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THE PATIENT'S BILL OF RIGHTS: A PROGRESS REPORT

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In Vol. 4, Issue 2 (Spring 2001) of the RX Law & Medicine Report, this author provided some background on the status and history of a federal patient's bill of rights. On June 29, 2001, the United States Senate passed the bill § 1052, as amended, also known as the "Bipartisan Patient Protection Act" of 2001. The Senate passed the bill by 59–36 votes, which included all Democrats and 9 Republicans in favor of it. Its chief sponsors were Senators McCain, Edwards and Kennedy. The alternative to § 1052 was a bill sponsored by Senators Frist, Breaux and Jeffords. Before passage, there were ten days of debate, often contentious and partisan. The most contentious of issues were liability and employers' exposure to lawsuit. The latter was resolved by passage of an amendment that limited employers' exposure to lawsuits. In lay terms, if the employer designate a "designated decision-maker" who accepts responsibility for that employer's decision-making, if any, regarding the care and treatment of any of its employees and that decision-maker is financially solvent to the extent of satisfying any claim or judgment, the employer will not be exposed to liability. The other major points made by opponents of § 1052 was that it (§ 1052) would open the floodgates for new litigation, and that any such litigation should remain in federal courts. Those points failed to carry the day.

The patient's bill of rights, as of the writing of this article (July, 2001) heads to the House of Representatives for debate and resolution. The legislation that is comparable to § 1052 is HR 526, which is chiefly sponsored by Reps. Ganske, Dingell and Norwood. What the Senate passed, as amended, was going to be studied to see whether HR 526 could conform itself to § 1052, as amended. Also, Rep. Fletcher introduced another version of the patient's bill

[47] Ferguson v. City of Charleston, 2001 U.S. LEXIS 2460, at *27 (2001).
[48] Ferguson v. City of Charleston, 2001 U.S. LEXIS 2460, at *28 (2001).
[49] Ferguson v. City of Charleston, 2001 U.S. LEXIS 2460, at *28–29 (2001).
[50] Ferguson v. City of Charleston, 2001 U.S. LEXIS 2460, at *29 (2001).
[51] Ferguson v. City of Charleston, 2001 U.S. LEXIS 2460, at *29–30 (2001).
[52] Ferguson v. City of Charleston, 2001 U.S. LEXIS 2460, at *30 (2001).
[53] Ferguson v. City of Charleston, 2001 U.S. LEXIS 2460, at *32 n. 23 (2001).
[54] Ferguson v. City of Charleston, 2001 U.S. LEXIS 2460, at *32 (2001) (*citing* Miranda v. Arizona, 384 U.S. 436, 16 L. Ed. 2d 694, 86 S. Ct. 1602 (1966)).
[55] Ferguson v. City of Charleston, 2001 U.S. LEXIS 2460, at *39 (2001).
[56] Ferguson v. City of Charleston, 2001 U.S. LEXIS 2460, at *39 (2001).
[57] Ferguson v. City of Charleston, 2001 U.S. LEXIS 2460, at *38–*39 (2001).
[58] Ferguson v. City of Charleston, 2001 U.S. LEXIS 2460, at *42 (2001).
[59] Ferguson v. City of Charleston, 2001 U.S. LEXIS 2460, at *43 (2001).
[60] Ferguson v. City of Charleston, 2001 U.S. LEXIS 2460, at *45 (2001).
[61] Ferguson v. City of Charleston, 2001 U.S. LEXIS 2460, at *45 (2001).
[62] Ferguson v. City of Charleston, 2001 U.S. LEXIS 2460, at *46 (2001).
[63] Ferguson v. City of Charleston, 2001 U.S. LEXIS 2460, at *51 (2001).
[64] Ferguson v. City of Charleston, 2001 U.S. LEXIS 2460, at *53 (2001).
[65] Ferguson v. City of Charleston, 2001 U.S. LEXIS 2460, at *53 (2001).
[66] Ferguson v. City of Charleston, 2001 U.S. LEXIS 2460, at *53 (2001).
[67] Gunshot or stab wounds; evidence of battery.
[68] Infectious diseases such as salmonella and various sexually transmitted diseases.

