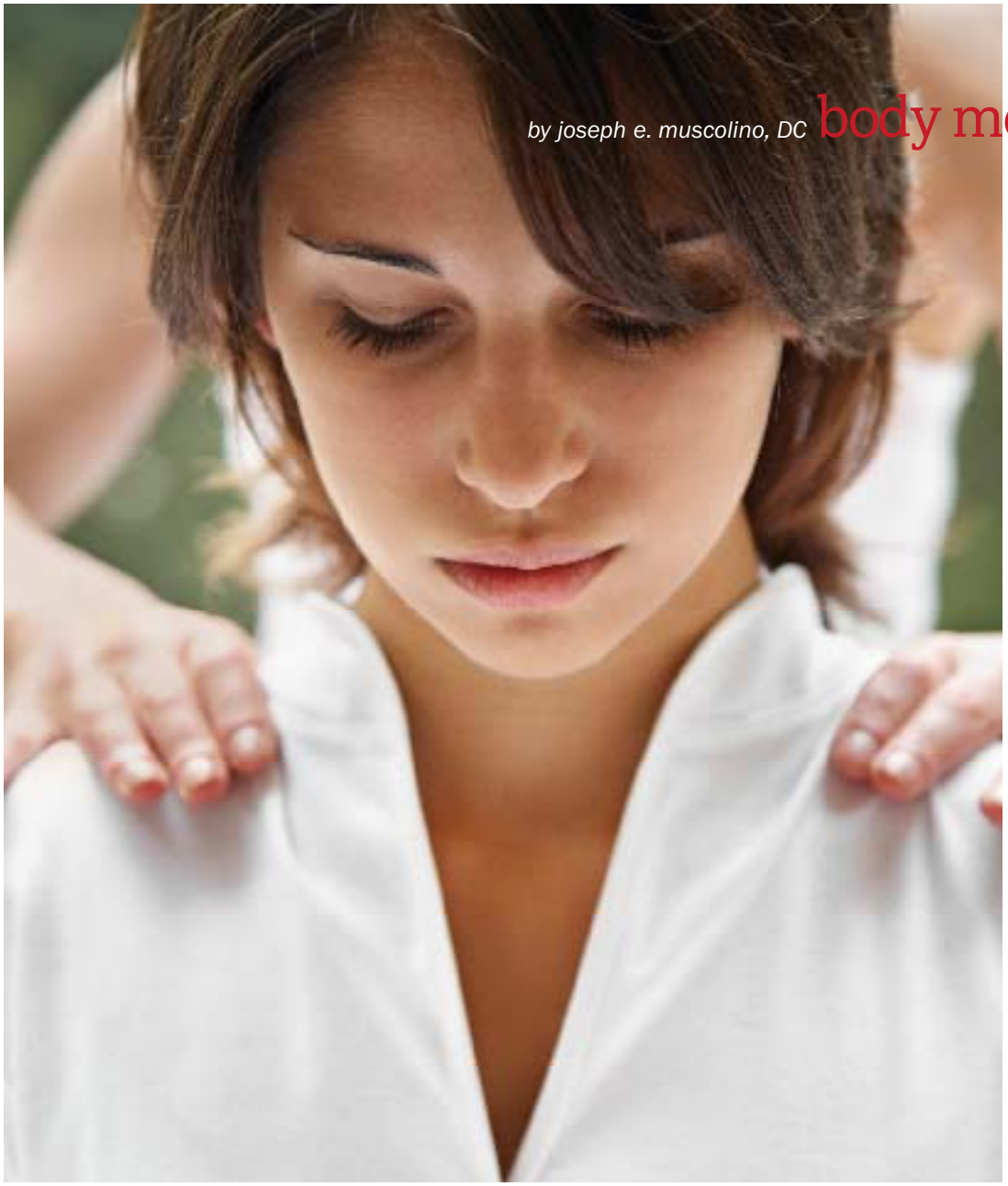

by joseph e. muscolino, DC

understanding & working with myofascial trigger points

As we venture into practice and actually begin working with our clients, it's all too tempting to disregard or forget the science that we were taught back in massage school. However, the ideal techniques are those that employ the science that we have learned. Understanding the pathomechanics of a condition can point us in the direction of the technique that will be most effective because it addresses the foundation of the problem.

Therapy for trigger points (TrPs) is a prime example of this. For years, the method that has been used for TrPs has been ischemic compression or some other form of sustained compression. Yet, as we come to better understand what creates and perpetuates a TrP, it seems that the best approach might actually be short stroking massage. But how to decide? Let's look at what a TrP is, what creates and perpetuates it, and then see if we can reason out what stroke would likely best resolve it.

