Insights Into Chiropractic

Discerning the true nature of an alternative health care method

Spinal Manual Therapy & Pain Modulation

INTRODUCTION

One of the major areas of study regarding joint manipulation has been its role in the modulation of pain. Although the exact mechanism by which spinal manual therapy relieves pain is unknown, researchers have postulated that the mechanism involves the abortion of incoming nociceptive signals from damaged tissues at the level of the spinal cord by the activity of spinal joint mechanoreceptors.

The role that manipulation plays in the modulation of pain by the stimulation of mechanoreceptor activity will be discussed in this issue of Insights Into Chiropractic.

ARTICULAR NEUROLOGY

Aritcular reurology is concerned with the morphology, physiology, pathology and clinical aspects of the nerve supply to the joints of the body. The following review of the literature is taken from the writings of Wyke(1-18), a leading authority on articular neurology. Wyke is well know for his contributions in this area and for his prolific writing on the neurological mechanisms of manipulation.

TYPES OF JOINT MECHANORECEPTORS

Three types of mechonoreceptors innervate the joints of the body, each with its own specific structure and function. By sending electrical impulses to the brain through the dorsal columns and spinocerebellar tracts, these receptors function to provide the body with proprioception and kinesthetic sense.

Type I mechanoreceptors are thinly encapsulated globular corpuscles resembling Rufinni endings. In the spine they are located in the superficial layers of facet joint capsules and have a low threshold of activation (i.e. very little joint motion is required to cause an impulse to be generated to the spinal cord).

Through a tonic discharge rate of approximately 20 impulses per second, Type I mechanoreceptors function to keep the central nervous system apprised of static joint position. They also signal intra-articular or atmospheric pressure changes, and the direction, amplitude and velocity of actively or passively produced joint movements by increasing their rate of discharge with those types of stimulation.

Type II mechanoreceptors are thickly encapsulated conical corpuscles resembling Pacinian corpuscles. They are located in the deeper layers of the facet joint capsules, throughout the entire annulus fibrosus in the cervical intervertebral discs, and in the outer one-third of the annulus in the lumbar discs.

Type II mechanoreceptors are inactive in immobile joints and become active in short bursts at the beginning and ending of joint movement, signaling the acceleration or deceleration of joint movement whether produced actively or passively.

Along with Type I receptors, Type II mechanoreceptors are responsible for the maintenance and modification of coordinated joint movements.

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Type III mechanoreceptors are thinly encapsulated fusiform corpuscles resembling the Golgi tendon organ. In the spine, Type III mechanoreceptors are located predominantly in the intertransverse ligaments, throughout the annular fibers of the cervical intervertebral discs, and in the outer one-third of the annulus in the lumbar discs.

The signals generated by Type III receptors function to inhibit the muscles operating over a specific joint, serving to protect joint muscles from damaging tensile forces.

THE NEUROLOGICAL PATHWAY OF PAIN

Before proceeding to the proposed mechanism of pain modulation produced by spinal manipulation, it might be helpful to review the pathway taken by nociceptive signals entering the spinal cord.

Nociceptive afferent fibers (pain receptor fibers) from peripheral tissues enter the spinal cord through the dorsal nerve roots where they synapse with a second order neuron in the posterior gray horn. The axon of the second order neuron then crosses to the opposite side of the cord where it becomes a component of the lateral spinothalamic tract. The axon of the second order neuron then passes upward to the thalamus where the conscious recognition of pain occurs. The second order neuron then synapses in the thalamus with a third order neuron which sends fibers to the somesthetic area of the cerebral cortex. There the information is analyzed for the precise source, severity and quality of the pain(19) (see Figure 1).

