thought to be a result of altered processing occurring in the central nervous system.[10] Many patients with maladaptive pain states may present with tender joints, tendons, muscles, and abnormally reactive peripheral nerves, resulting from remote sub-microscopic aberrations and altered processing in the CNS rather than local tissue disruption. The easiest introduction to the massive literature on this subject is Melzack and Wall's classic *The Challenge of Pain.*[11]

Pain Is a Multidimensional Experience

To help integrate pain concepts into the clinic, pain needs to be considered as three interacting dimensions: sensory-discriminative, cognitive-evaluative, and motivational-affective.[12,13] The sensory dimension is the awareness of the intensity, location, quality, and behavior of pain. The cognitive dimension relates to thoughts about the problem, influenced by experiences and previous knowledge. Finally, affective is the emotional response, usually negative, that motivates or governs responses to the pain (e.g., fear, anxiety, or anger).[14] All dimensions are essential parts of all pain experiences, and all dimensions interact to produce altered physiologic outputs and ultimately altered behavior. For example, negative thoughts about an injury and pain may arouse negative emotions, which may then arouse the autonomic and neuroendocrine response systems, potentially impacting again on the sensory system. Simultaneously, altered levels of activity and specific or general altered movement patterns occur under the influence of subconscious reflex activity and conscious processing of the pain experience.

The pathobiologic mechanisms that cause this multidimensional pain experience are (1) nociceptive from target tissues of nerve (e.g., muscle, ligament, bone, tendon, and fascia; (2) peripheral neurogenic (from neural tissue "outside" the dorsal and medullary horns—e.g., nerve root and in the hand the distal branches of the median, ulnar, and radial nerve trunks); (3) central (from the CNS); (4) affective (from central pathways and circuits related to emotions and their perception); and (5) some pain states are strongly influenced by brain output systems, in particular the somatic-motor system[15] and the autonomic system,[16] but the neuroendocrine and immune systems must also be considered (Table 1). In our view, hand therapists

should make reasoned attempts to diagnose pain in terms of these pathobiologic categories.

Pain Mechanisms Move

The pathobiologic mechanisms driving pain alter and shift with time. This is a natural event occurring in all pain states. Consider for example a patient who has an acute wrist sprain. Skilled manual and imaging examinations reveal no grossly significant damage. The primary pain mechanism is nociceptive; thus, pain driven from the periphery. However, if this patient returns 1 year later still complaining of pain, the structures in the hand are likely to have healed to the best of their ability. The pathobiology is unlikely to be dominated by nociceptive pain mechanisms but probably resides deeper in the circuitry and processing of the CNS. Ongoing pain states are often of great concern to the patient, especially if the problem has not been validated or has been unsuccessfully managed by clinicians.[17] In these often common circumstances, the patient is likely to have many unhelpful or maladaptive thoughts (cognitions) and feelings about their pain that, as already argued, are likely to further add to it. The cognitive and emotional dimensions of pain generally get more involved the longer a problem persists; thus, affective "mechanisms" play an ever-increasing part in the presentation. For instance, witness how quickly a subjective examination takes for a relatively acute problem and compare this with the lengthy talking and discussing that can occur when a patient with a chronic problem is first assessed.

All Pain Is in the Head

This is not meant in the derogatory sense that is so often applied to patients whose injuries do not heal in the expected time frame of current medical models. It is meant in the sense of an appreciation that all pain is ultimately in the brain.[18] A painful experience does not only involve the firing of high-threshold A beta or C fibers. All a general anaesthetic does is take away an awareness of pain. These high threshold fibers will still fire. Pain depends on the excitatory and inhibitory currents in operation in higher level neurones at the time of stimulation. Patients with severe hand trauma will often say the injury did not hurt at the time the injury occurred. This points to powerful control systems in the CNS (see below).