A TrP is usually defined as a focal area of tenderness that can refer pain to a distant site. The most common type of TrP is a myofascial TrP that occurs within muscle and its fascial tissue. The rest of this article will address myofascial TrPs.

Our knowledge and understanding of TrPs is largely due to the pioneering efforts of two individuals—Raymond Nimmo, DC, and Janet Travell, MD. A half century ago, Nimmo and Travell, working independently of each other, championed the idea that pain could result from focal areas of hypertonicity within a muscle—what lay people refer to as muscle knots. Nimmo named these focal areas *noxious generative points*, only later refer-

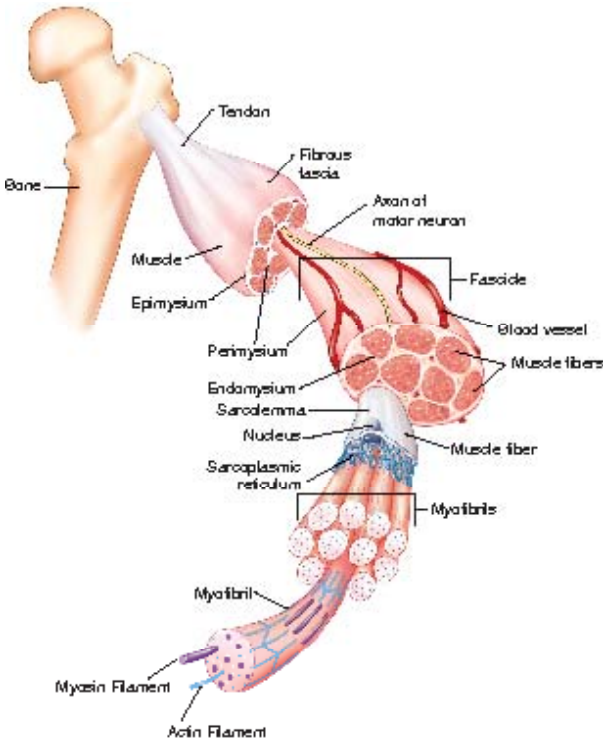

Figure 1. A muscle is composed of many muscle fibers. Each fiber is filled with myofibrils, which are composed of actin and myosin filaments.

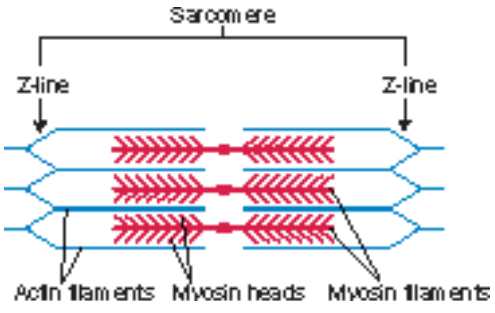

Figure 2. The actin and myosin filaments of a myofibril are arranged into units called sarcomeres.

ring to them as *trigger points* as that term came to be better known. Nimmo began publishing articles as far back as 1957; however his work remained largely confined to the world of chiropractic. It was in 1983 when Travell and David Simons published the first volume of their two landmark texts on TrPs that the mechanism and treatment of TrPs exploded in the larger world of mainstream and complementary alternative medicine, including massage.

To understand how a TrP is formed and what perpetuates it, a brief review of muscle anatomy and physiology is necessary. A muscle is an organ composed of thousands of muscle fibers. Each muscle fiber is filled with myofibrils, which are composed of actin and myosin filaments (Figure 1). These actin and myosin filaments are arranged parallel to each other, forming units called sarcomeres. A myofibril is essentially formed by successive sarcomeres laid end to end (Figure 2). Each of these sarcomeres can create a pulling force via the sliding filament mechanism, in which, as the name implies, these actin and myosin filaments slide along each other. When the actin filaments of the sarcomere are pulled toward the center of the sarcomere, they create a pulling force that is transmitted to the attachments of the muscle (Figure 3). Therefore, the sliding filament mechanism is the basis for how a muscle functions. Effectively, a muscle's contraction force is determined by adding up all the individual pulling forces of the millions and millions of sarcomeres of that muscle.