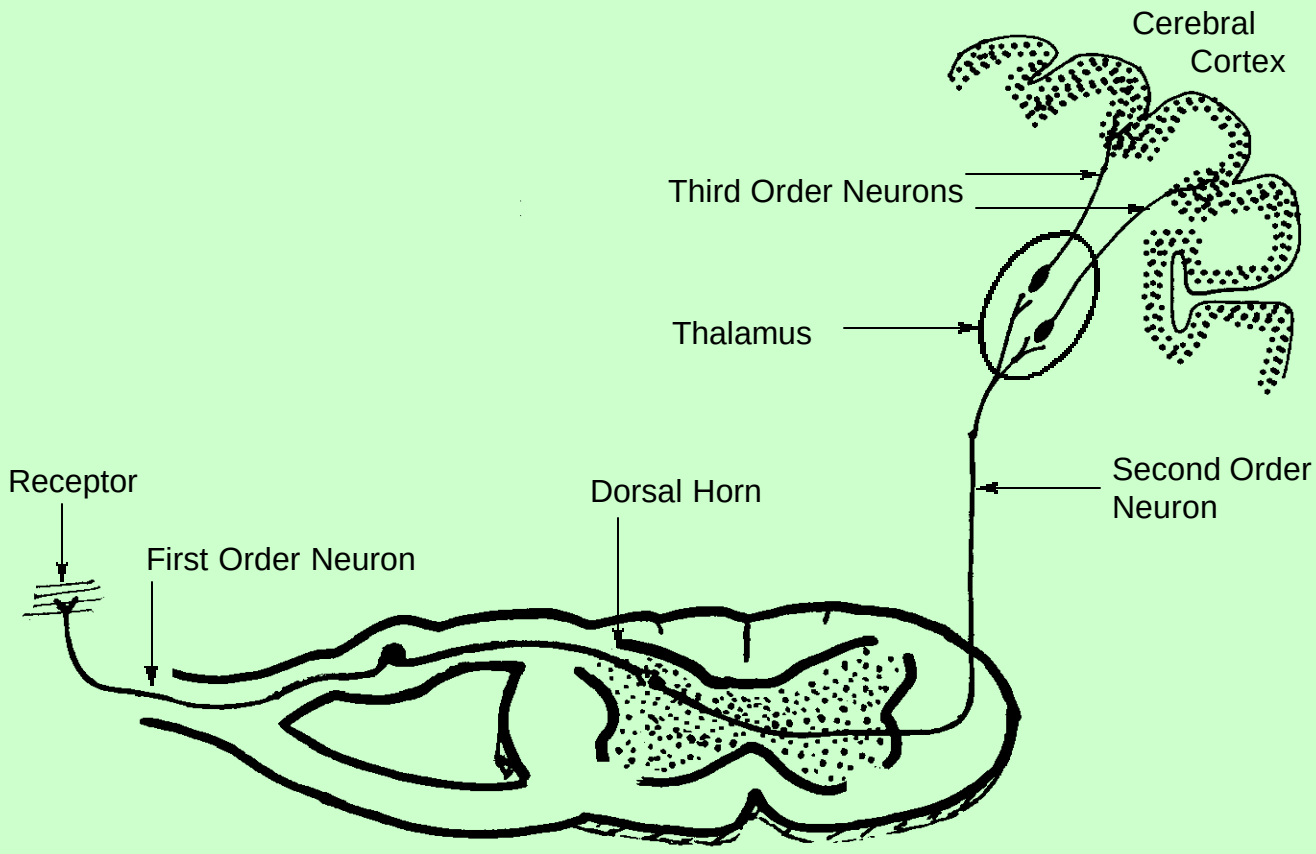

Figure 1. Neural pain pathway to the brain. An incoming nociceptive signal travels through the dorsal nerve root to the posterior gray horn of the spinal cord. It then synapses on a second order neuron and crosses to the opposite side of the spinal cord where it becomes a component of the lateral spinothalamic tract before ascending through the thalamus to the somesthetic area of the cerebral cortex.