CATEGORIES OF PAIN MECHANISMS

The categories of pain mechanisms are nociceptive, peripheral neurogenic, central, affective/cognitive, and autonomic/motor mechanisms. Each term relates to a physiologic/pathophysiologic process that can give rise to pain in sensory, cognitive, and emotional dimensions. Even though a patient may complain of pain solely in the hand, any or all of these mechanisms may contribute.

Unfortunately, there is no device or test that allows a therapist to decide instantly which particular pain mechanism is dominant. This requires careful history taking and analysis of the patterns and behavior of pain. For example, pain's geography, provoking factors, what the patient thinks and does about the pain, and responses to treatment are what allow for educated decision making. Hand therapists are in an excellent position to make these judgments because they see their patients over many treatment sessions, have time to talk to their patients in detail, and have the license to touch and move patients. No other profession dealing with hand pain will have such an involvement with the patient and their problems.

Nociceptive Pain—Pain Originating from Target Tissues Origin

This is pain at its easiest to understand. It is broadly the result of mechanical and physiologic processes in injured tissues that stimulate high-threshold primary afferent C and A delta fibers.[19,20] Nociceptive pain is generally linked to the occurrence of injury, inflammation, and repair. It frequently has a clear stimulus/response relationship. For example, if a stiff wrist postimmobilization is stretched, then this will usually elicit pain. Pain will increase proportionally to the force applied and will settle down when forces are removed. In the presence of repeated noxious stimulation, chemicals released by tissue damage or due to an ischemic state will cause nerve endings to dynamically lower their thresholds for firing.[21]

Nociceptive pain primarily relates to acute pain. It may be present in adaptive chronic pain states where poorly conditioned tissues need protection from potentially damaging movements and forces, or in disease states that maintain abnormal tissue biology, such as inflammation. Rheumatoid and osteoarthritis are examples of disease states that produce nociceptive pain in musculoskeletal tissues.

The nervous system via high threshold afferent fibers is also involved in the production and release of inflammation-enhancing chemicals. Their release occurs as a result of impulses passing back down afferent fibers in the "wrong" direction (antidromic impulses). These are thought to arise from the dorsal horn in the CNS[22,23] or sites of neuropathy in peripheral nerve.[24,25] The sympathetic efferent fibers are also known to secrete chemicals involved in the inflammatory process.[21,26]

The result of lowering of the firing threshold of C and A delta fibers is that tissues become tender to palpation and sensitive to normally innocuous movements. Spontaneous firing of nociceptors is thought to give rise to the ongoing background aching sensation that often accompanies many tissue injuries.[26]

This tenderness or hypersensitivity arising from changes in the damaged local hand tissues is referred to as *primary hyperalgesia.* However, tenderness often spreads after local injury and on ex-

amination appears to be in tissues that were not originally damaged. This is referred to as *secondary hyperalgesia.* This may be due in part to a local seeping of pain-producing chemicals through tissues, but there is now strong evidence that the spread of tenderness and sensitivity is much more related to changes in spinal and supraspinal circuitry (central sensitization).[26–28] It is important to understand that secondary hyperalgesia is abnormal mechanical sensitivity of tissues that are fundamentally quite normal. This is likely to cause many false positive results with physical examination of musculoskeletal and neural tissues.[29]