To understand how a TrP develops, we must take a closer look at how the sliding filament mechanism functions. When a muscle is at rest, actin and myosin filaments lay next to each other without attaching to each other. However, when the signal for contraction comes to the muscle from a motor neuron of a nerve, calcium ions are released from the sarcoplasmic reticulum into the sarcoplasm of the muscle fiber (Figure 1). It's the presence of calcium that triggers the sliding filament mechanism to begin and causes the muscle contraction. (For more information about this, see the box on the opposite page.) Calcium ions bond to the actin filaments, exposing their binding sites. Once exposed, myosin heads attach to these binding sites, creating cross-bridges. These cross-bridges then attempt to bend, creating a pulling force on the actin filaments toward the center of the sarcomere; this pulling force is the muscle contraction. If the force of the contraction is great enough to overcome whatever resistance force there is, the muscle will succeed in shortening (a concentric contraction) and one or both of its attachments will move toward each other.

This occurs by the cross-bridges bending and pulling the actin filaments toward the center, then the cross-bridges break and reconnect to the next binding sites and bend again, pulling the actin filaments farther toward the center (Figure 4).

Once a TrP forms, because of its pulling force toward its center, it pulls on its myofibril, causing a taut band to form. Therefore, TrPs usually reside at the center of a taut band. Further, this taut band pulls at the attachments of the muscle, creating an irritation that often creates TrPs at the attachments of the muscle.

This process continues as long as the calcium is present. However, the process also requires the presence of energy, supplied by ATP molecules. Ironically, the energy is not needed to form the cross-bridges—it's necessary to break them. It's also necessary to remove the calcium from the sarcoplasm and place it back into the sarcoplasmic reticulum of the muscle fiber. Therefore, if the muscle fiber is deprived of ATP molecules, the cross-bridges cannot be broken and the contraction of the affected sarcomeres will continue. It's this deprivation of ATP molecules that is the genesis of a TrP; it's called the *energy crisis hypothesis*. Research has continually supported this mechanism as the explanation for the formation and perpetuation of a TrP.*

Deprivation of ATP molecules occurs when a local ischemia, which results when the muscle's contraction closes off its own arterial blood supply. Therefore, a vicious cycle is created in which the muscle's contraction (if strong enough and held for sufficient time) causes an ischemia, depriving the area of blood supply and, therefore, the glucose necessary to create the ATP molecules needed to break the contraction (by both breaking the cross-bridges and removing the calcium from the sarcoplasm).

It stands to reason that whatever technique is used to treat a TrP must improve circulation to the area. Pressure from whatever stroke is performed can mechanically deform the muscle tissue and physically break the cross-bridges, but if the arterial circulation is not restored, then the continued presence of calcium will only cause the cross-bridges to immediately reform, perpetuating the TrP.

The classic technique for working TrPs that has been used for decades is *ischemic compression*. This tech-

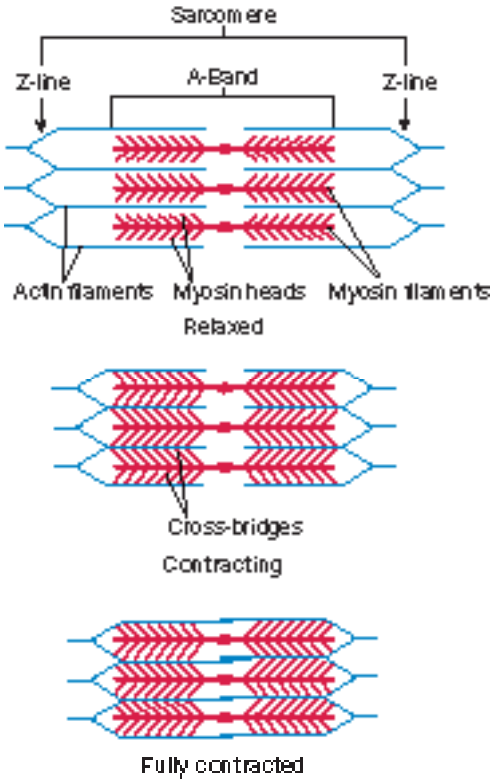

Figure 3a shows a sarcomere at rest. **Figure 3b** shows it partially contracted. **Figure 3c** shows the sarcomere fully contracted. The contraction of a muscle is defined by the presence of myosin-actin cross-bridges.

For more information about the sliding filament mechanism, check out "Kinesiology: the skeletal system and muscle function," (Mosby Elsevier, 2006).

* A corollary hypothesis for the formation of a TrP is the *dysfunctional endplate hypothesis*, which states that the motor endplate where the nerve signal comes in dysfunctions, causing an excessive release of neurotransmitters and therefore excess release of calcium ions in the sarcoplasm. Because these endplates are usually located at the center of a muscle's fibers, the first TrPs to form in a muscle are usually termed central TrPs.

