Dynamic Chiropractic

BACK PAIN

Compression Does Cause Pain

The concept that compression from herniated discs or bony stenosis, per se, does not produce pain is difficult to accept.

But why should this be so? Is it a lack of understanding? The teachings of our colleges? An apathetic attitude, or a function of, "It is so easy for the patient to understand," you know, the big black shoe on the green garden hose concept. Or is it just plain stubbornness that we must hang onto our heritage, philosophy, historical basis, and that which is unscientific at all costs, and damn the consequences attitude?

Before we look at the hard cold facts of life, let's look at the generally accepted picture of the patient with low back pain, let me quote from a recent text on these matters. "Most journals display a picture of low back pain with a picture of a patient with a herniated disc and, although this is uncommon, it is the image most often portrayed. Rosomoff et al., in New Approaches to Treatment of Chronic Pain, tell us that in a study of 5,000 patients with low back pain no evidence of herniated disc or tumor was found. Similarly, during the past 15 years, 75 percent of such patients at the University of Miami were seen as having no sensory, motor or reflex loss; this figure has currently risen to 99 percent. This percentage was confirmed by the Quebec Task Force on Spinal Disorders report of 45,000 patients. The report indicated that only one percent of cases of low back disorder involve deficits and indications for surgical intervention, of which approximately one half come to operation.[1]

The percentage of patients with "no objective findings" has increased because of an ever-growing awareness of the dysfunctional state, not deficit, caused by reflex inhibition of neural function"[2] For a comprehensive in depth look at the function of the dorsal horn with respect to nociceptive input and the resultant reflex muscular spasm and vasoconstriction, the reader is referred to the published works of Dr. D.R.Seaman.[3] Dr. Seaman tells us: "We could say that the subluxation complex is caused by muscle spasm and vasoconstriction ... muscle spasm and vasoconstriction are both reflexively induced by nociceptive afferent input." Obvious to all should be the fact that nociceptive input is the result of a dysfunctional state.

What actually happens to a nerve root with respect to the herniated disc or bony stenosis?

Listen to the words of Dr. P. Wall, who tells us: "If the axon is stimulated at the root level by pressure, a section of the nerve will be distorted. The nerve membrane will be stretched. Ionic flow occurs with depolarization and the generation of an action potential. The initial signal is only transient, because maintenance of pressure will evoke accommodation, a fall in current density, and axonal block or silence from disinhibition of central structures after the initial mechanical distortion."[4] In other words, nerve root compression from whatever source is painful only briefly, and sustained pressures lead to electrical silence and absence of sensation.

So how does the herniated disc or bony stenosis actually cause this short lived pain?

It is worth reminding the reader that vital to the transmission of pain is the induction of the state or condition of hyperalgesia and sensitization of nerve endings. The injury need not be gross. Microscopic tissue damage, as in inflammation, may be enough. Simply put, sensitization is the result of the breakdown of cell membranes and the production of arachidonic acid from which pro-inflammatory substances like prostaglandins and leukotrienes (nociceptive irritants) ensues. These substances are integral to the mechanism of inflammation and loss of function (see Dr. D. Seaman's notes[3] on chiropractic and the dorsal horn for an in depth review of these concepts and pathways). Once hyperalgesia has been established, tissue breakdown substances, such as bradykinin, histamine, and other neuropeptides, are released, which in the presence of hyperalgesia, combine to produce pain.[5] The herniated disc or bony stenosis causing pain can easily be explained in these same terms. When a nerve root is distorted by some structure the nerve is irritated, rendering the nerve and the tissues hyperalgesic.

In the chronic case the peripheral tissue injury perpetuates a continuing nidus for the production of hyperalgesia and algesic compounds, which interact to generate pain. Moreover, the nervous system may have a memory for pain, which, we are told, can remain active for a substantial time after the original injury has healed.