PAIN ASSESSMENT, EVALUATION AND NEW TREATMENTS

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Perhaps there is no other singular daunting memorable experience than the event of pain. Therefore, pain has distinct neurochemical links to our daily lives, learning, behavior and to memory. A child remembers well the event of pain after burning a finger, falling down, a bee sting or electric shock. Certainly, childbirth can be associated with pain. However, pain is also a phenomenon. Amputees often report of pain in a limb (phantom limb pain) even after it has been severed. Pain can be either chemically based, the result of impact from an injury, or a totally mental experience based on memory, or the emotions without the afferent or efferent signaling pathways. It is either at the level of the tissues, psychosomatic, or "in the mind," however, essentially, all pain phenomena exists at some point in time at the level of the neuron or cell body of the peripheral or central nervous system, or anatomical structures that link pain to memory centers such as the hippocampus and amygdala. (Portenoy, R.K. & Kanner, R.M. 1996).

The experience of pain is unique to the individual with personal, subjective sensory and extra-sensory components influenced by cultural learning, the meaning of the situation, attention, and other psychological variables. Apparently, pain does not immediately begin with stimulation of receptors but rather with neural signals that enter the brain evoking selection, abstraction, and synthesis of information from the sum total of the sensory input. (Melzack, R. & Wall, P.D. 1988).

Western Philosophy

It is difficult to find universal acceptance of a definition of pain in the medical and scientific community. Also, isolating the origin of pain is a challenge for the specialist (neurologist) or pain management team. The Western philosophy of pain currently identifies a group of afferent fibers located in the peripheral nervous system that respond to acute tissue damage. Once an action potential is signaled and a threshold achieved, the nociceptors

change their properties, some becoming sensitized and others less sensitized. Nociceptors (e.g., skin) are primarily afferent neurons that have the capacity to distinguish between innocuous and noxious events as the substrate of pain consciousness. (Perl, E.R. 1984).

However, other factors influence the perception of pain and sometimes become the major component of pain. Often, disturbances of somatosensory processing, independent of associated tissue damage result in psychological or neuropathic pains (e.g., trigeminal neuralgia, central pain, and painful polyneuropathy). A comprehensive assessment must list and categorize the possible influences that contribute—both neuropathic and psychological [affective]—to the patient's perception of pain and not to dismiss the perception of pain in the absence of determining any level of tissue damage. (Portenoy, R.K. & Kanner, R.M. 1996) (Melzack, R. & Katz, J. 1992).

Central Pain, Sympathetic Parasympathetic Peripheral

In the 1960's, Melzack and Wall (Melzack, R. & Wall, P.D. 1965) described the "gate control theory" of pain that emphasized the mechanisms in the central nervous system that mediated the perception of noxious stimuli and subsequently, synapse upstream afferent processes with downstream modulation from the brain. Their concept of nociceptive pain is detected by specialized transducers attached to primarily A delta and C fibers operating under a control mechanism in the dorsal horn of the spinal cord. The activity of the projection neurons in the dorsal horn of the spinal cord is regulated by segmental and supraspinal influences. (Portenoy, R.K. & Kanner, R.M. 1996). The large A-beta (fast) fibers closes the gate. The small, A-delta or C fiber opens the gate. The control mechanism is modulated by descending input.

The gate control theory has four components including:

1) Afferents-Nociceptive afferents appear to end in localized regions. The C afferents have two outer laminae and thought to transport a chemical message from the periphery about the condition of the tissue in which the fibers terminate in this area and also some extend to the mid dorsal horn lamina.
2) Six laminae arrange the cells that receive incoming afferents. It is proposed that deeper cells interpret many factors both from internal systems and from afferent patterns.
3) Descending controls appear to transmit signals faster and raphe nuclei and the reticular formation are thought to be origin of descending control (note that the raphe nuclei also produces serotonin) with inhibitory effects on the signaling of dorsal horn cells.
4) Synaptic decisions interpreted at the dorsal horn and trigeminal nuclei are collected by many types of cells.

