Journal of Bodywork & Movement Therapies (2015) xx, 1–12

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INVITED CRITICAL REVIEW

A critical evaluation of Quintner et al: Missing the point

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Received 26 January 2014; received in revised form 29 January 2014; accepted 30 January 2014

KEYWORDS
Myofascial pain;
Trigger points;
Trigger point hypotheses

Summary The objective of this article is to critically analyze a recent publication by Quinter, Bove and Cohen, published in Rheumatology, about myofascial pain syndrome and trigger points (Quintner et al., 2014). The authors concluded that the leading trigger point hypothesis is flawed in reasoning and in science. They claimed to have refuted the trigger point hypothesis. The current paper demonstrates that the Quintner et al. paper is a biased review of the literature replete with unsupported opinions and accusations. In summary, Quintner et al. have not presented any convincing evidence to believe that the Integrated TrP Hypothesis should be laid to rest.

Introduction

Quintner, Bove and Cohen stated that the objective of their recent paper "A critical evaluation of the trigger point phenomenon" (Quintner et al., 2014) was to demonstrate that the theory of myofascial pain is flawed in both reasoning and science. The Quintner et al. paper can be downloaded at no cost at http://www.painaustralia.org.au/images/pain_australia/Rheumatology-2014-Quintner-rheumatology_keu471.pdf. The authors offered two different hypotheses to replace the current trigger point (TrP) hypothesis. The hypothetical constructs of what this kind of muscle pain may be representing have gone through multiple stages and various points of view as new research emerges (Dommerholt et al., 2006; Gerwin et al., 2004; Simons, 1975, 1976), since British physician Balfour in 1816 described muscle pain as "patients having a large number of nodular tumours and thickenings which were painful to the touch, and from which pains shot to neighboring parts" (Stockman, 1904). We appreciate

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http://dx.doi.org/10.1016/j.jbmt.2015.01.009
1360-8592/

Please cite this article in press as: Dommerholt, J., Gerwin, R.D., A critical evaluation of Quintner et al: Missing the point, Journal of Bodywork & Movement Therapies (2015), http://dx.doi.org/10.1016/j.jbmt.2015.01.009

Quintner et al.'s efforts to critically review the current hypothetical constructs of myofascial pain and TrPs and to offer alternative perspectives and hypotheses, which eventually may lead to a better understanding of myofascial pain, although we disagree with many of their specific comments.

In spite of years of research into the nature of myofascial pain and significant gains especially during the past decade, several aspects remain elusive and are not well understood. A distinct mechanistic understanding of this disorder does not yet exist (Jafri, 2014). Recently, Jafri also proposed a new TrP hypothesis and it is striking to observe the different approach he took to share his novel ideas (Jafri, 2014). Where Quinter et al. seem mostly interested in boldly refuting the entire integrated TrP hypothesis of myofascial pain as "an invention without a scientific basis," Jafri recognizes many aspects of the integrated TrP hypothesis and extends it. More importantly, they have very different interpretations of the literature. In addition to the expanded Integrated TrP Hypothesis (Gerwin et al., 2004), several alternative hypotheses have been proposed, such as the Central Modulation Hypothesis (Hocking, 2013, 2010), the Neurogenic Hypothesis (Srbely, 2010), the Neurophysiologic Hypothesis (Partanen et al., 2010), and the Radiculopathy Hypothesis (Gunn, 1997).

Quintner, Bove and Cohen have made significant contributions to the scientific literature (Bove, 2008, 2009; Quintner and Bove, 2001; Cohen et al., 2013, 2011; Quintner et al., 2008). In this paper, they criticize the hypothesis of TrP formation as put forth initially by Simons and Travell, and later modified by others (Gerwin et al., 2004; Simons, 1996; McPartland, 2004; McPartland and Simons, 2006). In doing so, they specifically discredit much of the research on myofascial TrPs that has been published as unreliable, without providing any alternative studies specifically done on the pain phenomena that is attributed to TrPs. Moreover in the current paper they use the terms "hypothesis" and "theory" uncritically. They use the term "theory" in a non-scientific manner that is rather confusing in a scientific paper (Popper, 2002). Already in the first sentence of the abstract, the authors mention "the theory of myofascial pain syndrome (MPS) caused by trigger points (TrPs) … " and elsewhere in their paper, they expressed that "the theory is flawed … " and that "the theory of MPS caused by TrPs has been refuted."

Scientific inquiry commonly starts with observations, followed by the development of hypotheses, which through experiments are confirmed, modified, or refuted. A hypothesis suggests a mechanism and leads to experiments to either support the hypothesis or not. Through repeated experimental testing of the hypothesis, it is continually refined until a theoretical basis can be constructed that addresses different aspects of the hypotheses. The end goal of the process is to construct a scientific theory. Few, if any, phenomena in medicine have reached the stage of scientific theory, including the existing TrP hypotheses. This makes it even more puzzling that Quintner et al. criticized our 2004 publication in which we reviewed recent research findings to expand the hypothetical thinking at the time. We did not present new data as dogma, but followed the scientific process of re-evaluating the Integrated TrP Hypothesis as new data became available (Simons, 1996, 2001; Gerwin et al., 2004). In truth, hypotheses are just hypotheses and they are put forth to explain certain observations and to lead to further studies. Is that not what the scientific process is all about?

