Reprint from BC Medical Journal — Volume 31, Number 3, March 1989

"Whiplash" syndrome

Dr. F.P. Patterson has admirably summarized the facts on the 'whiplash' syndrome (*BCMA* 1988:10:659) from the orthopedist's standpoint; however, a pain perspective may be appropriate. Pain is a general reaction-pattern of three distinct, sequential, and natural behavioral phases.[1] (i) *Nociception* signals tissue threat or damage, but as a rule it abates quickly, unless there has been tissue damage; then, (ii) *inflammatory* pain ensues until healing is complete. Healing is usually quick, but (iii) *chronic pain* is likely if any of the following is present.[2]

Ongoing nociception or inflammation: These are easily recognized; and as Dr. Patterson has indicated, ongoing nociception (eg, compression fracture) is unlikely unless the collision has been violent.

Psychologic factors: Frustration compounds anguish when pain proves impossible to diagnose or treat; deactivation, iatrogenic, and psychologic factors may then perpetuate pain.[3]

Functional and/or structural alterations in the CNS or PNS: Medical diagnosis traditionally presumes that pain is conveyed to the CNS via a healthy nervous system; but pain may be associated with abnormal nerve function and/or hyperactivity in the pain sensory system, ie, *neuropathic pain.*[4] In a "soft tissue injury," even minor harm to the spinal nerve can cause neuropathy (ie, "any *functional* disturbances and/or pathological changes in the peripheral nervous system").[5] Neuropathy often occurs without denervation; thus, a standard neurological examination generally overlooks the condition.

Features of neuropathic pain may be any of the following: pain in the absence of tissue-damage; delay in onset after injury; unpleasant sensations (dysesthesiae or "deep" pain); pain in a region of sensory deficit; paroxysmal shooting pain; allodynia; and pronounced summation with repetitive stimuli.[6]

Clinical manifestations[5-9] are *autonomic* (eg, vasoconstriction, trophedema),[10] *motor*, or *mixed*. Dr. Patterson has mentioned a fundamental motor manifestation: muscle spasm. Spasm (often increased by emotional stress)[11] may be focally tender and painful (ie, "trigger points").[12,13] Spasm also leads to muscle shortening and a loss of joint range (and enthesopathic tendons may suggest a possible immunogenetic factor).[14]

Neuropathic pain usually resolves spontaneously, or with the temporary use of analgesics or physical therapies. However, pain can be relentless when accompanied by spasm.[12] When spasm is released, pain is usually alleviated.[12,13] Therefore, the release of spasm is an important part of treatment. When physical measures, including stretch and spray,[12] manipulation, or TENS fail, an injection technique is called for. Local anesthetics or saline are used,[13] but dry needling, without injected substances, is also effective.[15,16] The latter is preferred at the Pain Center, University of Washington, Seattle, because it avoids the iatrogenic effects of injected medications; and the use of a fine, whippy needle (borrowed from acupuncture) can determine spasm in deep muscles. Since ancillary tests are unhelpful, the diagnosis of neuropathic pain depends primarily on clinical examination, especially palpation (and preferably, needling) of individual muscles for painful spasm. In fact, the 'chronic pain syndrome' patient is rare: usually, the label is applied to an unfortunate patient when neuropathic pain has not been ruled out.

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