Information about the presence of injury is collected probably by short chain polysynaptic systems emphasized by long running pathways that appear to involve the spinothalamic tract. (Noordenbos, W. 1959) (Wall, P.D. & Melzack, R. 1984) (Wall, P.D. 1978). The gate control theory, however, does not account for long-term changes in the central nervous system to noxious input or other dynamic processes that impact the individual. Nociception is now understood to be affected by an "inflammatory soup" that mediates the site of the tissue damage. Dubner and Ruda (Dubner, R. & Ruda, M.A. 1992) have shown that huge nociceptive input can have a permanent effect on spinal cord function due to toxic effects of amino acids, which moves acute pain into chronic pain episodes. Moreover, research has shown that plasticity, or learning, has an

effect on pain. Psychosomatic, sensory, physiological, neurochemical and deep psychological influences can result in a neuromatrix that the brain generates a dynamic perception of pain with concomitant stress to the condition of homeostasis. Experience of pain can be anticipated and lead to a wide range of both neuropathic and psychological responses that involve the release of cytokine, corticosteroids, and immune system factors existing at the injury site. The resulting experiences are either transient, acute, neuropathic or chronic pain. According to the International Association for the Study of Pain: "Pain is an unpleasant sensory and emotional experience which we primarily associate with tissue damage, or describe in terms of such damage, or both." (Loeser, J.D. & Melzack, R. 1999) (Mersky, H. & Bogduk, N. 1994) (Wall, P.D. 1978).

Eastern Philosophy

The eastern philosophy of pain centers around the model of Qi (Chi). Accordingly, there are three layers of the energetic interface between body and mind known as "Three Vital Treasures;" Jing, Qi, and Shen. Jing is the innermost layer, the map that holds the physical body to the desired form of health. Shen is the outer energetic layer of the body through which the mind orchestrates the activities of the body. Qi is the middle layer, the interface that allows communication between the energetic fields of the body Jing and Shen. Acupuncture is an ancient Chinese method of encouraging the body to promote natural healing and to improve functioning. Successful outcomes are achieved by inserting needles and applying heat or electrical stimulation at specific acupuncture sites. (Lee, R.H. 1998).

Acupuncture theory states that channels of energy travel in regular patterns throughout the body and across its surface. The energy channels are like rivers flowing through the body to irrigate and provide nutrients to tissues. Any obstruction of the energy flow is like a dam that backs up the flow of energy in a part of the body restricting it in others. The meridians can be influenced by impacting the acupuncture points with needles that, in turn, unblock the obstructions at the dams and reestablish the regular flow through the meridians. Acupuncture claims to improve metabolism, correct imbalances in digestion, absorption and in the circulation of energy throughout the body. ("Doctor, What's This Acupuncture All About?" A Brief Explanation for Patients from The American Academy of Medical Acupuncture, Los Angeles, CA).

Regardless of the absence of a universal theory of pain, the ultimate task is to understand the fundamental cause, origin, management and eventual control of pain.

Nonreceptive Pain Transmission

Nonreception is the activity signaled in neural pathways by possible tissue-damaging stimuli. The neurons are composed of primarily two types of axons, A-delta fibers (lightly myelinated), or C fibers (unmyelinated). Both types of nociceptors have subtypes that are known as mechanoreceptors that respond to heat, as well as noxious mechanical stimuli, such as pressure from trauma. (Fitzgerald, M. & Lynn, B. 1977). Some fibers only discharge after noxious stimulation. Others remain silent, responding only to innocuous stimuli but increases their transmission rates with the eventual tissue damage. (Perl, E.R. 1968). Primary afferent neurons-unmyelinated-that discharge in response to noxious thermal, mechanical and chemical stimuli (C polymodal nociceptors or C mechano-heat nociceptors) may also discharge with nonnoxious stimuli and increase their firing rate as stimulus intensity elevates. Some of the C fibers signal transmission only in response to tissue damage. Both A-delta and C fibers may become sensitized following exposure to noxious

stimuli. (LaMotte, C.C. & Campbell, J.N. 1978).

