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Topical Review

Introduction to Myofascial Trigger Points in Dogs

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In dogs, muscles make up 44%-57% of total body weight and can serve as source of both pain and dysfunction when myofascial trigger points are present. However, rarely is muscle mentioned as a generator of pain in dogs, and even less mentioned is muscle dysfunction. The veterinary practitioner with interest in pain management, rehabilitation, orthopedics, and sports medicine must be familiar with the characteristics, etiology, and precipitating factors of myofascial trigger points. Additionally, the development of examination and treatment skill is needed to effectively manage myofascial trigger points in dogs.

Introduction

A myofascial trigger point (MTP) can be defined as a hyperirritable spot located within a taut band in skeletal muscle.[1] The presence of MTPs within muscle can be a source of pain and dysfunction in dogs; however, limited information exists in veterinary literature. The emergence of the disciplines of veterinary rehabilitation and sports medicine demand a better understanding of the role MTPs play in muscle pain and dysfunction.

Characteristics of MTPs

MTPs have the following 3 major characteristics: sensory, motor and autonomic. The muscle pain or myalgia associated with MTPs is described in humans as diffuse, deep, and difficult to localize with defined referred pain patterns. When the MTP is stimulated manually, a localized pain is appreciated. Very often, palpation of a MTP in dogs can result in a "jump sign," a pain response resulting in vocalization or withdrawal as pressure is applied. Other sensory aspects include peripheral and central sensitization. Peripheral sensitization can be described as a reduction in the threshold and an increase in the responsiveness of the peripheral ends of nociceptors. Central sensitization is best explained as a physiochemical change resulting in an increase in excitability of neurons within the central nervous system.[2]

Motor abnormalities of MTPs include the development of a taut band within the muscle, a local twitch response (LTR) with stimulation, muscle weakness without atrophy, and loss of reciprocal inhibition.[3] The taut band is a localized, linear, discrete band of hardened muscle within the softer homogeneous muscle. Taut bands are identified running parallel to muscle fibers and can be described as a localized contracture within the muscle without nerve-initiated activation of the motor end plate or neuromuscular junction.[4] To contrast the taut band from muscle spasm, the later is the result of increased neuromuscular tone of the entire muscle due to a nerve-initiated contraction. The contracture associated with the taut band also results in reduction in range of motion. The MTP is located within the taut band and is what distinguishes it from other painful areas within muscle.[5]

The LTR is another motor component of MTPs. The LTR is a unique spinal cord reflex resulting in a rapid contraction of the taut band following manual stimulation of the MTP. Manual stimulation can be accomplished by direct palpation or introduction of a needle. The LTR in dogs can also serve as verification of the presence of a MTP.

In people, weakness is recognized in muscles that have MTPs. This weakness occurs without atrophy and is not related to neuropathy or myopathy.[1] Weakness is usually rapidly reversible immediately on inactivation of the MTP. This rapid reversal suggests weakness is caused by inhibition of muscle activation. It is also recognized that a MTP in one muscle can inhibit effort or contractile force in another muscle, suggesting a central inhibition process.[5] Additional motor or muscle dysfunction from MTPs is brought about by disordered recruitment or altered muscle activation patterns in muscles that work together to produce a specific action.[5,6]

Reciprocal inhibition is defined as the inhibition of antagonist muscle contraction during contraction of agonist muscle. This central inhibition of muscle activity results in coordinated quality movement. Reciprocal inhibition becomes reduced when the agonist or the activated muscle contains a MTP, resulting in co-contraction and subsequent altered gait and decreased quality of movement.

In dogs, simple tests can be performed to evaluate muscle weakness. With a dog in standing position, slowly slide limb backward until non–weight bearing. A slight to profound drop of the contralateral side can be indicative of muscle weakness or altered muscle firing patterns associated with MTPs within the antigravity muscles of that limb. Sit-to-stand exercise can serve as a subjective assessment of weakness by observation of a dog's ability to sit and stand. Video analysis of gait patterns and quality of gait may be helpful when pretherapy and posttherapy videos are compared.

Etiology of MTPs

Trigger points are composed of contraction knots that can be described as segments of muscle fiber with intensely contracted

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sarcomeres and increased diameter. It remains unclear why these contraction knots form; however the hypothesis, known as the *Integrated Trigger Point Hypothesis*, postulates a problem at the motor end plate resulting in excessive release of acetylcholine. This excess of acetylcholine results in sarcomere shortening that has been observed histopathologically.[7] Contraction knots or areas of concentrated focal sarcomere contraction have been described in animals and humans.[8]

A better appreciation of the potential causes or etiologies of MTP formation in dogs will aid the clinician in recognition and therapy. Muscle-related mechanisms associated with the development of MTPs, in people and animals, include muscle overuse or overload and direct trauma. Low-level muscle contractions, uneven intramuscular pressure distribution, direct trauma, unaccustomed eccentric contractions, eccentric contraction in unconditioned muscle, and maximal or submaximal concentric contractions can lead to muscle injury and subsequent development of MTPs.[2,9]

The *Integrated Trigger Point Hypothesis*, postulated in 1981, was the first scientific hypothesis to explain the formation of taut bands and MTPs based on both electrophysiological and histopathologic data.[10] The hypothesis postulates that muscle injury leads to increased calcium concentrations outside the sarcoplasmic reticulum, possibly because of mechanical rupture of the sarcoplasmic reticulum or the sarcolemma. Increased calcium concentrations result in sustained muscle fiber contraction. This hypothesis was later refined in 2004 to include a dysfunctional motor end plate occurring secondary to muscle injury and resulting in excessive release of acetylcholine.[11] Sustained maximal contraction of the muscle fibers in the vicinity of the dysfunctional end plate causes increased metabolic demand and decreased concentrations of adenosine triphosphate. The calcium pump that returns calcium to the sarcoplasmic reticulum is adenosine triphosphate dependent, as is the uncrosslinking of actin and myosin, thus calcium concentrations and contractile activity remain increased.