Nociceptive Pain Patterns

Fundamentally, this is an easy pain pattern to identify. Nociceptive pain usually eases as the injury settles. It improves naturally or in response to various treatments, such as ice, anti-inflammatory and analgesic medications, splinting, and graded recovery of range of movement (ROM) and strength. It will also help recovery if any anxiety created by the pain and the painful event is decreased. It may be beneficial to consider three kinds of nociceptive pain, although overlap is certain (Table 2). With mechanical nociceptive pain, nerve endings may be mechanically distorted by scar tissue or abnormal pressures from tissues. Movement increases distortion of nerve endings, causing increased pain. This is closely related to ischemic and inflammatory pain. Ischemic nociceptive pain occurs as a result of ischemia altering the physical and chemical environment of tissues. This results in increased excitation and sensitization of nociceptors.[30,31] Ischemic tissues become more acidic, contain less oxygen (hypoxic), and are rich in chemicals such as bradykinin, potassium ions, and prostaglandins; all are known nociceptor activity enhancers.[32] Ischemia is a particularly potent contribution to painful contracting muscles that have a poor or inadequate blood supply. Additional consideration should also be given to collagenous tissues (e.g., ligaments and tendons) that may be deprived of essential circulation by continuous stretching or compression by sustained postures in concert with a sluggish general circulation. Keyboard operators who maintain their joints in one position for long periods, use hand and forearm muscles at high rates, and sustain shoulder and neck postures may be prone to inadequate perfusion, ischemia, and hence slowly developing discomfort. Acute nociceptive pains that are more mechanical or ischemic in origin tend to ease quickly when the abnormal forces are removed. Persistent provocation over time may confuse this simple on/off presentation as nociceptors become increasingly sensitized by the ischemically related chemical soup, and central mechanisms begin to play a greater role.

Inflammatory nociceptive pain relates to inflammatory processes in the injured or diseased tissues.[21] Afferent fibers, in particular the high threshold fibers, change their response properties during inflammation. The threshold for firing decreases, and some fire spontaneously. Silent nociceptors awaken and begin firing.[33] A link to stimulus still exists, but now just a small amount of movement or gentle pressure may evoke pain that takes some time to settle. A background ache, thought to be due to the spontaneous firing of large numbers of normally quiescent nociceptors, is often present. Some patients who are more concerned about sharp pains on movement may deny the background ache or feel that its presence is irrelevant to the problem. Patients with inflammatory nociceptive pain often feel worse in the morning, report morning stiffness, and may respond favorably to anti-inflammatory medication. Gentle passive or active movement often helps relieve discomfort and feelings of stiffness. One proposed tissue mechanism for this is that movement may ease mechanical pressures caused by inflammatory fluids. In comparison, too vigorous a movement will increase pain, but often not until the next morning.[34]

TABLE 2. **Three Types of Nociceptive Pain**

Mechanical nociceptive
Ischemic nociceptive
Inflammatory nociceptive

Peripheral Neurogenic Pain—Pain from Peripheral Neural Tissue Origin

Hand therapists are often confronted with peripheral nerve injuries in the hand, particularly following trauma. Injuries such as laceration, compression, overstretch, and/or the persistent presence of inflammatory fluids may create a neuropathy perverting impulse transmission or, worse, initiating impulses from the injury site on the nerve. The axolemma of peripheral nerves are designed for impulse transmission, not generation. For pain to arise mid-axon, the number and sensitivity of ion channels in the axolemma at the injury site must increase.[35] The injured or altered sites are known as abnormal impulse generator sites (AIGS). Pathologic changes allowing ion channel pooling and sensitivity changes within an AIGS include demyelination and neuroma development. Alpha adrenoreceptor expression on injured axolemma may also occur after injury, allowing depolarization from stress-induced circulating chemicals (epinephrine and norepinephrine) or raising the nerve's generator potential, allowing mechanical and thermal hyperalgesia. Ephaptic synapsing ("crosstalk") may also occur.[35] For the clinician, this means that a segment of nerve can become a source of pain; which may be evoked by mechanical forces, catecholamines, or metabolic changes such as ischemia. Some injury sites may fire spontaneously.[35] In response to the neuropathy, neuronal function farther along the nerve, especially in the dorsal root ganglion and in the spinal cord, may be altered, probably due to ion channel number and sensitivity changes.[35,36] Note that, if a nerve servicing a painful