Afferent fibers have also been found to be responsive to noxious stimulation located in somatic tissues such as muscle, joints, cornea, periodontium, and tooth pulp. Innervation of the heart is mediated by nonreceptors that travel with sympathetic pathways. Intra-abdominal and pelvic viscera are innervated by nonreceptors that travel with sympathetic or parasympathetic nerves. Some nonreceptor afferents such as neurons that travel with the vagus nerve signal pathways of the larynx, airways and so-called J receptors. (Portenoy, R.K. & Kanner, R.M. 1996).

Signaling of nerve terminals from injury involves impulses in nerve fibers of all sizes to include the following:

A. A delta fibers (small myelinated fibers). A large electrical shock is needed to excite these fibers, which results in intense and prolonged action and facilitations of many spinal cord cells and intense pain. There are several physiological types and many are nonreceptors

B. A beta fibers (large myelinated fibers). Minor electrical stimulation of peripheral nerves selectively activates these fibers with resulting non-painful sensation. They are related to pain in four ways. Inhibition of responses of cord cells to noxious stimuli from afferent barrages decreases pain. The promulgation of the gate theory is the result of this observation.

C. C fibers (unmyelinated afferents). These fibers are primarily nonreceptive in nature with selective stimulation of these afferents producing pain. Research suggests that C fibers have additional function in response to injury.

D. Mechanism and activation of neuronal activity due to injury or tissue damage.

Primary:

1) Pressure excitation
2) Temperature excitation
3) Chemical excitation

Secondary:

1) Chemicals from nerves
2) Chemicals from cells
3) Enzyme products

Tertiary:

1) Invasion by cells and blood vessels
2) Invasion by sensory and sympathetic nerves
3) Transport in C fibers of abnormal chemicals (Wall, P.D. & Melzack, R. 1984).

Central Pathways

Pain transmission from the first cells in the spinal cord depends on five variables: 1) the arrival of nociceptive messages; 2) the threshold level affecting onset of time and intensity of an afferent barrage; 3) the convergent effect of other peripheral afferents with possible exaggeration or reduction of the effects of the nociceptive message; 4) the presence of control systems which influence the central cells in the CNS; and 5) the degree of interaction between nonreceptive afferent impulses and signaling of other peripheral events and general excitability of other CNS mechanisms. (Wall, P.D. & Melzack, R. 1984).

Inhibition of Pain

The dorsal horn of the spinal cord is the anatomical site for control of painful stimuli. The grey matter that contains cell bodies of efferent and internuncial neurons is the origin of the tracts of several inhibitory pathways of the midbrain that synapses at the medullary

raphae. Pain is controlled by the activation of analgesic circuits in the brain or spinal cord, or peripherally located white matter made up of nerve fibers and glia that inhibit neurochemical mediators such as prostaglandins. Inhibitory pathways work by modulating the release of opioids or endorphins, but also other neurochemicals are involved including norephinephrine and serotonin.

The analgesic pathway is innervated by the locus ceruleus, a tiny blue pigmented nucleus on the floor of the fourth ventricle which generates norepinephrine. Serotonin is produced in a number of cell bodies including the arcuate nucleus, paraventricular nucleus and raphe nuclei, where it originates in the midline of the brainstem. The highest volume is located in the nucleus raphe magnus located in the dorsal brain stem.

The inhibitory circuits of norepinephrine and serotonin (5-HT) end in the dorsal horn of the spinal cord. Dense populations of opioid, norepinephrine and serotonin receptors are located in cell bodies of this region. Pain control can be mediated by centrally acting agents by binding at one or more of these cell bodies. Note that the activity of serotonin and norepinephrine analgesic pathways is potentiated by blocking the re-uptake of NE and 5-HT into presynaptic neurons such as in the use of serotonin specific uptake inhibitors (SSRIs), such as Fluoxetine (Prozac) and Sertraline (Zoloft). Pharmaceuticals that interfere with the reuptake of serotonin and norepinephrine, such as tricyclic antidepressants (amitriptyline) (Elavil) are valuable compounds in neuropathy and other particular pain models.

