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Review Article

Surgery for Chronic Back Pain or Chronic Neck Pain – Does It Work?

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Abstract

Chronic pain frequently results in drug abuse of pain medicines and potentially the death of the patient from drug toxicity. Surgical interventions have been examined as potential cures for chronic pain. Patients are eager to find anything to help their pain. The mechanism of chronic pain does not provide a rationale for surgical intervention. Alternative therapies can be effective against chronic pain.

Keywords: Chemokine; Chronic pain; Diterpenoid; IL-17; Monoterpenoid; Transient receptor potential cation channel

Introduction

There is no scientific evidence that surgery improves chronic back pain or chronic neck pain. Meta-analysis has shown that if anything, surgery makes the pain worse [1]. This study has been criticized for selecting the wrong patients [2]. However, patient selection was done by a rigorous Cochrane procedure and was correct. Why do so some surgeons insist on doing surgery for these conditions? Why do so many patients seek to do the surgery? After many years of talking to these surgeons and surgery patients the author can only conclude that some surgeons and patients believe in the surgery and hope that it will cure chronic pain. In fact, these patients believe they have no other hope. When the surgery fails, they feel doomed to live the rest of their lives in chronic pain. However, current clinical practice suggests using alternative therapies such as acupuncture and yoga instead of surgery [3].

Surgery

There are a few conditions where surgery can help with back or neck pain. Tumors of the spine or spinal cord cause pain that can be alleviated with surgery, as discussed in a review of randomized, clinical trials [4] Lumbar disc prolapse also results in pain that can be somewhat alleviated by surgery, as presented in a Cochrane analysis of clinical trials [5]. Prolapsed disc is frequently associated with age related disc degeneration. The pain from a prolapsed disc usually improves after a few days and is gone within 4 to 6 months, even without surgery, as discussed in a systematic review of clinical trials [6]. Surgery adds an unnecessary procedure for prolapsed disc, an extended recovery time and the potential for surgery induced chronic pain. However, prolapsed disc can produce chronic pain, or the medications used to treat prolapsed disc pain cause chronic pain [7]. Even after the prolapsed disc is healed, chronic pain remains and can endure for years.

What is Chronic Pain?

There is a perception that chronic pain comes from an internal problem, such as a spinal problem, neural inflammation in the brain or brain stem [8]. This central sensitization theory is supported by animal model work. However, this approach to chronic pain has not resulted in effective surgery, therapy or cures for chronic pain. In fact, all of the available oral therapies for chronic pain do not work or make the pain worse and prolong the pain [7,9]. In addition, surgery that might alleviate inflammation of the spinal cord is not effective against chronic pain [1].

Central sensitization involves the recruitment of signals from painful and nonpainful stimuli that come from an unspecified place outside the brain [8]. These painful and nonpainful signals are summated in the brain to produce a nociceptive signal. This especially involves dorsal horn neurons in the brain stem. It is not clear what change in the brain leads to the summation of nonpainful signals, which is not normal for the brain. In this mechanism, pain is no longer coupled with a painful stimulus and becomes autonomous. Brain regions such as the parabrachial nucleus, the periaqueductal gray, the superior colliculus, and the prefrontal cortex are involved in this mechanism. Glutamate receptors including NMDA receptors become automatically active in central sensitization. Ketamine, an NMDA receptor inhibitor, is effective in the treatment of chronic pain, but only for a short time, as discussed in a meta-analysis of randomized, clinical trials [10]. Long term ketamine therapy does not cure chronic pain. This implies that NMDA receptor sensitization is not the stimulus that causes nonpainful signals to be summated in the brain.

Various nociceptive signaling molecules are involved in the central sensitization mechanism in the brain including bradykinin, substance P, brain derived neurotrophic factor and serotonin [8]. Cyclo-oxygenase 2 becomes induced in dorsal horn neurons and makes prostaglandins that cause pain. Macrophages, neutrophils and T cells infiltrate into the brain stem and produce chemokines, prostaglandins and other inflammatory factors [8]. It is not clear what causes these changes to occur in the brain and brain stem. Pain dependent alterations to skin sensory neurons may cause these changes in the brain and brain stem. Damage to skin sensory neurons may incite neuropathic pain sensitization in the brain [11].