TABLE 3. **Symptoms and Signs Possibly Indicating Peripheral Neurogenic Pain**

Peripheral cutaneous or segmental distribution
Corresponding motor deficits
Mechanically evoked by nerve compression and/or tension
Made worse by negative emotional states
Deep aching, cramping
Superficial burning, stinging, and paraesthesia—often easy to localize
Ongoing, often difficult to ease for any length of time with rest or medication
"A mind of its own" pain behavior

tissue is cut, it does not mean the abolishment of pain. This dramatic alteration of input to the higher neurons may actually increase pain, since the cut ends of nerves can themselves become ectopic impulse generators.[11]

Many factors, such as the health of the neural connective tissues, endoneurial fluid pressure, axonal transport systems, and the quality of the vasa nervorum can influence impulse conduction.[37,38] The epineurium, perineurium, and endoneurium of peripheral neural tissues are highly innervated and reactive connective tissues and quite probable sources of symptoms in neuropathies.[39-42]

Peripheral Neurogenic Pain Patterns

Before listing patterns, therapists should be aware that some people sustain nerve injuries such as nerve entrapment or nerve root compression and may never complain of pain,[43,44] probably due to the regulatory influences of the CNS. Neural connective tissue injury or irritation should really be considered as a unique category of nociceptive pain, which may be responsible for more familiar and perhaps better localized pains.[45] The following features (reviewed in Table 3) may help identify peripheral neurogenic pain. Symptoms are within all or part of the innervation field. They may be common to a local cutaneous nerve or, in the case of the nerve root, within its dermatome, myotome, or sclerotome. The quality of symptoms may be influenced by the type of tissue innervated by the damaged fibers.[46] Thus, fibers that normally innervate muscle may give rise to deep aching or cramping pain, and fibers to skin may produce superficial burning, paresthesia or stinging sensations. Tests that mechanically influence nerve, such as a neurodynamic test or muscle contraction, and sustained postures that compress or stretch a reactive nerve may evoke a variety of pain symptoms.[35] These may be a short burst of pain that ends before the stimulus is removed, symptoms remaining for the period the stimuli is present, or symptoms that continue after the physical stimulus has been removed. There may also be a slow build-up of symptoms during the application of the stimulus, which may subside slowly once the stimulus is removed.

In contrast, some neuropathies fire spontaneously, and the pain may appear to "have a mind of its own" to the patient. Ectopic impulse-generator sites are known to be capable of spontaneous "pacemaker" activity in addition to becoming sensitive to circulating chemicals like epinephrine and norepinephrine.[35] Thus, some patients may volunteer that mental stress makes their problem worse. Small changes in local temperature or ischemic changes are also known to influence ectopic impulse-generator site activity and hence produce pain for no apparent "physical" reason.[47]

A peripheral neuropathy is likely to have effects elsewhere in the body, and spread of pain from an original injury is common. One neuropathy may predispose the rest of the nerve to injury and thus second neuropathies, known as "double crush."[48,49] Altered chemical signals from a neuropathy may alter output from the cell body, causing noradrenergic sprouting in the dorsal root ganglia.[36] A peripheral neuropathy may also contribute to an inflammatory nociceptive pain by creating antidromic impulses and the release of neurotransmitters such as substance P, which have a pro-inflammatory effect in target tissues.[50] Finally, a nerve injury continually injects abnormal trains of impulses and abnormal levels of transmitter chemicals into the CNS. This may have devastating effects on the subtly controlled balances of inhibition and excitation needed for normal sensory processing. Changes in synaptic efficacy, death of some inhibitory interneurons, and even structural reorganization of nerve cells and their connections are known to occur in the dorsal horn after peripheral nerve injury.[51,11] This could result in a long-standing, possibly permanent rewiring, allowing normal innocuous impulse traffic from uninjured tissues to excite ascending neural pathways linked to the sensation of pain.[52] Pennisi[53] has provided a summary of this process. Clinically, this translates into acute tenderness and mechanical sensitivity of tissues (secondary hyperalgesia) that may be physiologically normal but are often thought to be culpable for ongoing pain. Sadly, these patients may be subjected to inappropriate surgery and/or therapy directed at innocent tissues.