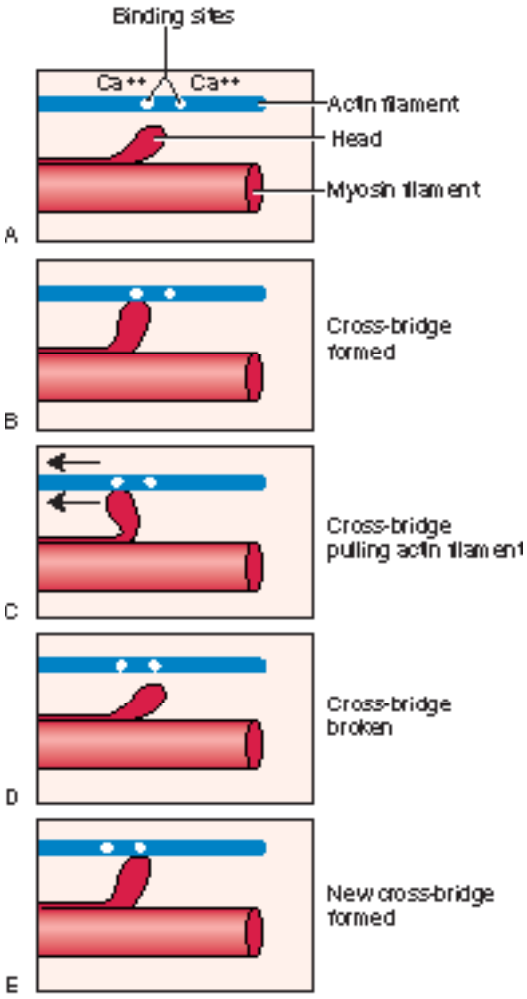

Figure 4. The sliding filament mechanism is initiated and continues to occur as long as calcium ions are present in the sarcoplasm of the muscle fiber.

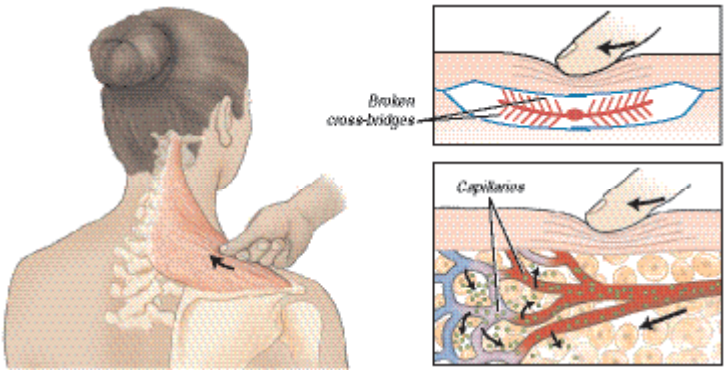

Figure 5. Deep stroking massage functions to increase arterial blood supply to the TrP. If done along the direction of the taut band of the TrP, it also helps to stretch and physically break the cross-bridges of the TrP.

nique required deep pressure to be applied directly to the TrP and held for upwards of 5 to 10 seconds. However, the second edition of volume one of *Travell and Simons' Myofascial Pain and Dysfunction* states: "Clinical evidence and the nature of TrPs indicate that, when applying digital pressure to a TrP to deactivate it, there is no need to exert sufficient pressure to produce ischemia." Indeed, the TrP is already ischemic, so creating further ischemia would make no sense. The authors recommended that if sustained pressure is applied, that its depth of pressure is not great. They renamed this technique of sustained compression *TrP pressure release*.

However, of even greater interest is their statement on the very next page where they describe deep-stroking massage as being "…probably the most effective way to inactivate central TrPs when using a direct manual approach." Looking at the mechanism of a TrP, if the underlying cause is a local ischemia, then it stands to reason that the most effective technique would be one that increases local circulation. A series of 30 to 60 short, deep strokes across the TrP would be more likely to accomplish this than would a few applications of sustained compression. While any stroke direction would increase circulation, if the strokes are applied along the direction of the taut band, they will cause a stretch as they are applied that will more likely help to mechanically break the cross-bridges (Figure 5).

TrPs are an excellent example of how the science that we learned can help us to better understand the foundation of a condition. This knowledge can then better inform our treatment approach. While any manual approach should help to deactivate TrPs, deep-stroking massage may be the most effective. If you have not yet tried deep-stroking massage as a technique to treat TrPs, perhaps you might like to begin.

RESOURCES:

Unless otherwise credited, images are from Muscolino JE: Kinesiology: the skeletal system and muscle function, enhanced edition. St. Louis, 2007, Mosby.

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