

Prespondylosis” and Some Pain Syndromes Following Denervation Supersensitivity

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FROM ABSTRACT:

Pain is determined by the neurologic properties of receptor organs, neurons, and their interconnections. These may become supersensitive or hyperreactive following denervation (Cannon’s Law).

A common cause of denervation in the peripheral nervous system is neuropathy or radiculopathy as a sequel to spondylosis. Spondylosis in its early stage may be “asymptomatic” or painless and hence unsuspected, because small-diameter pain fibers may not initially be involved despite the attenuation of the other component fibers of the nerve.

The term “prespondylosis” is introduced here to describe this presently unrecognized phase of insidious attrition to the other functions of the nerve, especially the trophic aspect.

It is postulated that many diverse pain syndromes of apparently unrelated causation may be attributed to abnormal noxious input into the central nervous system from supersensitive receptor organs (nociceptors) and hyperreactive control systems at internuncial pools.

Furthermore, trauma to a healthy nerve is usually painless or only briefly painful, unless there is preexisting neuropathy [radiculopathy].

KEY POINTS:

1) Pain receptors and neurons may become supersensitive or hyperreactive following denervation.

This is known as **Cannon’s Law** or the Law of **Denervation Supersensitivity**.

2) A common cause of denervation in the nervous system is as a sequel to spondylosis.

3) Spondylosis in its early stage may be “asymptomatic” or painless and hence unsuspected, because small-diameter pain fibers may not initially be involved despite the attenuation of the other component fibers of the nerve.

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4) "Prespondylosis" is asymptomatic, but causes an "unrecognized phase of insidious attrition to other functions of the nerve."

[This is a great description of chiropractic nerve interference.]

5) Many diverse pain syndromes of apparently unrelated causation may be attributed to abnormal noxious input into the central nervous system from supersensitive receptor organs and hyperreactive neurons.

6) Trauma to a healthy nerve is usually painless or only briefly painful.

7) Trauma to a nerve that causes prolonged pain indicates that the nerve was suffering from preexisting radiculopathy or neuropathy.

8) Regarding pain, there is no direct relation between the intensity of the applied stimulus and the intensity of the evoked [painful] experience.

9) Pain is transmitted to the central nervous system by primary afferent nerve fibers which synapse on motoneurons, either directly or through interneurons. This, in turn, commands actions by effectors such as muscle and gland cells.

10) When a nerve becomes supersensitive, it modifies both afferent inputs and the efferent response to the effector organs (muscles and/or glands).

11) *Prespondylosis*, is the early pain-free stage of spondylosis, and it may cause of unsuspected radiculopathy.

12) The early research on *denervation supersensitivity* was by Harvard's Walter Cannon, who proposed the Law of Denervation (Cannon's Law). Cannon and colleagues showed that denervated striated muscle, smooth muscle, salivary glands, sudomotor glands, autonomic ganglion cells, spinal neurons, and even neurons within the cortex develop supersensitivity.

13) Subtle irritations to a nerve that do not cause actual physical interruption will cause the nerve to become supersensitive. [Chiropractic Nerve Interference]

14) Sympathetic and other autonomic nerves do become supersensitive.

15) Chronic irritation of primary afferent neurons decrease the number of opiate receptors in the dorsal horn with a corresponding reduction of presynaptic nociceptive inhibition.

16) The spinal root within the spinal canal and intervertebral foramina is especially prone to irritation. "This may follow acute trauma, but more usually it is the long-term sequela of spondylosis which causes simultaneous damage to the nerve roots (radiculopathy) and cord (myelopathy)."

17) Although spondylosis may be silent and pain-free, it is not necessarily morbidity-free, including during the prespondylotic phase.

18) "'Prespondylosis' may be 'symptomless,' its symptoms and signs unsuspected, because **pain may not be a feature, pain occurs only when and if the degenerative changes impinge upon local pain-sensitive structures to produce local pain, or upon pain fibers of the nerve root to produce the transmitted pain of radiculitis.**"

19) **In prespondylosis and early spondylosis there is often no pain because pain neurons are "small and less liable to mechanically caused ischemia."** "Since **pain fibers are not necessarily involved there are no 'symptoms' and both patient and physician may be oblivious to the condition.**"

20) Acute mechanical injury to a healthy nerve root does not produce a sustained discharge unless there has been preexisting minor chronic injury to the nerve.