Central Sensitization—Pain Related to Altered CNS Circuitry and Processing

The dorsal horn is the first collection of neural tissues that houses a variable response potential to input. Far more than a simple relay station, this small piece of neural tissue contains complex circuitry with a variety of neurons, neurotransmitters, and receptors. This circuitry is controlled by excitatory and inhibitory influences from the brain, the periphery and spinal and segmental neurons.[11] While it is only a part of a much greater CNS response system, recent research involving dorsal horn cells has provided an indication of the enormous plastic potential of CNS circuitry.[54]

CNS cells change their response properties when subjected to high threshold input (i.e., nociceptor input). While an upregulation (increase) of CNS sensitivity is of great adaptive value to pro-

mote protective motor activity and healing behavior, sometimes this enhanced excitability state persists long after peripheral tissues have healed to the best of their abilities, and dominant sources of pain shift to the CNS. Changes in central sensitivity result from a complex cascade of events that originally relates to amino acid and neuropeptide driven sensitivity controlled by local and supra-spinal inhibitory neurons.[55] Later processes, perhaps irreversible, may involve local inhibitory neuron death,[56] new and inappropriate synapsing,[57] and cell membrane changes, allowing cells that under normal conditions only respond to nociceptive inputs to respond to inputs that are innocuous.[58] In addition these cells respond far more, for much longer and increase their receptive field (area of tissue which can excite a neuron). Cells therefore change their response properties. While this dorsal horn sensitivity may be maintained by a "trickle" of small fiber afferent input from the periphery, it may persist even when the original injury has healed.[51,57] Persistent cognitions that for instance focus on the injury are also "inputs" that may maintain the dorsal horn cells' excitability by lifting descending inhibitory currents from the brain that normally prevent undue increases in sensitivity. Dorsal horn cells that are in an enhanced sensitivity state may produce spontaneous bursts of impulses not unlike those produced by the ectopic impulse generators found in damaged peripheral neurons.

Although most studies on central sensitization have focused on the dorsal horn, similar concepts may relate to the rest of the nervous system. It may well be that pain states become imprinted in unique CNS pathways in ways not unlike those thought to produce memory.[59-62] If this is tenable, the rather sobering message is that once "pain" is imprinted it may be as hard to remove as most of our memories are. They key thing about memories, and the hopeful aspect of this analogy in relation to pain, is that we can seem to hold them in subconscious filing cabinets, emerging into consciousness only when specific cues are called upon. Many chronic pain sufferers can be taught to focus less on their pain and more on recovery of function once they have a better understanding of the pain mechanisms involved.[63,64] Like any other therapists dealing with ongoing pain states, hand therapists must integrate aspects of management strategies being promoted by the cognitive–behavioral approach to chronic pain if they are going to adequately address chronic pain conditions.[65] A key message is that it is far better to focus on slow and progressive functional recovery with a clear patient understanding of maladaptive pain than it is to focus therapy on pain relief before recovery of function.

Prolonged and maladaptive central sensitization is perhaps the biggest challenge for hand therapists to integrate. It is a dangerous state for the patient. Therapists' doubt creeps in as to the validity of the experience. This is compounded by the fact that there is no test, either imaging or biochemical, that can identify this subtle state of affairs. In this pain state, treatments such as unnecessary surgery, injection, and forceful manipulation are unwarranted, often leading to dashed hopes for an often gullible patient in the presence of a convincing practitioner.[66,67] Pain evoked by wrist-provocative tests or patient-demonstrated tenderness about the hand may have nothing to do with the local tissues. It may simply be quite normal inputs which are wrongfully processed by the CNS to be perceived as noxious. Unfortunately, those who want to persist with the hand to find the source of pain will usually find something. For those with an understanding of central mechanisms, it should be debatable whether the finding is relevant.

