Introduction to trigger points

D Prattley
AcuVet Ltd

What is a trigger point?

A myofascial trigger point (TrP) is defined as "… a hyperirritable spot in skeletal muscle that is associated with a hypersensitive palpable nodule in a taut band. The spot is tender when pressed, and can give rise to the characteristic referred pain, motor dysfunction, and autonomic phenomena…" (Simons *et al.* 1999). In humans, characteristic features of TrPs are (Bennett 2007):

1. Focal point of tenderness on palpation of the affected muscle.
2. Reproduction of the familiar pain using approximately 3kg of pressure to palpate the TrP.
3. A palpable taut band in the muscle.
4. Restricted range of motion in the affected muscle.
5. Possible pseudo-weakness of the affected muscle (without atrophy).
6. Often referred pain on continued pressure over the TrP (approximately five seconds).

The taut band of muscle is a localised area of muscle contraction, known as a contraction knot, which consists of contracted sarcomere units. This causes a slight overall shortening of the muscle and decreased range of motion, as the muscle is unable to be completely stretched. Overall muscle contraction might also be compromised. Ibarra *et al.* (2011) produced evidence for increased EMG activity in muscles antagonistic to contracting agonists containing stimulated TrPs.

When pressed, a TrP can cause a local twitch response as muscle fibres contract and relax. The patient can also exhibit a jump sign, or sudden involuntary movement. Animals can exhibit a range of signs indicating that they feel pain, for example growling, yelping, biting, flinching or moving away, although in some individuals the signs can be rather more subtle.

In humans, TrPs cause referred pain that occurs in consistent patterns, which are not dermatomal. For example, quadratus lumborum TrPs refer pain to the posterior iliac crest/upper gluteal region. Upper trapezius trigger points refer upwards along the ipsilateral side of the neck to the back of the ear and to the temple and can be responsible for headaches. TrP referred pain is generally deep, dull, aching and diffuse.

It has been shown that TrPs are affected by, and affect, autonomic activity. Ge *et al.* (2006) showed that increased sympathetic outflow resulted in a reduction of pain caused by pressure, a decreased referred pain threshold and increased pain intensity at both the local and referred sites. Reduced skin blood flow following stimulation of latent TrPs is thought to be due to increased sympathetic vasoconstriction (Zhang *et al.* 2009).

Classifications of trigger points

TrPs can be classified as follows (Lindley and Cummings 2006)

Active: causing a clinical complaint of pain even when not being palpated.
Latent: capable of causing pain when palpated, but does not cause spontaneous pain. Latent points can also cause

restriction to movement, and can become active.
Primary: a TrP activated by conditions occurring in the muscle it is located in, for example as a result of muscle strain.
Secondary: occurring as a consequence of a primary TrP or the dysfunction caused by it, or as a consequence of other pathology.
Satellite: a secondary TrP occurring in the referred pain area of a primary TrP or other pathology.
In veterinary work TrP's can be challenging to classify. For example, distinguishing between latent and active TrPs requires knowing whether or not the point is causing spontaneous pain and this is usually difficult to determine.

Aetiology and pathophysiology of trigger points

Overuse and trauma of muscle are generally accepted causes of TrPs in people, and are likely causes in animals. Muscle fatigue or overload, for example from eccentric contraction or from repetitive or sustained contractions, are thought to contribute to TrP formation (Bron and Dommerholt 2012). Contractions at 10-25% of muscle capacity can cause sufficiently increased intramuscular pressure that capillary blood flow is compromised, and local ischaemia is considered an important factor in TrP formation (Bron and Dommerholt 2012).

