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 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. 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. 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. 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. Based on
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.

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 nocicep-
tors.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 vasodila-
tion 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.
REFERENCES