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INVITED CRITICAL REVIEW

A critical evaluation of Quintner et al: Missing the point

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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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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