Quintner et al. take issue with the concept of TrPs as a cause of muscle pain. They deny the existence of muscle pain related to TrPs, although worldwide, clinicians report finding these clinically as the authors dutifully acknowledged. To the contrary, Quintner et al. claim that focal areas of muscle pain defined as associated with TrPs cannot be reliably identified. They offered an alternative explanation for such focal pain, but do not specify whether there would be any palpable areas of hardness. Although there is indeed a rich literature investigating and supporting the concept of myofascial TrP pain (Jafri, 2014), Quintner et al. misrepresent or discount much of the data, and fail to adduce similar data to support their own hypotheses. Moreover, their literature review is outdated, as less than 10% of the articles cited in their review accepted in October 2014 were published in or after 2011. Our objective is to critically analyze the Quinter et al. paper, point out its strengths and its flaws, and as such contribute to the scientific literature and thinking about myofascial TrPs.

Scientific basis vs. non-scientific bias

Quintner et al.'s paper is a biased review of the literature replete with unsupported opinions and accusations. The article is comprised of different sections, starting with the "evolution of MPS theory", followed by a "review of the evidence", and a final section in which the authors revisited two previously suggested explanatory models, including a neuritis model and a secondary allodynia model.

In the section "evolution of MPS theory" the authors used pejorative terms and expressions like "speculation" as in "*speculation* took a new turn when Travell and Rinzler conceptualized that pain felt in voluntary muscles is myofascial in origin", or "Travell and Simons *found it necessary to invent* the latent TrP …" (italics added). Travell simply attempted to develop a reasonable and testable hypothesis based on her clinical observations. Is conceptualization not an essential component of developing a hypothesis within the context of scientific inquiry? In 1981, Simons and Travell published "Myofascial trigger points, a possible explanation" in which they presented a TrP hypothesis predating the current Integrated TrP Hypothesis (Simons and Travell, 1981). As the title of the paper indicates, Simons and Travell were merely interested in developing a testable hypothesis without resorting to dogma and without suggesting that they had solved all dilemmas prior to formulating a scientific TrP theory.

In the evolution section, Quintner et al. presented several antiquated concepts, which, while perhaps of historical interest, have no significance in the current debate. For example, they cited Stockman, who over 100 years ago did not provide evidence for his hypothesis (Stockman, 1904). While it is historically correct that over 50 years ago several clinicians, including Travell, considered a vicious pain-spasm-pain hypothesis, which assumed that pain would excite alpha-motor neurons and possibly even gamma-motor neurons. More recent experimental and human research showed that both alpha- and gamma-motor

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neurons generally are inhibited by nociceptive input from the same muscle. The pain-spasm-pain cycle has been refuted and has no place in the current scientific thinking (Simons and Mense, 1998; Mense and Skeppar, 1991; Le Pera et al., 2001; Burke, 1983).

The authors concluded the evolution section claiming that "belief in TrP theory and the associated concepts of MPS … exemplify circular reasoning … " quoting their own opinion paper published in 1994 (Quintner and Cohen, 1994). The placement of the authors' opinion at the end of this section has little to do with the evolution of the MPS theory, but interjects the opinion of the authors that the TrP model is flawed even before making any effort to present supporting data. The authors claim that the concept of TrPs is a prime example of circular reasoning: "TrPs cause myofascial pain, because painful muscles contain them." Their argument can easily be applied to other tissues, e.g., arthritis in the knee causes knee pain because some painful knees are arthritic. Similarly, not all painful muscles have TrPs. There are many other painful muscle pathologies, such as various forms of myositis, for example. As the authors' claim is not the basis of the argument that myofascial TrPs cause pain, we will leave this straw-man argument and proceed to more relevant points.

Use of references

Throughout the paper, the authors demonstrated their preconceived bias and objections as evidenced, for example, by their rather selective choice of supporting references and omission of many other relevant studies. When the authors cite Lewit's observation about Travell's referred pain diagrams as "sometimes been chosen arbitrarily, there being no accepted standard" they were misquoting the source document, which stated "When our table of pain spots and trigger zones is compared with those of Travell and Rinzler or Hansen and Schliack, or with the periosteal points of Vogler and Krauss, it is obvious that there are very many such points and that they have sometimes been chosen arbitrarily, there being no accepted standard" (Lewit, 1979). Whereas Lewit expressed nothing but an opinion about three entirely different schools of thought and did not comment at all about diagrams of referred pain, Quintner et al. used a small part of a sentence to discredit Travell and Rinzler's representations of their observations of referred pain (Travell and Rinzler, 1952). In the same paper, Lewit reported, that "dry needling is highly effective in the therapy of chronic myofascial pain. Immediate analgesia without hypesthesia (the needle effect) can be produced by needling precisely the most painful spot", which curiously did not make it into the Quintner et al. paper (Lewit, 1979).