No discussion on central mechanisms can be complete without reference to the powerful supraspinal control systems.[68,69] These have projections from many parts of the brain, including the cortex and diencephalic systems, the periaqueductal grey and periventricular grey, the rostroventral medulla (NRM), and the spinal and medullary dorsal horns. These are powerful tonic systems that are generally inhibitory; however, with the remarkable plasticity of the CNS, various inputs can lift these inhibitory currents, or destructive life events may weaken these pain control systems.

Pattern of Centralization of Pain

These patterns (Table 4) are only an assumption based on the overall picture of clinical findings linked to current experimental work. Thus, they are not pathognomic, but rather patterns suggestive of central sensitization. It should be noted that nociceptive and peripheral neurogenic pain mechanisms also have some of the following features, too. It is the overall presentation that is important.

Pain may be ongoing after tissues have had time to heal. Therapists may consider the symptoms weird and wholly inappropriate to the history. There may be no familiar anatomic "textbook" patterns to the symptoms. Evaluation reveals excessive sensitivity to inputs that would not normally provoke pain, yet the tissues under scrutiny seem to be healthy (secondary hyperalgesia). There is rarely a physical test that does not hurt in some way, and rarely a test where the patient reports an improvement in symptoms. Thus, everything may hurt: ligament tests, muscle tests, instability tests, and neural tension tests. In some instances, movements and

TABLE 4. **Symptoms and Signs Possibly Indicating Central Sensitization**

Ongoing pain after expected tissue healing time
Unfamiliar anatomic pain patterns
Secondary allodynia and hyperalgesia
Latency to input
Atypical pain behaviors
Pain has "a mind of its own"
Exacerbated by emotional and physical stress
Often significant affective and cognitive components
Variable responses to passive treatment
Poor response to medication, even opioids

activities can be non-painful at the time they are performed but produce a reactive latent response. Occasionally grossly abnormal movement patterns, (which may be related to fear of pain or a need to demonstrate the pain) are displayed; other patients prefer to keep silent. Symptoms may be more intense under stressful situations and, similar to some peripheral neurogenic pain, often are described as having "a mind of their own." Patients may provide histories of psychologically traumatic events that weaken the patient's overall coping capacity and be significant to ongoing pain states. Some studies of populations of chronic pain sufferers have noted increased incidences of childhood physical and sexual abuse, abandonment, and emotional neglect and abuse.[70,71] Factors such as dislike of work[72] and heightened anxiety and frustration[73] are a few of the many constellations of features that can influence the patient's recovery, levels of distress, and suffering.[74] Finally, responses to passive treatments are quite variable. A specific treatment may vastly improve symptoms on one occasion, yet the same treatment performed another time may exacerbate the symptoms.

Affective Mechanisms

For some hand therapists, the domain of the brain—emotion and cognition—can sometimes seem a long way away, while for others it is considered the province of other professions. This must be overcome, especially if the profession is to make any impact on the chronic, ongoing problems where maladaptive thoughts and emotions are very dominant and to make useful interprofessional links. We must come to terms with the fact that the way we think and feel has vast repercussions on the brain processing that orchestrates the sensations we perceive as well as on the health and vitality of the body's physiology.[14] For instance, there is now vast literature on the influence of mental stress on disease states via the sympathetic, neuroendocrine, and immune system pathways.[75] If we can favorably alter the way people think and feel about their disorders, we can greatly enhance the rehabilitative process of functional restoration and perhaps the bodily recovery processes. Future therapies must recognize and integrate the multidimensional nature of pain in every patient every time.

This affective mechanism, viewed in isolation, is seen as pain arising purely as a result of emotional turmoil. This is a much-debated and disputed area.[76] The safe stance is to accept that emotion or affect is a dimension of the pain experience as well as a possible mechanism of it. Thus, pain is more likely to cause emotional disturbance than be a precipitator of it. Having said that, it is conspicuous from the stress literature that emotional disturbance has detrimental effects on the health of the body.[77,78] Referring to the IASP pain definition, therapists should consider the importance of inputs like loneliness, hopelessness, sadness, fear, anger, and frustration that are caused by a pain state and may also serve to drive it farther.