Peripheral Analgesic Pathways

All cell bodies synthesize prostaglandins except red blood cells. Prostaglandins are released in response to tissue damage and inflammation. Nonreceptors are stimulated by prostaglandins to mediate the pain-producing effects of bradykinin. Inhibition of enzyme activity required for synthesis of prostaglandins can be achieved by the use of nonsteroidal anti-inflammatory drugs (NSAIDS). (Analgesic Pathways, Ortho-McNeil Pharmaceutical, Spring House, PA 1996).

Pharmaceutical Intervention—Side Effects

1) NSAIDS

Compromising the synthesis of prostaglandins NSAIDS potentially results in gastrointestinal ulcers and bleeding. Prostaglandins protect the gastric mucosa by reducing gastric acids, maintaining mucosal blood flow, and by stimulating bicarbonate and mucus secretion. Maintaining the blood-filtering functions of the kidneys is also a function of prostaglandins. NSAIDS may compromise renal function and consequently induce hyponatremia or hyperkalemia. Elderly patients are particularly affected by NSAIDS treatment due to possible existing impairment of renal or cardiovascular function.

2) Opioid Analgesics

Agents that act centrally activate descending analgesic pathways and have no action on peripheral mediators such as prostaglandins. Opioids tightly bind to receptors in the thalamus and the forebrain. They do not bind to receptors for serotonin and norepinephrine. The physiological effects associated with opiate use varies and includes euphoria, analgesia, sedation, respiratory depression, changes in thermoregulation, muscle rigidity, inhibition of gastrointestinal motility, and a great potential for physical abuse and dependence and addiction.

Specific G-protein coupled receptors (GPCRs) that bind opioids control the physiological effect. (Loew, G.H. 1999).

The analgesic role of opioids may be further accentuated by binding at these sites. They mimic endogenous opioids or endorphins as

agonists, binding to mu 1 and mu 2 opioid receptors, thereby activating the descending analgesic system. Inhibitory pathways are activated by the binding of opioids and thus control pain perception. Nausea, dizziness and constipation are other side effects of opioid therapy. Because of widespread presence of opioid receptors it is thought that binding of opioids in the thalamus and forebrain may be responsible for their abuse in young persons and the elderly. Oxycontin is currently being monitored very closely for such abuse throughout the country. This opioid has a long acting effect, and has become popular on the "street" as a drug of abuse. Patients, physicians and drug addicts all interact with the legal system to create a crisis in pain management.

The regimen of combining opioids with reuptake inhibitors has been shown to potentiate the analgesic efficacy of the opioid or to reduce its side effects.

3) Narcotics

Narcotics are level 4 controlled substances and subject to addiction and widespread abuse with serious adverse effects and even lethality in populations. Oxycodone is an example. (Analgesic Pathways, Ortho-McNeil Pharmaceutical, Spring House, PA 1996).

Pain Assessment

Pain assessment can be difficult due to a myriad of phenomena that may co-exist with symptoms of pain. During injury afferent neurons transmit signals to central cells as previously mentioned. These cells can generate false signals when they encounter unusual messages from injured peripheral nerves, tracts, or roots. Understanding these phenomena have stimulated a life time of studies at McGill University as well as other centers of excellence around the world. It describes the rapidly acting mechanisms which receive and control the transmission of signals from afferent input fibers to cells which consequently activate a number of effector systems that evoke sensation, McGills' Professor Melzack's gate theory.