When the authors criticized the Integrated Trigger Point Hypothesis and several of our previous papers in which we described or expanded the hypothesis (Dommerholt et al., 2006; Bron and Dommerholt, 2012; Gerwin et al., 2004), they selected one study of the use of botulinum toxin, that showed no effect on pain intensity or mechanical pain thresholds, while reducing motor endplate activity and electromyographic activity as evidence that the TrP concept is flawed (Qerama et al., 2006). It is somewhat ironic that of all the published botulinum toxin TrP studies, Quintner et al. selected this particular paper, as it has several serious methodological flaws and was poorly interpreted (Simons and Dommerholt, 2007). Therefore, the paper does not provide much support for the Quintner et al. argument. As we, and others, have reported, there is a paucity of good studies on the effectiveness of botulinum toxin, however, overall there is a tilt in favor of the benefit of botulinum toxin in the treatment of trigger point pain (Gerwin, 2012). The literature on the use of botulinum toxin is hampered by the considerable variability in the diagnostic and selection criteria, the dosage of botulinum toxin, the choice of muscles and the number of trigger points, the actual injection techniques, the parameters for control groups, the outcome measures, and the duration of the follow-up period (Gerwin, 2012; Climent et al., 2013; Dommerholt et al., 2015).

In a section labeled "Treatment", the authors selectively pick references to support their claims, while ignoring many other references that do not support their point of view. The authors explained the concept of *post hoc ergo propter hoc* (after this, therefore because of this), which is a common fallacy when a treatment is not matched with the pathogenesis of the condition it is supposed to address. They cited a recent study comparing TrP dry needling to manual TrP therapy published in this journal (Ziaeifar et al., 2014). Ziaeifar et al. acknowledged that the lack of a significant difference between the two groups could be explained by the small sample size or by the relatively small between-group differences (Ziaeifar et al., 2014). Quinter et al. cited the paper as an example of a *post hoc ergo propter hoc* argument.

Towards the end of the paper, Quinter et al. wrote "treatment directed to the putative TrPs elicits a response that is indistinguishable from the placebo effect." The authors did not provide any references in support of their opinion, but they also failed to consider the extensive literature in support of treatments effects beyond the placebo effect (Hains et al., 2010a, 2010b; Anderson et al., 2006, Anderson et al. 2011; Bron et al., 2011; Tekin et al., 2013; France et al., 2014; Kietrys et al., 2013; Mayoral et al., 2013; Fogelman and Kent, 2014). The double-blind controlled dry needling study by Mayoral et al. demonstrated clearly that the treatment of TrPs was superior to placebo, but this innovative study was either overlooked or conveniently left out of the paper (Mayoral et al., 2013).

Identification of trigger points

We acknowledge that there has not been a study to demonstrate the minimum essential features of the TrP needed to identify it for diagnosis and treatment purposes. Several older interrelater studies were marred by methodological problems that skewed the results of subsequent systematic reviews (Wolfe et al., 1992; Njoo and Van Der Does, 1994; Lucas et al., 2009), but we disagree that TrPs cannot be identified reliably as recent inter- and intrarater studies have confirmed (Gerwin et al., 1997; Bron et al., 2007; Al-Shenqiti and Oldham, 2005; Barbero et al., 2012; Myburgh et al., 2011). Quintner et al. concluded that

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examiners could only agree if they were informed where a problem existed. In support of their conclusion, they cited several studies. One study showed that experienced clinicians were better in identifying TrPs than those with less experience (Myburgh et al., 2011). Another study showed "the assessments of an individual examiner were consistent from one test to another" (Al-Shenqiti and Oldham, 2005), which is what you would expect from an intrarater reliability study. In other studies, palpation was limited to certain muscles (Bron et al., 2007; Gerwin et al., 1997; Sciotti et al., 2001), which does not minimize the validity of these studies and does not provide any evidence that TrPs cannot be reliably identified. Once again, Quintner et al. opted to cite an older study with methodological problems to support their impression that TrPs cannot be reliably identified (Wolfe et al., 1992). A recent study confirmed that a systematic musculoskeletal evaluation can distinguish patients with active TrPs from subjects with no pain (Gerber et al., 2013).

