J Shoulder Elbow Surg (2015) 24, 1322-1335

ELSEVIER

Journal of Shoulder and Elbow Surgery

www.elsevier.com/locate/ymse

REVIEW ARTICLE

The role of the peripheral and central nervous systems in rotator cuff disease

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Rotator cuff (RC) disease is an extremely common condition associated with shoulder pain, reduced functional capacities, and impaired quality of life. It primarily involves alterations in tendon health and mechanical properties that can ultimately lead to tendon failure. RC tendon tears induce progressive muscle changes that have a negative impact on surgical reparability of the RC tendons and clinical outcomes. At the same time, a significant base of clinical data suggests a relatively weak relationship between RC integrity and clinical presentation, emphasizing the multifactorial aspects of RC disease. This review aims to summarize the potential contribution of peripheral, spinal, and supraspinal neural factors that may (1) exacerbate structural and functional muscle changes induced by tendon tear, (2) compromise the reversal of these changes during surgery and rehabilitation, (3) contribute to pain generation and persistence of pain, (4) impair shoulder function through reduced proprioception, kinematics, and muscle recruitment, and (5) help explain interindividual differences and response to treatment. Given the current clinical and scientific interest in peripheral nerve injury in the context of RC disease and surgery, we carefully reviewed this body of literature with a particular emphasis on suprascapular neuropathy that has generated a large number of studies in the past decade. Within this process, we highlight the gaps in current knowledge and suggest research avenues for scientists and clinicians.

Level of evidence: Narrative Review.

Keywords: Shoulder; rotator cuff tear; pain; muscle; nerve; spinal cord; brain

The human shoulder complex exhibits a unique anatomic design to allow a wide range of motion at various speed and force levels. The shoulder joint complex has an unstable bone configuration secured by connective tissues and dynamic stabilizers (rotator cuff [RC] muscles) controlled by a sophisticated neuromuscular system.[157,161] As a consequence, shoulder structures, particularly RC tendons, are prone to various injuries and degenerative disorders.[19,121] RC tendon tears are common in the general population[104,123] and can lead to shoulder pain, impaired functional capacities, and reduced quality of life.[88,164]

Funding: The source of funding for this study was NIH R01 HD073180.

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1058-2746/$ - see front matter © 2015 Journal of Shoulder and Elbow Surgery Board of Trustees.
http://dx.doi.org/10.1016/j.jse.2015.04.004

RC tendon tears are not necessarily associated with pain or patient-reported loss of shoulder function[91,164,165]; however, asymptomatic patients may develop symptoms in a relatively short time.[107] Symptomatic patients usually undergo surgery when nonoperative and pharmacologic options have been exhausted.[112,130] Surgical management decisions are mainly driven by patients' pain, disability, and functional requirements rather than by the severity of local tissue damage.[15] In the short term, nonoperative treatment may be effective in a fraction of patients,[35,50,76] but tissue damage and symptoms may progress over time,[91,107,164] further limiting surgery and rehabilitation.[79,92,96,98] RC tendon repair is not universally successful; ~25% of repairs fail to re-establish the integrity of the RC[98] (up to 70% in massively retracted tears[36]), and patient-reported improvements are limited.[79,98] Preoperative factors, such as age, chronicity, and severity of muscle-tendon unit impairments, have been repeatedly associated with higher retear rates and poorer clinical outcomes.[79,98] Paradoxically, 2 recent meta-analyses[98,130] suggested that patients with intact repairs might not have significant differences in symptom improvement compared with patients with recurrent tears. Another major concern is that muscle impairments do not seem to be reversed, even when repair is intact and function is improved at follow-up.[26]

During the past decades, RC disease has been extensively investigated within the framework of tendon pathophysiology, tendon to bone healing, and muscle changes after tendon tear.[30,72] A smaller set of studies have investigated how peripheral, spinal, and central neural factors are likely to contribute to muscle-tendon unit changes, impaired shoulder function, and responses to treatment. Expanding our knowledge, or at least considering the potential involvement of both peripheral and central nervous systems, is critical to improve our understanding of RC disease and our ability to appropriately intervene along the continuum of RC injury processes. Therefore, this review aims to scrutinize and highlight the gaps in current knowledge about the nervous system that may be altered in patients with RC disease from the peripheral receptors to the brain and from the brain to the neuromuscular junction. We summarized how these factors may (1) exacerbate structural and functional muscle changes induced by tendon tear, (2) compromise the reversal of these changes during surgery and rehabilitation, (3) contribute to pain generation and persistence, (4) impair shoulder function by impairing shoulder proprioception, kinematics, and muscle recruitment, and (5) contribute to explain interindividual differences in symptoms and response to treatment. Given the current and lively interest in peripheral nerve injuries in the context of RC disease and surgery, we carefully reviewed this body of literature with a particular emphasis on suprascapular nerve (SSN) injury that has generated a large number of studies in the past decade. Within this process, we highlighted the gaps in current knowledge and suggested research avenues for scientists and clinicians.

Proprioceptors and related spinal reflexes

Shoulder movements and positional changes induce a deformation of tissues surrounding joints, including skin, muscles, tendons, fascia, joint capsules, and ligaments.[24,27,47,122,144,156] All these tissues are innervated by mechanically sensitive receptors termed proprioceptors that relay information to the central nervous system about movement, position, and forces exerted on shoulder structures (e.g., muscle spindles, Golgi tendon organs, Ruffini endings, Pacinian and Meissner corpuscles). The distribution and the function of proprioceptors in shoulder joints and soft tissue have been investigated in both animal and human studies.[40,51,139,141,144,147,156] Glenohumeral joint and ligament receptors probably play a minor role in shoulder proprioception,[122] as illustrated by the small proprioceptive deficit observed after shoulder arthroplasty.[21] However, they may act as limit detectors, triggering protective and synergistic reflex muscle activity during movement.[27,46,64,141,149,158] In RC muscles and tendons, a large concentration of muscle spindles and Golgi tendon organs have been demonstrated in rabbits and rats,[3,22,105,166] but no human data exist. Current theory suggests that muscle spindles are the most important proprioceptors, especially during movement.[122] They also play a critical role in regulating muscle contraction through spinal reflexes that are essential for joint stability and accurate motor control.[101] Golgi tendon organs are equally important proprioceptors, signaling information about force and mass, and are also involved in the regulation of muscle contraction.[122]

The effect of tendon disruption on muscle spindles and Golgi tendon organs has been studied in a limited number of animal experiments concerning hindlimb muscles only. After tenotomy, muscle shortening and changes in the surrounding extrafusal tissue modify the morphology of muscle spindles that become slack and distorted.[169] In the chronically tenotomized muscle, atrophy of intrafusal fibers, degeneration of supplying axons, and fibrotic thickening of the capsule have been reported.[67,95] Functionally, acute tenotomy decreases muscle spindle discharge,[56,160,169] but interestingly, responsiveness of muscle spindles from the chronically tenotomized muscle has been shown to increase.[56,57,169] Shortening of intrafusal fibers, increased preliminary stretch caused by kinking of intrafusal fibers, change in passive mechanical properties, and increased sensitivity of spindles have been subsequently proposed as potential explanations for this phenomenon. These increases in muscle-tendon afferent outflow have also been suggested to result from non-proprioceptive discharge.[57,78] Increase in the amplitude of the monosynaptic reflex has also been repeatedly observed