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Regular spinal manipulation therapy may enhance athlete performance even in the athlete who has no symptoms but whose performance is or remains hindered

Significant weakness has been found in healthy uninjured muscles which have shared innervation with a dysfunctional joint. Despite strengthening of the weak muscle, no significant improvements were shown (Rice and McNair, 2010: 250; Rossi et al., 2002: 523). This is due to an ongoing inhibition that prevents the weak muscle from being fully activated; a phenomenon known as arthrogenic muscle inhibition (AMI). A weak correlation has been established between pain and AMI; several studies have affirmed the presence of significant AMI in the absence of pain. As little as 10 ml of fluid infused into joints can cause notable muscle inhibition, and even small, clinically undetectable joint effusions can cause significant AMI (Hopkins, 2006: 177; Hopkins et al., 2001: 123; Rice et al., 2014: 504; Rice and McNair, 2010: 254).

Arthrogenic muscle inhibition can reduce athletic performance (Joyce and Lewindon, 2015: 6) and hinder any treatment or rehabilitation outcomes (Rice and McNair, 2010: 250; Rossi et al., 2002: 523). Treatments that specifically aids in the reduction of AMI could be an important tool in athletic performance and to aid musculoskeletal therapists in improving treatment and rehabilitation outcomes that would have been impeded by the inhibition (Rice and McNair, 2010: 250; Rossi et al., 2002: 523). Many studies have showed immediately after spinal manipulation therapy; an increase in muscle activity (electromyography) (DeVocht et al., 2005: 465; Pickar, 2002: 364; Colloca and Keller, 2001: 489; Herzog et al., 1999: 146; Olsen, 2015: 86) and a significant increase in muscle strength and decrease in AMI (Dunning and Rushton, 2009: 512; Pickar, 2002: 358; Suter and McMorland, 2002: 541; Suter et al., 2000: 385) of the segmentally innervated muscles.

Raised Aβ-fiber afferent discharge from type I-III nerve endings (mechanoreceptors) in joint tissue is strongly associated with AMI and it is postulated that joint afferent input has competing excitatory and inhibitory influences on the homonymous motor neuron pool; in a dysfunctional joint, the net effect can be inhibitory (Konishi et al., 2003: 1805; Rice and McNair, 2010: 255). Repetitive postural strain or overuse of the spine or trauma to the spine can cause alterations in the normal anatomical, physiological and biomechanical dynamics of individual vertebral segments and produce relatively large vertebral motions that achieve a new position of stable equilibrium. The higher energy level needed to achieve the new position of stable equilibrium can place additional mechanical stress or overload on the facet joint capsular tissue and cause uneven or increased unilateral facet joint loading (Gatterman, 2005: 8; Pickar, 2002: 364; Vernon, 2010: 29). These alterations in the vertebral segment can cause tension, pressure, stretching or irritation of the facet joint capsular tissue as well as the displacement of collagen in the facet joint capsular ligament (Gatterman, 2005: 8; Vernon, 2010: 29), and thereby depolarize and sensitize mechanoreceptors within the facet joint tissue and subsequently increase their Aβ-fiber afferent discharge frequency (Dunning and Rushton, 2009: 512; Vernon, 2010: 29), in the presence and absence of pain (Rice et al., 2014: 504; Rice and McNair, 2010: 254). The raised Aβ-fiber afferent discharge from facet joint tissue can cause impairment of its arthrokinetic reflex functioning and produce abnormal patterns of spinal reflex arc activity (Middleditch and Oliver, 2005: 247) and thereby result in weakness of the spinal segmental innervated muscles (Middleditch and Oliver, 2005: 247; Porter, 2013: 581).

Spinal manipulation therapy is a high-velocity low-amplitude thrust delivered at the end range of motion of facet joints in the spine, in the direction of the orientation of the facet joint articulation, and is often accompanied with an audible cracking sound (Millan et al., 2012: 24; Pickar, 2002: 35). Spinal manipulation may improve alpha motor neuron functioning in the symptomatic (DeVocht et al., 2005: 465; Keller and Colloca, 2000: 585; Suter and McMorland, 2002: 541; Suter et al., 2000: 385) and asymptomatic (Dunning and Rushton, 2009: 508; Olsen, 2015: 86; Pickar, 2002: 364) individual, by desensitizing altered arthrokinetic spinal reflex activity over time (Victor 2016) via stimulating mechanoreceptors in the vertebral segment's facet joint tissue (DePalma, 2011: 81; Millan et al., 2012: 24; Pickar, 2002: 35; Sterling and Kenardy, 2011: 19); and by causing disinhibition of the homonymous lower motor neurons (Dunning and Rushton, 2009: 512; Olsen, 2015: 86; Suter and McMorland, 2002: 541) via mechanically reducing alterations present in the biomechanical dynamics of the vertebral segment (Dunning and Rushton, 2009: 512; Gatterman, 2005: 8; Vernon, 2010: 29) which caused the aberrant afferent discharge from the somatic receptors in the vertebral segment’s facet joint tissue and consequently AMI (Victor 2016).