Pathogenesis

Quintner et al. refer to "fibrositic nodules" which have nothing in common with TrPs and their relevancy escapes us. Apparently, they believe that TrPs are some kind of anatomical entity, although there has never been a credible anatomic pathology associated with myofascial TrPs. Fibrosis occurs when skeletal muscle is traumatized and damaged and the regenerative process triggers the development of abnormal increased collagen and accumulation of extracellular matrix between myofibers (Lieber and Ward, 2013). On the other hand, TrPs are phenomena representing a physiologic change in muscle function. They differ from normal muscle tissue and have different mechanical characteristics. They are palpable as hard nodules within a band of contractured fibers and feature spontaneous or induced pain and local or referred tenderness. Gerwin and Duranleau demonstrated already in 1997 that TrPs can be visualized using sonography (Gerwin and Duranleau, 1997). In addition, there is an increased retrograde diastolic flow in the immediate vicinity of TrPs indicating the presence of highly resistive vascular beds (Ballyns et al., 2012). Quintner et al. maintain, however, that there is no experimental evidence that blood vessels in the vicinity of TrPs are being compressed by the contractured muscle fibers of the taut band leading to ischemia and hypoxia.

The differentiation between normal muscle tissue, active and latent TrPs reflects the degree of contraction, as confirmed objectively by vibration elastography. Their mechanical attributes are, however, not directly correlated with pain pressure threshold scores (Ballyns et al., 2012), which Quintner et al. interpreted as another nail in the coffin of the TrP hypothesis. Furthermore, Quintner et al. accused the researchers of not providing sufficient data, although they clearly referenced one of their previous studies that included detailed descriptions of the methodology (Sikdar et al., 2009). The study by Ballyns et al. is part of an ongoing clinical research protocol and builds on their previous research. Quintner et al. considered the lack of a control group "yet another flaw," although the authors acknowledged this shortcoming and a year later published another study of 50 subjects with an adequate control group without pain (Turo et al., 2013). In the end, a total of 29 subjects were examined. The researchers used entropy analysis to characterize the echotexture of tissues, including TrPs. Turo and colleagues described some potential pitfalls of sonography in the identification of TrPs, but also demonstrated that echotexture analysis using local entropy can distinguish between subjects with painful trigger points and those without pain (Turo et al., 2013). Quintner et al. did not include the Turo paper in their analysis.

Quintner et al. question the nature and possible even the validity of latent TrPs, although Ballyns et al. clearly were able to distinguish and visualize contractured nodules in muscle that were only painful when stimulated (Ballyns et al., 2012). Researchers from Denmark have shown that latent TrPs have distinct physiologic effects, including hypersensitivity to hypertonic saline and glutamate not seen in normal muscle, affecting central sensitization and other phenomena (Ge et al., 2008, 2009b; Li et al., 2009; Wang et al., 2009; Xu et al., 2010; Zhang et al., 2009; Ge et al., 2014). Mense reviewed the differences between active and latent TrPs and concluded that both are sources of peripheral nociceptive input potentially initiating and maintaining central sensitization (Mense, 2010). Quintner et al. rejected that TrPs may be peripheral sources of nociceptive input leading to the initiation and maintenance of central sensitization and cited two studies demonstrating that referred pain elicited by pressure over active TrPs resembled the patients' pain complaint in patients with fibromyalgia.

Quintner et al. also failed to acknowledge that the presence of local contractions combined with a decreased local blood flow or increase in outflow resistance point to local ischemia and hypoxia, one of the key features of the integrated TrP hypothesis, as Ballyns and coworkers reviewed (Ballyns et al., 2012). A German-language study of the oxygen saturation near and in TrPs confirmed that the oxygen saturation is increased in the immediate vicinity of a TrP (Brückle et al., 1990), but sharply declined in the core of a TrP, which confirms the observations of Ballyns et al., 2012. Along the same lines, they discredited the studies by Chen et al., who visualized the contractured bands using magnetic resonance elastography for not making "any comment on the relationship of a taut band to a TrP," although that was not the purpose of the studies (Chen et al., 2008b, 2007). Rather than appreciating that researchers are exploring different aspects of the current hypothesis, Quintner et al. opted to select a few references that support their biases. Ultrasound elastography is a validated technology to directly measure the mechanical properties of tissues, including areas of increased stiffness of muscles (Brandenburg et al., 2014). Sonography is not only used for research and possible diagnostic purposes, but can also be applied to guide trigger point needling (Botwin et al., 2008; Bubnov 2010, Suh et al., 2014) and to objectively measure the outcome of TrP interventions such as dry needling (Maher et al., 2013).

Low pH and its consequences

Quintner et al. criticize the findings of Shah et al. (Shah et al., 2003, 2008, 2005), who have reported relatively

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higher concentrations of neurotransmitters and cytokines in the extracellular fluid in the immediate vicinity of TrPs as being non-specific. They comment that these findings could be caused by inflammation as a result of tissue damage or due to altered peripheral nerve function, which indeed is a possibility. Shah et al. have not claimed that their observations are unique to TrPs, but only that these have been found and are consistent with the clinical findings of tenderness and pain, and with the presence of local muscle contractions (Gerwin et al., 2004). Of interest is that Hsieh et al. reported similar results in a more recent animal study (Hsieh et al., 2011). They measured the concentrations of a variety of biochemicals, including β-endorphine, substance P, tumor necrosis factor-α, cyclo-oxygenase-2, hypoxia-inducible factor 1-alpha, inducible nitric oxide synthase, and vascular endothelial growth factor and noted that dry needling of TrPs can modulate these concentrations in a dosage dependent manner (Hsieh et al., 2011). Quintner et al. did not include this paper in their analysis. Quintner et al. do not believe that a valid animal model of myofascial pain exist. Yet, experiments on taut bands in rabbit muscles exhibited local twitch responses, which in humans have been confirmed to be characteristic of TrPs and taut bands.