Autonomic and Motor Mechanisms

The sympathetic nervous system (SNS) is involved in all injuries and pain states. It is key in promoting general survival to any threat physically or mentally registered via the "fight or flight" response and in providing more focused circulatory and chemical alterations in the environment of damaged or abnormal tissues. The way we think and feel powerfully influences sympathetic activity and therefore the levels of the circulating catecholamines, epinephrine and norepinephrine.[78] That the SNS is made blameworthy for pain states is due to the successful alleviation of symptoms following sympathetic block techniques.[79,80] However, there are three considerations. First, there are many patients who appear to have sympathetically maintained pain symptoms that do not respond to sympathetic block techniques. Second, ongoing pain states have multiple mechanisms, and all aspects of ongoing pain need consideration; the importance of the SNS may have been overemphasized. Finally, the weight of modern evidence suggests that where a disorder has a component of SMP it is not so much the fault of an abnormal sympathetic system but that abnormal sensitivity to *normal* sympathetic secretions occurs in injured nerves and peripheral nociceptor terminals.[81]

Hand therapists, unfortunately, confront one of the most challenging of human conditions—reflex sympathetic dystrophy (RSD). Recent pain sciences investigations have challenged the dominance given to the SNS in the RSD construction. "RSD" is now included as part of the complex regional pain syndrome (CRPS).[82] This is useful because it challenges the focus of diagnosis on one element of a very complex and disabling pain state. It also encourages a re-examination of the role of the SNS in pain states generally. Hand therapists are urged to review the current debate in pain sciences.[81]

The key message for hand therapists is that no matter what they do to the patient they will be influencing the SNS. Thus, the activity of this important but relatively primitive system largely depends on how a patient perceives the situation.[83] Graduated strengthening and return of ROM and function may be the traditional goals of hand therapy. However, decreasing catecholamine levels by ameliorating anxiety, fear, and frustration and using relaxation techniques may also be a vital aspect of management. In chronic pain, the educative process may be more important for a beneficial outcome than actual passive physical maneuvering. It is well worth explaining to the patient the relationship of negative/unhelpful thoughts and emotions to increased tissue sensitivity to catecholamines. Not only does this give patients sound reasons for odd pain behavior, it also encourages them to make use of cognitive and behavioral psychologic strategies and relaxation techniques in their rehabilitation.[84]

The other major output-related pain mechanism is via the motor system. Injured muscle is a potent site of nociceptive pain; ongoing tension in muscles from ongoing pain may provide a provoc-

ative environment for further enhancing symptoms.[85]

A REASONING MODEL TO BRING IT TOGETHER

We suggest that to integrate pain in the clinic, the clinician must have clinical reasoning skills.[86] The clinician must not accept a particular recipe or protocol for each patient but must accept that a patient's pain and the resultant emotions and thoughts about it are unique. No one pain pattern will be the same, and no two people would react and think the same way about it (Table 5).

The key part of a reasoning strategy is to recognize that therapists must make decisions based on information collected in a number of categories. Information is needed in all these categories for the best understanding of the problem and hence the best management process.

The first is pathobiologic mechanisms. Hand therapists must attempt to identify the predominant pain mechanisms in operation. This must be in addition to their existing reasoning skills related to tissue mechanisms (tissue health, stage of healing, etc.). Just as knowledge of healing stages provides information about prognosis, precautions, and management, so does the pathobiologic pain state. Next, dysfunctions are the clinical expressions of the pathobiology which the therapist finds on examination of the patient. A key reasoning issue is the relevance of the finding. For example, a few stiff joints may be of little relevance to a patient with a long-standing central mechanism-related hyperalgesic state. Attempts to "loosen the joints up" may simply be an additional input into the system that the body is unable to handle and that additionally serves to focus the patient on tissue abnormalities and weaknesses that need therapeutic "fixing."