Muscle injury alters the normal equilibrium between the release and breakdown of acetylcholine and its removal by acetylcholinesterase from acetylcholine receptors in the postsynaptic membrane. Neuropeptides such as calcitonin gene–related peptide and substance P, released during muscle injury, facilitate increased release of acetylcholine, inhibition of breakdown, and upregulation of acetylcholine receptors.[11] A persistent muscle fiber contraction develops, leading to the development of the taut band and subsequent MTP.

Perpetuating Factors in the Development of MTPs

Perpetuation of MTP formation in dogs is most often because of mechanical stresses resulting in chronic muscle overload. Postural changes in dogs resulting from orthopedic injury, postoperative surgical trauma and pain, neuropathy, joint dysfunction, and pain related to osteoarthritis create muscle overload. Many of the same muscle-related mechanisms that lead to development of MTPs also perpetuate them.

Chronic osteoarthritis creates compensatory postural changes that activate and perpetuate MTPs in numerous muscles. Moderate to severe osteoarthritis of the coxofemoral joints activates and perpetuates MTPs in the functional unit muscles of the coxofemoral joint, flexors (including iliopsoas), adductors, and extensors. The cranial shift in weight overloads muscles in the thoracic limbs, namely the musculus infraspinatus, the deltoideus muscle, and the long head of the musculus triceps brachii. Repeated lateral flexion of the spine, which assists in ambulation by advancing the pelvis and pelvic limb while limiting coxofemoral flexion and extension, results in overloading of the musculus iliocostalis lumborum. A dog with a non–weight-bearing pelvic limb adopts hopping actions in the contralateral pelvic limb, resulting in unaccustomed eccentric contractions of the coxofemoral and stifle extensors in an attempt to limit flexion. Lumbar paraspinal muscles become overloaded, as they must now assist with ambulation and not just spinal stabilization. The musculus iliopsoas, which is actually 2 separate muscles, the musculus psoas major and musculus iliacus, develops MTPs and contracture, resulting in a kyphotic posture.

In people, additional perpetuating factors are identified: nutritional, metabolic, nerve impingement, and visceral-somatic pain. Nutritional insufficiency of vitamin B_{12} (cobalamin) and folic acid have been described as perpetuating factors for myofascial pain syndrome.[1,2,5] There are no references in the veterinary literature pertaining to insufficiency of these substances causing pain of any type; however, cobalamin insufficiency is reported to cause malaise and failure to thrive. Both substances currently have clinical application as markers for small bowel disease. Cobalamin insufficiency in dogs may also occur with exocrine pancreatic insufficiency.

Hypothyroidism, a metabolic and endocrine disorder, is recognized in people as a perpetuating factor for MTP. Clinical signs in people include myalgia, stiffness, weakness, cramps, and pain on exertion.[1] Hypothyroidism is the most common endocrine disorder in dogs and is associated with a variety of clinical signs; however, veterinary literature does not mention pain as a consequence of hypothyroidism.

In people, visceral pain can activate and perpetuate MTPs in the area of referred pain.[12] Neurons in the dorsal horn of the spinal cord receive input from the viscera and from receptors in the skin and deeper soft tissues. As a result of this overlap, visceral nociceptive activation of the dorsal horn neurons may result in muscle pain and may also be a cause of MTPs in animals.

Examination for MTPs

Examination of muscles for myalgia is not part of the standard physical, orthopedic, or neurologic examination in veterinary medicine. Identification of taut bands and hypersensitive MTP within muscle is an acquired skill that requires an understanding of these changes, skilled instruction, and repeated practice.

There are 2 basic palpation techniques employed in a myofascial examination:

Flat palpation: Examination by finger pressure that proceeds across the muscle fibers at a right angle to their length while compressing them against a firm underlying structure, such as a bone. This technique could be used for the musculus infraspinatus, musculus supraspinatus, and musculus psoas major (Fig 1).

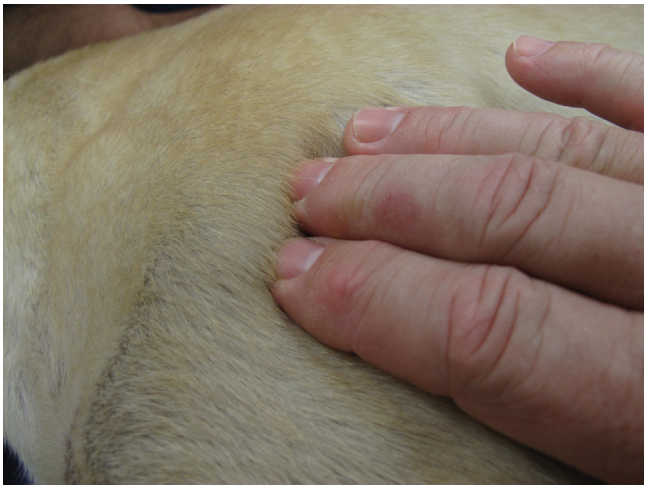

Fig. 1. Flat palpation of a muscle.