How ART® Treatment Works

Active Release Techniques® treatment has several mechanisms by which it affects a response. Several studies that have measured associated responses of hypoalgesia and sympathetic activity following soft-tissue therapy suggest a mechanism of action mediated by the periaquaductal gray as noted by Wright, as well as lessening of temporal summation, suggesting a mechanism mediated by the dorsal horn of the spinal cord, as per George et al. Musculoskeletal injuries induce an inflammatory response in the periphery which initiates the healing process and influences pain processing. Inflammatory mediators and peripheral nociceptors interact in response to both injury and soft-tissue therapy and they can directly affect this process. In 2006, Teodorczyk-Injeyan et al., observed a significant reduction of cytokine levels in both blood and serum in individuals receiving soft-tissue therapy that was not observed in the control group. Changes of blood levels of β–endorphin and serotonin were noted by Degenhardt et al. and McPartland et al. following soft-tissue therapy. Additionally, soft-tissue therapy has been shown to alter acute inflammation in response to exercise and substance P levels as noted by Smith et al. and Field et al. When pooled together, these studies suggest a peripheral nervous system-mediated mechanism of action on musculoskeletal pain from an application of ART® treatment.

ART® treatment also exerts an effect on the spinal cord. Authors have noted hypoalgesia (George et al., Mohammadian et al. and Vicenzino et al.), afferent discharge (Colloca et al.), motor neuron pool activity (Bulbulian et al. and Dishman & Burke), and changes in muscle activity (Herzog et al. and Symons et al.). In an examination of the human fascia profunda of the leg in 1996, Staubesand and Li documented the presence of smooth muscle-like cells in addition to a rich presence of sympathetic nerve fibers. This fortified the idea of a strong link between the autonomic nervous system (ANS) and fascial tone. Increased palpatory "tightness" has been noted in those experiencing psychological stress, despite these changes being electrically silent during electromyography (Basmajian & DeLuca). The suggestion by several authors, including Masi and Hannon, is that human resting muscle tone may be significantly influenced by changes in fascial stiffness. The link, noted by Bhowmick et al. is that sympathetic activation induces an increased TGF-b1 expression. This cytokine, along with platelet derived growth factor, is known as the most potent stimulator of myofibroblast contraction, which leads to increased fascial contractility. It is important to note that this is a two-way interaction, such that fascial tone can also influence the ANS. Stimulation of non-nociceptive free nerve endings can influence sympathetic tone and Ruffini corpuscles that respond to slow shear and, thus, decrease sympathetic activation. This information collectively implicates a spinal cord-mediated effect.

Finally, direct support for a supraspinal mechanism of action of ART® treatment comes from Malisza et al. They used functional MRI to assess the activity of the supraspinal region following capsaicin injection in the lower extremity of rats as compared to baseline and then reassessed it after applying soft-tissue therapy. The outcome measure was to quantify the response of the hind paw to light touch. A trend was noted towards decreased activation of the supraspinal regions responsible for central pain processing.

In addition to neurophysiological mechanisms, biochemical processes have also been theorized to coexist with the mechanical stimulation. Work by Hou et al. have suggested that analgesia may be the result of a reactive hyperemia in the affected tissue which may, in fact, be modulated by Type IV mechanoreceptors found in the tissues being mechanically stimulated (Schleip). With the mechanical stimulation of tissue to generate an analgesic effect, the release of endocannibinoids is an emerging topic of discussion.

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