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Lateral Epicondylalgia or Epicondylitis: What's in a Name?

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The condition first known as "tennis elbow" has been recognized for over a century. Typical signs and symptoms include pain and tenderness over the lateral epicondyle, exacerbated by resisted wrist extension and passive wrist flexion, and impaired grip strength. Although many tennis players may experience this condition, most cases are associated with work-related activities or have no obvious precipitating event. As a result, the term now most widely used is *lateral epicondylitis*. Yet, this name implies a pathological basis that is contrary to longstanding, albeit evolving, evidence that it is not an inflammatory condition. It is therefore recommended that it is time to adopt a new and more appropriate term, such as *epicondylalgia* (suffix *algia* means *pain*), that does not reflect such underlying pathology. This *Journal* has recently embraced this terminology. A review of the current histopathological evidence as detailed below provides a strong rationale for this change in nomenclature.

The traditional view is that this condition is initiated by macroscopic or microscopic tears at the common tendon of the wrist extensor muscles due to chronic overuse. As these tears attempt to unite, the healing surfaces are pulled apart with continued use of the hand, resulting in self-perpetuating and chronic inflammation. This theory was initially put forward by Dr J. Cyriax in 1936[5] and has since been widely accepted as the most plausible pathophysiological mechanism. Consequently, treatment has focused on controlling the inflammatory response through the use of nonsteroidal anti-inflammatory drugs and physical modalities such as ultrasound and ice. This inflammatory model also led to the use of the term *epicondylitis*, which was considered an improvement over the colloquial term *tennis elbow*.

Yet this theory has never been substantiated and indeed has been refuted as early as the 1970s by Dr R. Nirschl, based on the histopathological examination of over 600 cases of chronic lateral epicondylalgia.[13,17] His studies consistently demonstrated that the affected tendon (usually the extensor carpi radialis brevis [ECRB] tendon) was characterized by a dense population of fibroblasts, disorganized and immature collagen, and an absence of inflammatory cells. These findings are considered characteristic of a degenerative process, which he called "angiofibroblastic hyperplasia," now commonly known as tendinosis. Further histopathological and magnetic resonance imaging studies have revealed similar histological features and have confirmed the absence of an inflammatory process.[4,19-21] Recently, a study using an in vivo microdialysis technique demonstrated normal levels of E2 prostaglandin, a biochemical marker of inflammation, in ECRB tendons of patients with chronic lateral epicondylar pain.[1] Histopathological examination of similar chronic tendon conditions (Achilles, rotator cuff, and patellar tendinopathies) provide further support for the tendinosis-degenerative noninflammatory paradigm for chronic overuse tendon injuries.[2,10] Based on

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this growing body of evidence, Dr K. Khan and colleagues have been urging acceptance of the tendinosis model for several years.[3,11,16] Despite this, the traditional view, perhaps because of its inherent simplicity, has remained entrenched as reflected by the continued use of the term epicondylitis.

Certainly questions remain regarding the underlying pathophysiology of lateral epicondylalgia. It could be plausibly argued that because biopsy studies involve recalcitrant cases, the documented histological features represent the end stage of a process that commences with an early inflammatory phase. However, as reviewed by Khan et al,[9] to date neither animal models nor human studies provide support for the concept of a transition from normal tendon, through tendonitis to tendinosis. Moreover, duration of symptoms has not been found to be associated with recovery.[18,25] One would anticipate that someone in the acute inflammatory phase as indicated by a short duration of symptoms would respond more quickly to treatment, especially to treatment directed towards reducing inflammation; but this has not been shown to be the case.

Much debate and inquiry has also been directed towards identifying the cause of pain in the absence of an inflammatory mechanism of pain production. Some have hypothesized that certain byproducts of increased cellular activity or tendon degeneration, such as lactic acid and chondroitin sulphate, act as biochemical irritants that activate peritendinous nociceptors.[12,13] Findings from several recent studies suggest a potential neurogenic pain mechanism. In 1 study, increased concentrations of glutamate, an excitatory neurotransmitter and important pain mediator, were found in ECRB tendons in patients with chronic symptoms.[1] In 4 other studies,[6,7,14,15] 2 neuropeptides, substance P and calcitonin gene-related peptide, were located within sensory nerve bundles at the origin of the ECRB muscle in rats and humans. It is theorized that mechanical stimulation of the nociceptive fibers in the ECRB tendon from overuse causes local release of these neuropeptides, which subsequently precipitates vasodilation and plasma extravasation, or so-called "neurogenic inflammation." Finally, 1 study demonstrated an abnormal microvascular response of the skin overlying the painful epicondylar area, indicating a potential local dysfunction of the sympathetic nervous system.[22]

Lateral epicondylalgia is undoubtedly a complex condition and it may be years before all questions are answered. However, we cannot ignore the current literature, which provides consistent evidence that there is an absence of an inflammatory component. This would indicate that the current term, *epicondylitis*, is inaccurate and misleading, and use of the term epicondylalgia may, in its generality, be more appropriate. In addition to the ECRB tendon, many anatomical structures have been identified as possible sources of lateral epicondylar pain. These include local articular, ligament, and nerve lesions, as well as involvement of cervical spine structures leading to direct somatic pain referral or secondary facilitation of pain through altered nociceptive afferent transmission within the central nervous system.[8,23,24] The term *epicondylalgia* can encompass all causes of lateral epicondylar pain without assuming underlying pathology.

It is time to acknowledge that the traditional inflammatory model is both flawed and simplistic, and to abandon terminology that perpetuates an erroneous viewpoint. This is more than just a demand for accuracy of nomenclature: appropriate management and realistic treatment goals and prognosis are dependent upon a correct comprehension of pathoetiology. The term *epicondylalgia* reinforces the concept that this is a complex condition with potentially several pathophysiological mechanisms and underlying causes of pain. It reinforces the need to conduct thorough clinical assessments on each and every patient to identify, as best as possible, the contributing source(s) of pain in order to provide optimal management strategies. And, it reinforces the need to pursue ongoing research to elucidate the underlying pathoetiology and develop new and innovative treatment options that reflect evolving evidence. Embracing this new terminology is an important step in moving beyond the traditional perspective towards a progressive approach to understanding and managing this perplexing condition.

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