Consider a concept of pain that is not the sum total of anatomical pathways involving isolated systems of afferents and central cells. Emotions of anxiety and depression as well as psychological influences can affect pain perception. Transmission of pain stimuli from peripheral systems influence injury produced afferent transmission that passes through gate control has surfaced as a primary mechanism along with the sensory "disposition" of the CNS. Pain can be elevated or diminished due to the arrival of of nonreceptive afferent impulses and signaling of peripheral events as well as the setting of excitability by CNS mechanisms. The brain is a dynamic in that it can analyze pain phenomena as a function of experience and its relationship to concepts of past, present and future conditions. (Wall, P.D. & Melzack, R. 1984).

Pain Assessment Measurement

The McGill Pain Questionnaire (MPQ) is a highly sensitive tool that yields valid, reliable, consistent results in a reasonable amount of time. The descriptors fall into four major groups: sensory, 1–10; affective 11–15; evaluative, 16; and miscellaneous, 17–20. The rank value for each descriptor is based on its position in the world set. The sum of the rank values is the pain rating index (PRI). The present pain intensity (PPI) is based on a scale of 0–5. (Copies of the Questionnaire may be obtained by contacting this author). When administered to a patient by reading each subclass it can be completed in about 5 minutes. A paper and pencil test filled out by the patient is also effective although scores are somewhat different. The MPQ has been refined to achieve a high degree of agreement on the intensity relationships among pain descriptors by subjects who have varying socioeconomic, educational, and cultural backgrounds the refined MPQ is thought to be an accurate depiction

of pain measurement. In addition to the list of pain descriptors, the questionnaire has line drawings of the body to show the spatial distribution of the pain, words that show temporary properties of pain, and descriptors that rate the overall present pain intensity (PPI).

The PPI is measured using numbers from 1–5 with the following meanings: 1-mild; 2-discomforting; 3-distressing; 4-horrible; 5-excruciating. It is thought that this rating represents equal scale intervals providing "anchors" for the determination of the pain intensity. The descriptor lists of the MPQ are read to a patient with the explicit instruction that he or she choose only those words that describe his or her feelings and sensations at that moment. Three major indices are obtained:

1) The pain rating index (PRI) based on the rank values of the words. In this scoring system, the word in each subclass implying the least pain is given a value of 1, the next word is given a value of 2, and so forth. The rank values of the words chosen by a patient are summed to obtain a score separately for the sensory (subclasses 1–10), affective (subclasses 11–15), evaluative (subclass 16), and miscellaneous (subclasses17–20) words, in addition to providing a total score (subclass 1–20) shows MPQ scores (total score from subclasses 1–20) obtained by patients with a variety of acute and chronic pains. (Based on a comparison of pain scores using the McGill Pain Questionnaire, obtained from women during labor (Melzack, et al., 1981), and from patients in a general hospital pain clinic (Melzack 1975) and an emergency department (Melzack, et al., 1982). The pain score for causalgic pain is reported by Tahmoush (1981). Copies of the score comparisons may be obtained by contacting this author).
2) The number of words chosen (NWC).
3) The PPI, the number-word combination chosen as the indicator of overall pain intensity at the time of administration of the questionnaire. (Melzack, R. & Katz, J. 1992).

Pain Evaluation

Evaluation of pain should begin with a patient history though in many emergency situations this can be compromised. If possible, a complete patient history should be recorded, including family history, social history (inquiry regarding use of alcohol, drugs, tobacco, family/social relationships, and environmental factors that may cause stress), medical history, and a complete workup of pain; onset, location, duration, intensity and perception. After the history, a very careful physical exam is performed, appropriate laboratory tests are then ordered and results interpreted, a differential diagnosis is established. Anatomical, physiological and pathophysiological mechanisms are then developed. Treatment needs to be specific and sufficient to control the pain if the underlying cause cannot be eradicated. For instance, a study has analyzed the phenomenon of patients experiencing pain at the onset of acute trauma ranging from cuts to fractures and amputations. Patients remarked that they were not initially aware of the pain. Three mechanisms were suggested that are unrelated to shock; 1) time course begins with instant analgesia and not initial pain, 2) Eventually, all patients experience pain and 3) local controls explain the primary intervention of pain perception as the analgesia is spatially limited to the region of the injury suggesting the lack of involvement of hormonal or generalized internal system. (Melzak, R., Wall, P.D. & Ty, T.C. 1982).

