Myofascial trigger points: does recent research give new insights into the pathophysiology?

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The paper by Ge et al (see page 150) may be an important milestone in the investigation of the pathophysiological substrate of the myofascial trigger point (MTrP). The paper appears to show that there is a significantly reduced threshold and increased amplitude of an electrophysiological reflex (similar to the Hoffman (H-) reflex) when it is stimulated electrically from within an MTrP compared with normal muscle nearby.

It appears to be the first paper in which an electrophysiological reflex similar to the H-reflex has been recorded following an intramuscular electrical stimulus. The important finding of the paper is that the amplitude of this reflex as a proportion of the maximum direct muscle stimulus effect (measured peak to peak from the M-wave—the electromyography (EMG) recording of the direct muscle or motor nerve stimulus) appears to be significantly greater when elicited from within an MTrP than from within normal muscle tissue (non-MTrP).

The H-reflex was first described in 1910 by the German physiologist Paul Hoffmann.2 It is an electrophysiological equivalent of the stretch reflex (usually elicited as a tendon jerk in the physical examination), but it bypasses the muscle spindle.3 It is used in research as a non-invasive neurophysiological probe to study the neural control of movement.34 The H-reflex is stimulated by a transcutaneous electrical impulse (square wave of short duration, ie, 0.5–1 ms) over a major nerve (eg, the tibial nerve behind the knee) and recorded with EMG electrodes over a muscle in the motor distribution of the same nerve (eg, the gastrocnemius or soleus muscle). The electrical stimulus over the nerve is increased until the H-reflex appears (typically 4–10 mA), which corresponds to stimulation of the Ia muscle spindle afferents (MSAs)—Ia MSAs are the largest diameter and fastest conducting nerves. A monosynaptic (or oligosynaptic)5 reflex results in stimulation of the associated Aα motor nerves. At higher stimulus intensities the M-wave appears—this is caused by direct stimulation of the Aα motor nerves to the muscle. The H-reflex appears before the M-wave because the Ia MSAs have a lower electrical threshold than the Aα motor nerves.

In the paper by Ge et al the M-wave was stimulated from within muscle, either by intramuscular stimulation of Aα motor nerves or by direct depolarisation of muscle cell walls. Despite this, the latency of the M-wave recorded from surface EMG was similar whether elicited by stimulation of the tibial nerve or elicited by intramuscular stimulation—presumably because the speed of transmission of a muscle action potential is some 13 times slower than the nerve action potential in the Aα motor nerves.5

We need to be cautious in our interpretation of the result in Ge et al, since the H-reflex is not generally stimulated from
within muscle, and this may well increase the potential for confounding factors. There are already many factors that are known to influence the size of the H-reflex. In order to compare the H-reflex in different circumstances of stimulation, it is generally expressed as a proportion of the maximal M-wave stimulated under the same experimental conditions (M_max). This is where the use of intramuscular stimulation may result in an important difference from direct nerve stimulation, since the efficiency of stimulating Ia MSAs is likely to vary with distance from the needle, but the M-wave may be more consistent as muscle cells can be stimulated directly from anywhere in the muscle rather than via the Ax motor nerves.

So what were the differences in the sites that were compared? The MTrP sites were found by palpation of a taut band and spontaneous activity on needle EMG, and the non-MTrP sites were in nearby muscle without a taut band or spontaneous EMG activity. Spontaneous activity on needle EMG in resting muscle is attributed to end plate noise: miniature end plate potentials and end plate spikes. Miniature end plate potentials appear to be related to spontaneous release of acetylcholine vesicles from motor nerve terminals at motor end plates, and the subsequent excitatory post-synaptic potentials. The origin of end plate spikes is less clear. One group suggests that they originate from within muscle spindles as muscle action potentials in intrafusal fibres. However, it seems conceivable that they could be related to the combined effect of the simultaneous release of two or three vesicles of ACh on the excitatory post-synaptic potentials.

So the sites found by Ge et al may be endplate zones or muscle spindles (this reviewer suspects they are the former). This may explain the results purely through anatomical considerations, since Ia MSAs are likely to be much closer to both these structures than to the control points. Nerves tend to enter muscle with the principal blood vessels on the deep surface near to the least mobile attachment. They proceed within the connective tissue septa and appear (assumption of the reviewer) to innervate the endplate zones around the mid-fibre region before tracking further afield to the muscle spindles and Golgi tendon organs. Therefore an electrical stimulus at the endplate zone is likely to excite Ia MSAs more readily than the same stimulus outside the endplate zone. The electrical field strength produced by a potential around a point source (the needle tip of an insulated needle) will diminish in proportion to the square of the distance from the point source (the inverse-square law). This means that nerves near to the needle tip will be stimulated at a much lower potential than those further away.

This may be a major limitation in interpreting the results of this paper, however, we can still say that MTrPs (defined as taut bands with spontaneous needle EMG activity) are either closer to Ia MSAs or are associated with more sensitive or responsive Ia MSAs or muscle spindles than non-MTrP areas in muscle.

The hunt for the unique substrate of the myofascial trigger point goes on!

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REFERENCES


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