Of great interest is the fact that Shah et al. found the pH of the extracellular fluid in the region of active TrPs to be very acidic with values well below 5 (Shah et al., 2008, 2005). Quintner et al. did mention the finding of a lowered pH, but did not consider this any further. Hagberg noted that a drop in pH from 7.0 to 6.6 was sufficient to activate nociceptive acid sensing ion channel - 3 (ASIC3) receptors in nearby neurons in a rabbit model (Hagberg, 1985). In addition to acid sensing ion channels, there are several other types of receptors that can get activated by a drop in the pH, such as transient receptor potential cation channel subfamily V member 1 and 4 (TRPV1 and TRPV4) and short transient receptor potential channel 4 and 5 (TRPC4 and TRPC5) (Eisenhut and Wallace, 2011; Lee et al., 2005; Schaible et al., 2011). Sluka and her colleagues have shown that ASIC receptors can be activated by protons and can cause pain without causing inflammatory or anatomic changes in muscle. In fact they established that an acidic pH has a profound effect on the initiation and perpetuation of muscle pain and activation of ASIC1 or ASIC3 muscle nociceptors can result in mechanical hyperalgesia and central sensitization (Sluka et al., 2001, 2002; Walder et al., 2010). Perhaps Quintner et al. would consider our reasoning an example of conjecture, but the facts are that a low pH is common at active TrPs, and can cause muscle pain and hyperalgesia. We continue to believe that this is a very interesting model for the pain of myofascial TrPs (Gerwin et al., 2004).

EMG studies

In 1959, Travell reported that TrPs may have a unique electromyographic signature (Travell, 1959) and in 1966, Arroyo found continuous motor activity only in the region of TrPs (Arroyo, 1966). It is was not until 1993, however, that Hubbard and coworkers confirmed spontaneous electrical activity at TrPs (Hubbard and Berkoff, 1993). Previous efforts to identify such activity had failed mostly because of methodological issues or poor definitions of myofascial pain. For example, the paper cited by Quintner et al. only required that subjects had a history of regional musculoskeletal pain with localized muscle tenderness and occasional referred pain (Durette et al., 1991).

The majority of the studies on the electrical activity of the TrP were carried out on animals and were based on palpation of locally contracted muscles (Chen et al., 1998a, 2001, 2008a, 1998b; Fu et al., 2012; Hong and Yu, 1998; Hou et al., 2002; Hsieh et al., 2011; Kuan et al., 2000, 1998; Simons et al., 1995). Macgregor and Graf Von Schweinitz studied the electromyographic activity and other characteristics of myofascial TrPs in equine cleidobrachialis muscles and confirmed that they have similar objective signs and electrophysiological properties to those documented in human and rabbit skeletal muscle tissue (Macgregor and Graf Von Schweinitz, 2006). Upon further examination of the spontaneous electrical activity, it became clear that this electrical activity is indeed endplate noise (Chou et al., 2009; Kuan et al., 2002, 2007; Qerama et al., 2004; Simons, 2001, 2004). Identification of endplate noise is not recommended in clinical practice, but some have suggested that it should be the gold standard in TrP research (Ge et al., 2011a).

We agree with Quintner et al. that it is possible to record insertional electrical activity from single muscle fibers, but that has no relevancy in the current context. Quintner et al. dismissed any animal research for a lack of relevant TrP models and suggest that a local twitch response, which is an entirely different feature of the taut band, is nothing but a myotatic stretch reflex. They did not address the common observation that strumming a TrP taut band produces a local contraction only of that band, and not of the entire muscle as would be expected of a stretch reflex, and why this is not replicated in normal, non-taut band muscle. Thus, their assertion that the twitch response is a stretch reflex is not consistent with common clinical observation, current research, and objective visualizations of the twitch response (Audette et al., 2004; Dexter and Simons, 1981; Fricton et al., 1985; Hong and Torigoe, 1994; Hong, 1994a, 1994b; Rha et al., 2011; Simons and Dexter, 1995; Wang and Audette, 2000). Quintner et al. are correct that there is no evidence for a reflex increase in fusimotor drive, although that has been suggested by some (Partanen et al., 2010). They erroneously characterize the taut band as a "focal muscle contraction modulated by muscle spindle afferents" for which there is no evidence either. Once again, Quintner et al dismiss the experimental evidence out of hand, but they have not published any studies in which they try to reproduce any of the work and come to different explanations for the data found. Indeed, when they dismiss the local twitch response of the taut band as a stretch reflex, their interpretation of neuromuscular physiology is wrong.