The integrated trigger point hypothesis is the primary current theory explaining the pathophysiology of TrPs and is based on available evidence (discussed in Dommerholt and Huijbregts 2011). Excess acetylcholine release is thought to cause high-frequency endplate noise, characterising dysfunctional motor endplates. Intracellular calcium levels are increased as a result of acetylcholine affecting sodium channels, and this generates persistent localised muscle contracture. This in turn compromises local circulation, resulting in hypoxia which triggers release of mediators that stimulate muscle nociceptors and cause pain. Peripheral and autonomic nerves can be stimulated, along with second-order neurons, followed by central sensitisation, referred pain, hyperalgesia and formation of new receptive fields. An alternative theory (Hocking 2010) proposes that alpha motor neuron function is abnormal, with altered regulation of calcium and potassium channels causing increased calcium levels that result in acetylcholine release.

Treating trigger points

In veterinary patients it is recommended that muscles under the highest biomechanical strain during normal activities and those near areas of pathology be closely examined for the presence of TrPs (Lindley and Cummings 2006). Areas compensating for pathology (e.g. muscles in limbs that are given increased loading during lameness) should also be checked. Careful palpation of muscle tissue is required, and is usually carried out perpendicular to the direction of muscle fibres.

A variety of methods have been used to deactivate TrPs in people, with varying efficacies (discussed in Dommerholt and Huijbregts 2011). There was a paucity of well-conducted clinical trials evaluating non-invasive treatments. Transcutaneous electrical stimulation was considered to be effective in the short term but post-treatment effects were not determined. Manual therapies studied included a variety of techniques and treatment protocols with some being supported. Conflicting evidence exists for efficacy of laser therapy, while magnetic therapies required further research. Conventional ultrasound was considered ineffective.

Dry needling of TrPs is considered effective, and can be performed with either superficial or deep insertion of the needle. Deep needling is intended to penetrate the TrP, requiring refined palpation skills, while in superficial needling the needle is inserted over but not into the TrP.

In veterinary treatment of TrPs, dry needling and stretching are considered efficient (Lindley and Cummings 2006). Pressure and stretch can also be used although sufficient pressure to deactivate the TrP can cause excessive pain responses, thereby requiring alternative techniques to be used. Transcutaneous electrical stimulation followed by stretching is also practised.

Postural training is considered an important part of TrP management in people if postural disorders are considered as having contributed to TrP formation (Dommerholt and Huijbregts 2011). Similar considerations should be applied to animals being treated for TrPs and a variety of techniques can be used for rehabilitation.

References

Bennett R. Myofascial pain syndromes and their evaluation. *Best Practice & Research Clinical Rheumatology* 21 (3), 427-45, 2007

Bron C, Dommerholt JD. Etiology of myofascial trigger points. *Current Pain and Headache Reports* 16, 439-44, 2012

Dommerholt J, Huijbregts P. Myofascial Trigger Points, Pathophysiology and Evidence-Informed Diagnosis and Management. *Jones and Bartlett Publishers* 2011

Ge HY, Fernandez-de-las-Penas C, Arendt-Nielsen L. Sympathetic facilitation of hyperalgesia evoked from myofascial tender and trigger points in patients with unilateral shoulder pain. *Clinical Neurophysiology* 117, 1545-50, 2006

Hocking MJL. Trigger points and central modulation – a new hypothesis. *Journal of Musculoskeletal Pain* 18, 186-203, 2010

Ibarra JM, Ge HY, Wang C, Vizcaino VM, Graven-Nielsen T, Arendt-Nielsen L. Latent myofascial trigger points are associated with an increased antagonistic muscle activity during agonist muscle contraction. *The Journal of Pain* 12, 12, 2011

Lindley S, Cummings TM. Essentials of Western Veterinary Acupuncture. Blackwell Publishing, 2006

Simons DG, Travell JC, Simons LS. *Travell and Simons' Myofascial Pain and Dysfunction: The Trigger Point Manual. Volume 1: Upper Half of Body. 2nd ed.* Baltimore, MD: Williams & Wilkins 1999

Zhang Y, Ge HY, Yue SW, Kimura Y, Arendt-Nielsen L. Attenuated skin blood flow response to nociceptive stimulation of latent myofascial trigger points. *Archives of Physical Medicine and Rehabilitation* 90, 2009