Injury of any kind, whether it be macro or micro trauma, produces inflammation, which is a continuing process, resulting in a delayed or exaggerated onset of pain.[6] With respect to the intraspinal pathology, it now seems clear that the biochemistry of the disc is altered by both injury and the normal ongoing aging process. Degradation of the collagen/proteoglycan matrix produces glycosylation products, which act as foreign proteins, resulting in an inflammatory response.[7] The healing potential of the annular fibers is limited, and provides for a continuous escape of inflammation-provoking substances into the perineural spaces.[8] The nerve endings are now sensitized and inflammation spreads. The concept of spreading neurogenic edema, as outlined by Dr. H. Fields,[9] clearly explains the aforementioned in extreme detail and should be a part of every doctor's knowledge base. Sensitization by repeated stimulation lowers the threshold for pain perception. As inflammation spreads throughout the tissues and sensitization increases, pain proceeds from the originating autonomous area to a much larger area.[10] The delayed, secondary onset of inflammation is an extension of prostaglandin sensitization, hyperalgesia, and algesic substance release combined with spread of the reaction to injury from the primary site of tissue insult.

Regardless of the site or type of the injury, a soft tissue component is always and necessarily present. The soft tissues, i.e., annulus, ligaments, tendons, and muscles, maintain the integrity and stability of the spine (see MPI core course notes on the Back Force Transmission System). Because the peripheral tissue is primarily myofascial and muscle contraction (review Dr. Seaman's notes on nociception and the dorsal horn), plastic evolution to contracture follows with restricted range of motion, loss of function, and subsequent disability. (The reader is advised to refresh his or her memory about the processes of creep, hysteresis and set, with special attention given to why chiropractic treatment should be directed towards the children of our society). This process is another form of chemical reaction. The injury releases calcium ions to combine with ATP to produce an uncontrolled contraction. We see this clinically as muscles that are tight, rigid, and stiff. For more information on this topic the reader is directed to the 1994 text, Muscles as Molecular and Metabolic Machines, by

Dr. Peter Hochachka, published by CRC Press.

In short, pain is defined by most as a sensation that arises from tissue injury, that is, any tissue injury. The pressing and obvious question is, what tissue? The tissue in question can only be that tissue which has the chemistry to provide the aforementioned responses. It should be noted that "sustained pressure" is not one of causes. When dealing with low back or leg pains the tissues are the large muscles masses, as described by Bogduk, Vleeming and Gracovetsky in the proceedings of the first and second World Congresses on the Sacroiliac Joint and Its Relationship to Low Back Pain. These muscles maintain the integrity of the body through the back force transmission system. The spine is a loosely articulated group of bones which can be converted into a rigid lever arm by vertical axial contraction of the paraspinal muscles. Motion and weight displacement are translated from above down and from below upwards, i.e., the lateral compartment muscles, the long head of the biceps, and the G-max are coupled to the contralateral latissimus dorsi for the transmission of forces; coiling of the latissimus is coupled with the coiling of the contralateral sacrotuberous ligament. To talk about back pain without appreciation of the role of the hips, knees, and fibular mechanics, which when mechanically dysfunctional produce back and lower extremity pain, is impossible.

Approximately 50 years ago the concept of the ruptured disc as "the" source of pain was put forth by Mixter-Barr. Continued failure to solve the dilemmas of low back pain makes it time to look elsewhere for the source of the pain generator. A statement from the Miami Comprehensive Pain Center may help you with this: "All surgeons of experience have had a patient with the so-called classic herniated disc who suddenly has a coronary occlusion the night before the scheduled surgery, which precludes operation. The cardiac condition is treated, and three months later the patient is ready to have the disc removed for the foot drop which he or she presented with originally. One problem exists: the patient no longer has a footdrop, still has the abnormal disc, and is clinically fine."

From the above, it should be clear that although the disc can be responsible for our patients pain, it is indeed rare and, if it exists at all, it is for a very short time. This column has attempted to point out some of the causes of our patients pain; to point out that chiropractors need to be aware of the current literature as it pertains to what is really causing the pain, and therefore what structures to treat.

MPI core courses and the MPI Diplomate Program in Neurology cover this topic and many others.

References

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NOVEMBER 1996