We agree with Quintner et al., that fibromyalgia tender points and myofascial TrPs are not necessarily the same, although the overlap between the two syndromes and painful spots in muscles has been confirmed (Staud et al., 2009; Staud, 2011; Ge et al., 2009a, 2010, 2011b; Affaitati et al., 2011). Some of the phenomena that seem to trouble Quintner et al. result from the fact that TrPs

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induce central sensitization and referred pain. Muscles do have nociceptors and activation of those nociceptors can initiate and perpetuate central sensitization (Graven-Nielsen and Mense, 2001; Mense, 1991, 2003, 2008; Simone et al., 1994; Rubin et al., 2009, 2010, 2012; Izumi et al., 2014; Mense, 1977) as acknowledged by Bove in his outstanding chapter "Nociceptors, Pain, and Spinal Manipulation" co-authored with Swenson (Swenson and Bove, 2011). Pain referral and triggering central sensitization are, however, not unique to muscles and may involve nerves (Oaklander et al., 2013; Markman, 2014; Quintner and Cohen, 1994), joints (Fukui et al., 1996; Cooper et al., 2007), discs (O'Neill et al., 2002; Oikawa et al., 2012), and viscera (Procacci and Maresca, 1989; Giamberardino and Vecchiet, 1995; Giamberardino et al., 1999; Bajaj et al., 2003). Moseley confirmed that especially myofascial TrPs and joints are widely held to be common contributors to somatic referred pain (Moseley, 2012). According to Moseley, with somatic referred pain the "disrupted transmission" happens within the central nervous system and he considers somatic referred pain from TrPs and joints as the brain's efforts to localize the pain in response to ambiguous input (Moseley, 2012).

Delayed onset muscle soreness

Quintner et al. correctly state that delayed onset of muscle soreness (DOMS) has been related to TrPs: "although DOMS has been related to TrPs in only one study [66], this model was proposed for MPS [67]."

But we don't understand why Quintner et al. insist that there was only one study as both cited studies (references 66 and 67 in the Quintner et al. article) explored the possible role of eccentric loading. According to Quintner et al., the study by Itoh and colleagues (Itoh et al., 2004) does not clarify how eccentrics or DOMS would be related to TrPs as they attempted to locate a taut band in the band-like extensor digitorum muscle of the middle finger. The second study focused on the role of nerve growth factor in persistent muscle pain associated with a taut band (Hayashi et al., 2011). The researchers concluded, that "repeated eccentric contractions produced a muscle hyperalgesia associated with taut bands that was similar to the feature of MTrP" (Hayashi et al., 2011).

In our 2004 article on the expansion of the integrated TrP hypothesis, we introduced the notion that perhaps eccentric loading could be related to the formation of TrPs (Gerwin et al., 2004). We provided basic background information about disruption of the muscle sarcomere as a result of eccentric loading and suggested that the combination of sarcomere disruption, hypoperfusion, and the resultant ischemia and hypoxia resembles the process that is thought to lead to the formation of TrPs. We did not present this as dogma supported by scientific studies, but as a possible expansion of the integrated TrP hypothesis. We concluded that these are areas need further investigation (Gerwin et al., 2004). In our 2012 paper on the etiology of TrPs, we emphasized again that there is no solid evidence that eccentric loading would lead to the development of TrPs as there are no studies that have confirmed or refuted our previous suggestion (Bron and Dommerholt, 2012). Even the paper by Itoh et al., which Quintner et al. cited, is hampered by methodological issues, as the study combined isometric loading and eccentric loading, which precludes any definitive conclusions (Bron and Dommerholt, 2012; Itoh et al., 2004). The lack of evidence does not diminish the value of the expanded hypothesis, although we are disappointed that the potential role of eccentric loading on the development of TrPs has not yet been investigated. Therefore, we agree with Quintner et al. that the relevance of DOMS to TrPs remains unclear, but that is not a reason to dismiss the concept as worthy of further study.

Integrated Trigger Point Hypothesis

Many of the issues Quintner et al. have with the Integrated TrP Hypothesis have already been addressed, however, they questioned a few other aspects. For example, Quintner et al. cited us for postulating that low-level isometric contractions could result in muscle dysfunction and the formation of TrPs, but they did not reference the original source documents. In 2006 and 2011, two complementary studies were published that explored whether the Cinderella Hypothesis could apply to the formation of TrPs (Hoyle et al., 2011; Treaster et al., 2006). Both studies provided evidence that low-level exertions can lead to the formation of TrPs.