Pain Management

The study of pain and its treatment requires a comprehensive team approach. The team should be composed of the following professional members:

1) Neurologist: Trained in pain evaluation, assessment and treatment
2) Neuropsychologist: Experienced in neuropsychological testing, administering pain questionnaire such as McGill
3) Anesthesiologist: To administer blocks and do procedures
4) Neurosurgeon: To perform surgical procedures to eradicate, control or treat pain
5) Orthopedic Surgeon: To perform orthopedic procedures and work with the neurosurgeon to treat or control pain
6) Physiatrist: Provides expertise in Physical Medicine and rehabilitation and assist in assessment and treatment
7) Physical Therapist: Helps with treatment of pain
8) Occupational Therapist: Also helps with pain management
9) Other Professionals: Psychologist, Social Worker, Psychiatrist, Chiropractor, Osteopath, Acupuncturist and other specialist helping with pain management

Pain as a diagnostic tool is instructive not only in many illnesses and tissue damage, i.e. coronary diseases and events, arthritis, musculoskeletal pain after trauma, acute trauma (blunt force trauma), childbirth, surgery, pain as a result of violence with concomitant acute and psychological pain, combat and also emotional pain due to loss, anxiety, rejection, divorce, stress, etc. Its accurate diagnosis and treatment can only be met by the assessment and treatment of a qualified pain management treatment team.

Treatment of Pain:

1) Transient Pain

Pain elicited by the activation of nonreceptive receptors in skin or other tissues of the body in the absence of any tissue damage. The individual has a definite perception as to the speed of onset and offset that the disturbance has been diminished such as venipuncture of injection or tattooing. Common over-the-counter analgesics and antipyretics are usually sufficient treatment for pain from these ailments (such as Tylenol, or Aspirin, or Advil).

2) Acute Pain

Acute pain is characterized by recent onset that ends or is anticipated to end during a period of days to weeks. When it is due to tissue damage or injury a biological response by both somatosensory and inflammatory mechanisms. Anxiety and the systemic signs of sympathetic hyperactivity ("fight or flight" response) may accompany acute pain; including, elevated blood pressure, increase in pulse, mydriasis, diaphoresis, rise in alveolar ventilation, respiratory rate, and oxygen consumption. (Behbehani, M.M. & Fields, H.I. 1979).

Acute pain is localized by substantial injury of body tissue and response of nonreceptive receptors at the site of the tissue injury. This type of pain is seen after trauma, surgery, and some diseases. Healing may occur without medical intervention. Acute pain includes migraine, some arthritis and musculoskeletal pain.

3) Headache and Facial Pain

Headache pain is reported to recur with more frequency than any pain complaint. Headache pain can be nonreceptive. The extracranial vessels, scalp, periosteum, sinuses,

meninges, and large intracranial vessels are innervated by small nerve fibers. The tentorium cerebelli and some of the supratentorial structures are innervated by the trigeminal nerve. Eye pain and forehead are ascribed to pathology of these structures. Retro-orbital pain is often reported from the cavernous sinus, the tentorium, or the carotid artery. Diagnosis of headaches include migraine-classic (with aura) or common (without aura), tension type headache, acute, cluster and headache associated with trauma. The sudden onset of an extremely severe headache (acute) is a common symptom of subarachnoid hemorrhage. Acute and chronic headache can be associated with cerebrovascular disease some of which may be due to transient ischemic attack or stroke. (Bernad, P.G. 2000) (Portenoy, R.K. & Kanner, R.M. 1996).

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THE VALUE OF COMMUNICATING REMORSE

Frank D. Heckman*

All hospitals have mission statements. Physicians, clinics and other health care provider organizations have, in some form, stated values or goals and objectives. However, when a patient becomes a plaintiff, health care providers behave as if the patient is an adversary. The practical effect of this change in attitude is that health care providers' mission statements or values or goals seem to become moot. Communication channels close. Plaintiffs and defendants retain legal counsel. In