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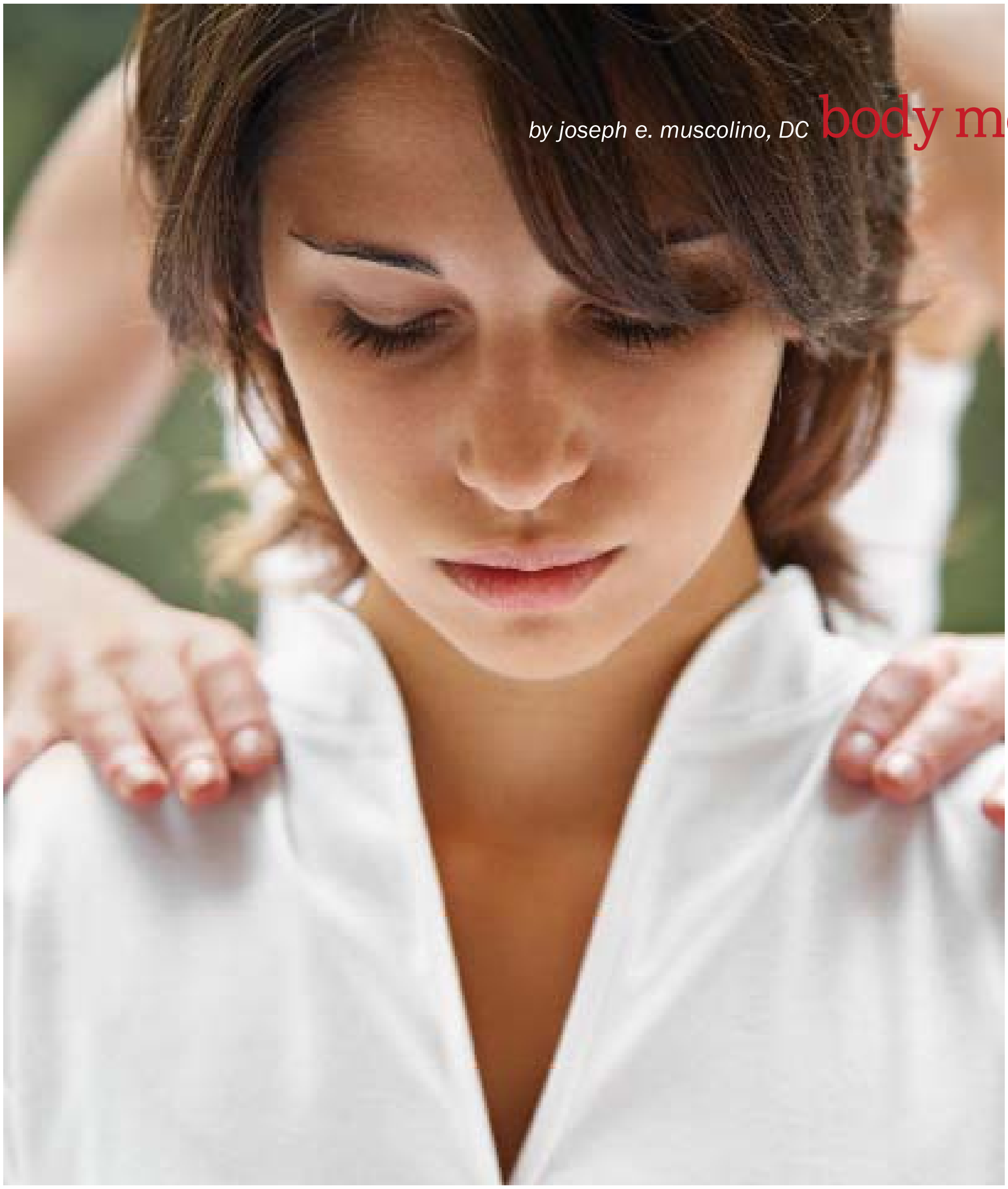

understanding & working with myofascial trigger points

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-

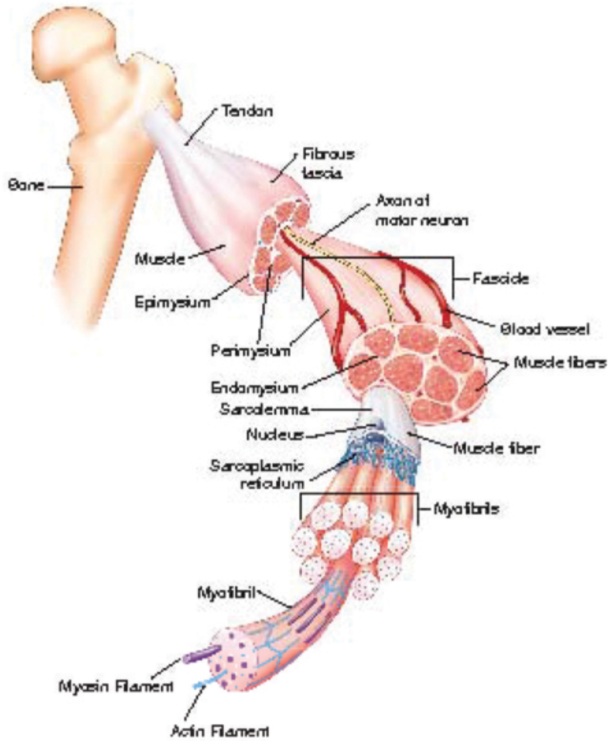

Figure 1. A muscle is composed of many muscle fibers. Each fiber is filled with myofibrils, which are composed of actin and myosin filaments.

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