Dysfunction can be general—difficulty with writing, grasping, or performing a particular task. It can be specific—weak lumbricals, a restricted median nerve, or an unstable scaphoid. There may also be mental/psychologic dysfunction, recognizing maladaptive thoughts, beliefs, and emotions about the pain. Examples may be refusal to use a hand due to fear of a tendon snapping and continued dependence on a splint. Within limits, and in tandem with other therapies if required, hand therapists can deal with modest mental/psychologic dysfunction. Good therapists have always done this. A simple explanation and the reassurance that follows along with some hands-on demonstrations may be all that is required to move unhelpful thoughts and feelings in a more adaptive direction. Some dysfunctions, however, are clearly necessary and adaptive. Others may be maladaptive and deeply entrenched and may need broad or focused therapeutic attention from trained clinical psychologists and appropriate medical specialists.

The third category is sources. These could be defined as the site at which therapeutic intervention should be theoretically targeted. Dominant sources of a pain mechanism may be in a tendon sheath in nociceptive pain. With central sensitization, the sources may be widely dispersed throughout the CNS; for example, in the dorsal horn, reticular formation, and cerebrum. We hope it is clear that, the more complex and chronic a problem, the more complex and diverse the pain mechanisms; therefore, the more futile it is to direct intervention at a specific target "source." The location of the pain and enhanced sensitivity does not necessarily site the underlying mechanism.

TABLE 5. **Clinical Reasoning Hypothesis Categories**

Pathobiologic mechanisms
Tissue mechanisms
Pain mechanisms
Nociception
Peripheral neurogenic
Central
Affective
Sympathetic/motor
Dysfunction
General
Specific
Psychologic/mental
Sources
Contributing factors
Prognosis
Precautions
Management

Prognosis is a crucial reasoning category that hand therapists should contribute to, rather than leaving it to the sole discretion of the patient or the referring physician. Integration of pain science and the pain mechanisms discussed empowers this category of decision making. It can be reasonably easy to make a prognosis for nociceptive pain, and of course this may be dictated by the specific dysfunction present. Peripheral neurogenic and central pain can be quite difficult to prognosticate. Sometimes dramatic changes can occur with explanation and validation of pain, mastery of tasks, and changes in fitness. The patients perceive they are gaining greater control of their problems. Therapists must consider in many cases that pain may be unalterable, but the patient's suffering and disability may be vastly diminished. Prognosis can be reasoned in terms of pain, the likely pain mechanisms operating, and in terms of functional recovery by reflecting on the mechanisms of pain and the findings of the examination of physical and mental dysfunction. The potential for functional recovery is often extremely good when the potential for pain resolution is extremely poor.

Contributing factors relate to predisposing features and factors relating to the development and maintenance of the current problem. These include familial and ergonomic factors, past physical and mental traumas, and the patient's underlying physical and mental ability to cope. Hand therapists should be aware that adequate explanation of therapy, explanation of the problem, and attention to anxiety,[87] job satisfaction,[88] and fear of pain[89] are contributing factors. These have been shown to af-

fect outcomes of therapeutic intervention to spinal pain. It should be no different for pain in the hand.

Management requires hand therapists to take on pain in all its dimensions and the new opportunities it brings. It is a professionally empowering necessity. Pain provides a common link with many medically related professions. It provides a common language, it stimulates research, and it must direct therapy. As always, outcome studies are needed, but for the patients who cannot wait there is an overwhelming scientific revolution that powerfully indicates that we must begin to change our ways now, before current therapies are either embarrassing or are rejected. These are exciting times. Our role is to make sure they are exciting for patients as well.

SUMMARY

A scientific revolution focusing on the phenomenon of pain has been occurring for three decades. Hand therapists are urged to use this revolution in their clinics. In particular, they are urged to consider the clinical decision-making consequences of attempting to diagnose pain, rather than continuing common practices of accepting pain as a mechanical event solely related to peripheral tissues.

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