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Recent evidence demonstrates that Transient Receptor Potential (TRP) cation channels in skin sensory neurons are the primary nociceptors in the body [12,13]. There are at least 28 types of TRP channels that exist in nonoverlapping populations of skin sensory neurons. TRP channels are activated by painful stimuli, heat, cold, stretch and other stimuli. Medicines that inhibit these receptors in the skin are more powerful than morphine at relieving pain [14]. In fact, many monoterpenoids are effective inhibitors of various types of TRP channels [13]. They penetrate into the skin, inhibit pain and evaporate from the skin without poisoning the body.

Chronic pain comes from the skin [9,15,16]. In fact, the skin generates pain in chronic pain, the pain chemokine cycle. Damage to a sensory neuron by a painful stimulus causes the neuron to release chemokines that attract monocytes and neutrophils to the site. Monocytes release prostaglandins to cause pain. Neutrophils release leukotrienes that cause long term pain by activating TRP channels. Bradykinin is released by damaged keratinocytes to cause pain and inflammation. Chemokines induce IL-17 release by skin resident T cells. IL-17 induces chemokine production by macrophages and sensory neurons.

The pain chemokine cycle also causes neurogenic inflammation [17] due to the release of neurokinins, including substance P, and other inflammatory proteins from sensory neurons. These inflammatory proteins may penetrate into the brain from the blood [18] and may cause changes in brain stem and brain neurons leading to central sensitization. In addition, chemokines released into the blood activate macrophages, neutrophils and T-cells that penetrate into the brain, cause inflammation and chemokine release in the brain [19] that may be involved in central sensitization. Therefore, chemokines generated at the site of pain in the skin can result in the generation of chemokines in the brain. This establishes a connection between the peripheral site of chronic pain in the skin and central sensitization.

How is Chronic Pain Treated?

Chronic pain is frequently treated with chronic drug therapy that is only effective for a short period, as discussed in a review of randomized, clinical trials [20]. This may be followed by hyperalgesia if the drug therapy induces chemokine formation, such as with opioids and nonsteroidal anti-inflammatory drugs [7]. For the patient, drug toxicity problems may become life threatening. None of the drugs available cure chronic pain. The current approach to drug development for chronic pain appears to be focused on finding new opioids or new nonsteroidal anti-inflammatory agents that may not be life threatening for patients.

Acupuncture is used in the treatment of chronic pain and may improve function,patient attitude and pain [21,22]. A recent Cochrane meta-analysis study found that acupuncture is better than no treatment, but did not provide a cure for chronic pain [21]. Even though acupuncture does not cure chronic pain, at least patients do not die from acupuncture.

Exercise can decrease chronic pain in many patients, including patients with fibromyalgia and chronic low back pain, as presented in a review of randomized, clinical trials [23]. Of course, exercise is not recommended in exercise induced injury. The therapist must work with the patient to individualize exercise and help with the initial pain when starting the exercise regime. Moderate exercise is anti-inflammatory since it decreases chemokine receptor expression and inhibits the migration of inflammatory leukocytes into the brain [24]. The decrease in chemokines is especially found in life long exercisers [25].

Other alternative therapies for chronic back pain include chiropractic care, osteopathic manipulation, yoga and tai chi. Clinical experience with these techniques has shown they decrease chronic pain, are safe, but do not cure chronic pain [26-28].

Two Cures for Chronic Pain

Plant derived mixtures of monoterpenoids, diterpenoids and other compounds such from *Salvia mellifera* or Artemisia californica can cure chronic pain [9,15,16,17,29]. Monoterpenoids inhibit TRP channels, chemokine formation, pain and help patients stop taking opioids [9]. Inhibition of TRP channels can stop the pain chemokine cycle. It is crucial to use a mixture of monoterpenoids to inhibit as many types of TRP channels as possible. This may provide effective inhibition of chemokine formation. Diterpenoids inhibit IL-17 formation in the skin which may result in inhibition of chemokine formation in the skin and brain [29,30]. Use of a mixture of monoterpenoids and diterpenoids results in inhibition of TRP channels and chemokine formation. These plant derived medicines have been used for many years to treat and cure chronic pain patients.

Conclusion

Chronic pain can be cured with plant medicines such as from S mellifera or A californica. Moderate exercise, such as walking, is recommended in the treatment of all chronic pain patients. Surgery should not be recommended in these patients.

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