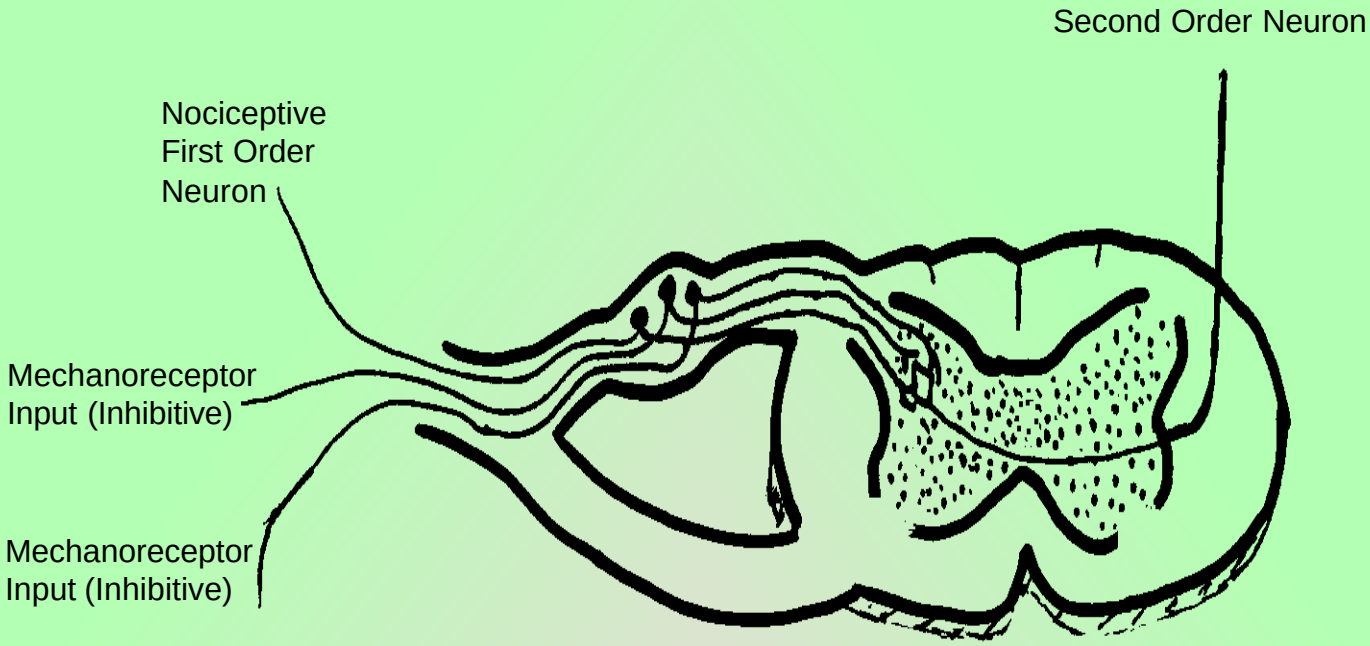

Figure 2. Inhibition of second order nociceptive neuron by mechanoreceptor activity. As mechanoreceptor afferent fibers enter the spinal cord, collateral branches synapse on second order nociceptive neurons and exert an inhibitory effect upon further generation of pain signals to higher brain centers.

PERIPHERAL MODULATION OF PAIN

As each mechanoreceptor fiber enters the spinal cord through the dorsal spinal nerve root, it not only communicates with interneurons that will eventually make connections to the somesthetic cortex and cerbellum, but also gives off collateral branches that pass into the posterior gray horn. There it terminates on the presynaptic terminals of the second order nociceptive neurons upon which they exert and inhibitory effect. This inhibitory effect is thought to prevent the transmission of nociceptive signals to higher brain centers. In other words, activity in the mechanoreceptor relay system, initiated by passive joint movements (manipulation) results in presynaptic inhibition of the nociceptive afferent activity of the second order nociceptive neurons(1,5,10,11,16-18) (see Figure 2). The probablitiy that peripheral tissue damage will evoke the experience of pain is less likely as the ongoing frequency of discharge in the mechanoreceptor afferents coming from the affected or more remote tissues is being stimulated.

It is for this reason that clinical procedures which enhance the frequency of discharge from tissue mechanoreceptors, such as superficial and deep tissue massage, passive joint manipulation and active exercise diminish the intensity of many varieties of pain, or even abolish it altogether. However, this effect will not be long lasting if the provoked mechanoreceptor afferent discharge is not of a high enough frequency or is not maintained for periods of long enough duration.

CONCLUSION

The preceding information illustrates the proposed mechanism of pain modulation as a result of the inhibitory effects of joint mechanoreceptors on nociceptive interneurons in the posterior gray horn of the spinal cord. From what has been presented it may be concluded that joint manipulation may be an effective aid in the reduction or relief of pain, along with other active forms of treatment such as range of motion and active resistive exercise.

Although the exact mechanism of pain relief secondary to spinal manipulative therapy is not conclusively know at this time, scientific studies are beginning to unveil the likely neurological components responsible for the pain relieving effects of this ancient manual healing art.

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