Treatment

We agree that there are few outcome studies of good quality and although some studies showed reduction in pain scores and pressure pain thresholds, the literature has neither convincingly supported or refuted the effectiveness of some invasive and non-invasive modalities beyond placebo (Rickards, 2006). Quintner et al. report that in 2001, Cummings and White were unable to find evidence that needling therapies have any specific effect (Cummings and White, 2001). The effect of TrP injections was independent of the injected substance, which in most studies was an anesthetic such as lidocaine, bupivacaine, or procaine. A few papers utilized other injectates, including botulinum toxin (1 study), Diclofenac (1 study), and Prednisolone (1 study). The authors reported that three studies found no difference between injections and dry needling and they did not find any evidence for sub- or intracutaneous injection of water or saline (Cummings and White, 2001). Cummings and White concluded that "direct needling of myofascial trigger points appears to be an effective treatment, but the hypothesis that needling therapies have efficacy beyond placebo is neither supported nor refuted by the evidence from clinical trials. Any effect of these therapies is likely because of the needle or placebo rather than the injection of either saline or active drug" (Cummings and White, 2001).

We agree with Quintner et al. that studies of the efficacy of TrP interventions have shown such marked statistical heterogeneity that it can be difficult to evaluate outcomes (Tough et al., 2009). A 2010 feasibility study by Tough and coworkers cautiously mentioned that significantly more patients who received TrP acupuncture had discontinued taking analgesic medications compared to

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those in a sham acupuncture group, but treatment effects were not the main objective of this study and the authors suggested exploring this further in a future study (Tough et al., 2010).

There are several other studies and review articles that were left out from Quintner et al.'s analysis. For example, they did not include a German study that demonstrated that injections of a serotonin antagonist were superior to injections of an analgesic in reducing pain from TrPs (Müller and Stratz, 2004). We have already mentioned the double-blind randomized controlled study by Mayoral and colleagues (Mayoral et al., 2013), but there are many others in addition to the few we cite here (Cotchett et al., 2014; France et al., 2014; Nicol et al., 2014; Moghtaderi et al., 2014; Maher et al., 2013; Cagnie et al., 2012; Rocha and Sanchez, 2012; Suh et al., 2014).

Since common treatment options, such as TrP compression, dry needling and TrP injections tend to be painful, Quitner et al. suggest that TrP therapy may work because of counter-irritation. We agree that counter-irritation is likely one of the potential mechanisms as to why TrP therapy is usually very successful. Elsewhere in the paper, they describe that "digital pressure or other stimuli that evoke pain will decrease the tone of the muscle stimulated," which is probably why sustained pressure over a TrP usually eliminates the local contracture, allowing oxygen supplies and pH levels to be restored. Of interest is that brief noxious stimuli at TrPs do not result in more sensitization as is frequently suggested in the literature (Jull, 2012). There is emerging evidence that a painful stimulus can inhibit a primary source of nociception in the context of a therapeutic encounter, leading to a larger inhibitory conditioned pain modulation (Bjorkedal and Flaten, 2012). In other words, a painful stimulus, such as TrP dry needling can activate an endogenous pain inhibitory mechanism that inhibits early nociceptive processing (Bjorkedal and Flaten, 2012). Patients are usually able to dissociate the pain intensity associated with the treatment procedure from the magnitude of responses in the "pain matrix" (Legrain et al., 2011). There is no evidence that in the presence of sensitization, physical therapy interventions need to be pain-free (Jull, 2012) as long as the noxious stimulus is short in duration and delivered in the context of a therapeutic encounter.

To be clear, we do not agree with the conclusion by Fogelman and Kent, who recently suggested that the lack of precision and a high risk of bias in studies of dry needling should be laid to rest with more recent research and meta-analyses of the data, but we do share their observation that rigorous large scale clinical observational trials are needed (Fogelman and Kent, 2014), which is a more sensible approach than discarding the entire TrP concept.

Quintner et al.'s hypotheses

Quintner et al. offer an alternative proposal of an inflammatory neuritis for the pain that has been described as myofascial. Over 20 years ago, they suggested that myofascial pain would have a peripheral nerve origin because of their close proximity to TrPs (Quintner and Cohen, 1994), but to date their hypothesis has never been tested. They claim that "subsequent research has emerged in support of their hypothesis," but the three included references do not provide any support for their assumption. While we appreciate the paper by Bove et al. about increased ectopic mechanical sensitivity in the axons of nocicepters, this paper does not provide any explanation of myofascial pain or even of the formation of TrPs (Bove et al., 2003). The other two references by Dilley et al. are valuable contributions to the scientific literature about mechanosensitivity of peripheral nerves, but the connection to myofascial pain remains unclear (Dilley and Bove, 2008; Dilley et al., 2005). Even a more recent publication by Bove and Dilley about ongoing or spontaneous nociceptive activity does not add anything to a better understanding of the underlying mechanisms of myofascial pain and TrPs (Bove and Dilley, 2010).

Since TrPs occur in virtually all voluntary muscles and are located in the innervation zone of muscles (Barbero et al., 2013), it is difficult to see how the anatomic relationship of nerves to muscles provides much insight into the pathogenesis of pain. We are not aware of any signs of postulated nerve dysfunction other than an abnormal electromyogram that shows endplate noise that has been found to be more commonly associated with the TrP taut band than evenly distributed throughout muscles (Simons, 2001). One would have to postulate that this EMG activity could represent an inflamed or irritated nerve, however, there are no studies that convincingly show other evidence of nerve injury, and none of the usual electrodiagnostic signs of nerve injury are seen in the vast majority of patients with myofascial TrP pain.