21) "Clinically, it is also common knowledge that in asymptomatic subjects the mere appearance of degenerative changes in spinal roentgenograms is not of much clinical significance, but in these persons, disability after injury will tend to be prolonged and signs of radiculopathy more commonly found. It would therefore appear that for pain to persist after trauma, a prerequisite is the existence of chronic nerve irritation." [Important]

22) *Myalgic hyperalgesia* is excessive tenderness of a muscle to digital pressure. It is not a normal feature of muscle because their mechanosensitive nociceptors are located deep within the muscle bulk and have high thresholds.

23) *Myalgic hyperalgesia* may occur following local muscle injury and tissue damage when algogenic chemical substances are liberated.

24) *Myalgic hyperalgesia* will exist secondary to nociceptor supersensitivity.

25) In *myalgic hyperalgesia* secondary to nociceptor supersensitivity, the tenderness is maximum at the neurovascular hilus where nociceptors are most abundant around the principal blood vessels and nerves as they enter the deep surface of the muscle to reach the muscle's motor zone of innervation. As this zone is fairly constant in position for each muscle, tenderness in muscles secondary to radiculopathy is easily found. Tenderness at the muscle's zone of innervation is often loosely referred to as the "motor point" (a point on skin where a muscle twitch may be evolved in response to minimal electrical stimulation). These motor points spatially coincide with the trigger points of Janet Travell, MD.

26) The degree of tenderness at an extremity motor point (*myalgic hyperalgesia*) usually parallels the degree of spondylotic radiculopathy supersensitivity.

27) The degree of tenderness at a motor points within an affected segmental myotome is a useful diagnostic and prognostic aid following spinal injuries.

28) Simple palpation revealing hyperalgesia in the muscles supplied by both anterior and posterior primary rami of the same root level is indicative of a subclinical radiculopathy at that segmental level. Although such conditions may be labeled as vague clinical entities ("tendinitis," "bursitis," or "fibrositis"), "treatment should logically be addressed to the underlying spinal problem; in our experience, this has been followed by resolution of symptoms." **[Very Important]**

29) Supersensitivity nerve interference can cause increased muscle tone or even spasm.

30) "Supersensitivity in autonomic pathways can lead to the increased blood vessel tone [and ischemia] of virtually all tissues and cause secondary pain by structural disintegration."

31) Supersensitivity in autonomic pathways reduces the total collagen in soft and skeletal tissues. "Replacement collagen has fewer cross-links and is markedly weaker than normal mature collagen. Because collagen provides the strength of ligaments, tendons, cartilage, and bone, this may contribute to many degenerative conditions in the weight-bearing (spinal and intervertebral disc) and activitystressed parts of the body (tendinitis, cuff tears, epicondylitis, ruptured tendons and so forth)."

32) "Degenerative disc disease itself may not be a primary condition. The disc is particularly vulnerable to altered vascular tone, being almost avascular and dependent largely upon diffusion through adjacent spongy bone for nutrition."

33) **Prespondylosis, supersensitivity, and myalgic hyperalgesia are often found in young (under 30 years), apparently normal, and asymptomatic subjects.**

Prespondylosis "is generally painless, though not necessarily devoid of morbidity," and is a frequent cause of radiculopathy, which leads to degenerative conditions.

COMMENTS

This article is an excellent review of the orthopedic neurology of the subluxation and nerve interference:

Altered position causes *prespondylosis*.

Prespondylosis causes *radicular supersensitivity* nerve interference.

Radicular Supersensitivity causes *myalgic hyperalgesia* in the segmental muscles innervated by the anterior and posterior primary rami.

Radicular Supersensitivity also adversely affects the autonomic (sympathetic) nerves, affecting the glands and viscera.

Radicular Supersensitivity causes degradation of the innervated collagen, accelerating degeneration and *spondylosis*.

Treatment is to the spine.