In the current paper, they specifically claim that "focal inflammation of peripheral nerves leads to ectopic axonal mechanical sensitivity and spontaneous discharge of some but not all of the nociceptors within the inflamed nerve. These changes can be expected to lead to focal areas of neurogenic inflammation and *possibly to sensitization in the muscle innervated*" (italics added). We do not disagree with that statement, yet we find no evidence put forth by the authors that this is actually the case in muscle with focal tenderness and referred pain that is described as myofascial pain.

There are several other concerns about their hypothesis. First, the postulate must be that the inflammation is sterile, as there is no literature about cellular infiltrates around peripheral nerves in muscles in myofascial pain or fibromyalgia. Second, the involvement would have to be confined to motor nerves or motor nerve endings, since these patients generally do not have sensory loss associated with their pain, and paresthesias are uncommon. Third, many of the patients with myofascial pain have persistent pain for long periods of time. One would expect that if there were a neural basis for this that affected motor nerves, there would be denervation changes in the muscle, manifest by denervation atrophy on biopsy or by electrodiagnostic changes. On the other hand, if Quintner et al. postulate that only the sensory nerves are involved, they have to explain why there are no sensory changes other than pain, such as hyperesthesia or hypesthesia, associated with the muscle pain. If they postulate that only nerves that go to muscle are involved, then they must explain how that can happen and how that can involve multiple

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muscles. They would also have to explain how that could result in the contracted bands of muscle or taut band that always accompany pain other than saying that they do not exist, which seems to be their argument.

Quintner et al. offer a brief alternate hypothesis suggesting that a TrP is a site of secondary allodynia due to altered central nociceptive mechanisms. This concept has been explored by Srbely, leading to the Neurogenic Hypothesis (Srbely, 2010), but to date there is no convincing evidence that TrP are secondary to central mechanisms, although there is some preliminary evidence that central sensitization can promote TrP activity (Fernandez-De-Las-Penas and Dommerholt, 2014). Increasing referred pain areas in patients with chronic pain are a consequence of higher central neural plasticity (Arendt-Nielsen et al., 2000). In addition, maintenance of referred pain is dependent on ongoing nociceptive input from the site of primary muscle pain (Arendt-Nielsen and Svensson, 2001; Rubin et al., 2009).

Conclusion

Based on our understanding of the literature, Quintner et al. have not presented any convincing evidence to believe that the Integrated TrP Hypothesis should be laid to rest. They view the literature from a bias, concluding that when there are insufficient studies to reach a conclusion, there would be no evidence or reason to proceed. Instead, insufficient evidence is to be taken that further studies are needed, not that a condition does not exist or a treatment is not effective. Rather than ignoring the worldwide developments in this field, we prefer the approach by Jafri, who critically reviewed the current thinking and contributed to a more in-depth understanding of possible underlying mechanisms (Jafri, 2014).

We welcome Quintner et al.'s attempt to provide an alternative explanation for the pain that we see in our patients. We look forward to their studies on diagnosis and treatment of the various manifestations of myofascial TrP pain syndromes that have already been delineated. Perhaps their further work in the field will provide us with better tools than we have now. Although Quintner and Cohen suggested alternate explanatory models over 20 years ago, to this date, they have not performed a single trial comparing their way of treating these conditions with the techniques that have been successfully used in treating myofascial TrP pain. In the mean time, we will continue with the scientific exploration of the underlying mechanisms of myofascial pain.

In summary, Quintner et al. have not succeeded in providing sufficient evidence that the current TrP hypotheses should be rejected.

Statement of interests

Dr. Dommerholt is a physical therapist in private practice and works primarily with patients with chronic pain syndromes, including myofascial pain. He is president of Myopain Seminars, a post-graduate continuing company that offers courses in multiple subjects, including myofascial trigger point therapy and dry needling. Dr. Dommerholt teaches courses worldwide. He has written close to 60 book chapters and nearly 100 articles on pain syndromes, including myofascial pain and dry needling. He is a guest lecturer at several universities, including the University of Maryland, Department of Physical Therapy in Baltimore, Maryland; Shenandoah University in Winchester, Virginia, and the Universidad CEU Cardenal Herrera in Valencia, Spain.

Dr. Gerwin is in private practice and is on the faculty of the Johns Hopkins University School of Medicine Department of Neurology. He treats myofascial pain syndromes. He also teaches about myofascial pain in workshop programs throughout the United States and Worldwide. He has written articles and book chapters about myofascial pain and has co-edited a number of books about the subject.

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Please cite this article in press as: Dommerholt, J., Gerwin, R.D., A critical evaluation of Quintner et al: Missing the point, Journal of Bodywork & Movement Therapies (2015), http://dx.doi.org/10.1016/j.jbmt.2015.01.009

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