# Tennis elbow

## A clinicopathologic study of 22 cases followed for 2 years

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**Twenty-two elbows in 20 patients with persistent lateral pain due to epicondylitis had release of the common extensor origin and decortication of the lateral epicondyle. Specimens taken from the lateral epicondyle were examined histologically and compared with controls, and patients were reviewed clinically 2 (1/2–4) years after operation. Following surgery, symptoms were improved in 17 patients. Histologic examination of the bone-tendon junction revealed evidence of a repair response of variable degree, the most frequent features being mucopolysaccharide infiltration and bone formation. Fibrofatty degenerative changes were also present in some cases. There was no correlation between the intensity of the histologic reaction and the clinical outcome.**

We have examined the pathologic processes in the region of the lateral epicondyle in patients operated on for resistant tennis elbow. These were compared with age-matched controls and the findings related to the clinical outcome of surgery.

## Patients and methods

Twenty-two elbows in 20 patients were studied. There were 9 males and 11 females with a mean age of 44 (27–59) years. Seventeen cases involved the dominant arm. Symptoms were present on average for 2 years (range 6 months to 10 years), and all the cases had failed to respond to treatment with anti-inflammatory drugs, physiotherapy, or steroid injections.

Release of the common extensor origin and decortication of the lateral epicondyle were carried out under general anesthesia. With a tourniquet on the upper arm, a curved lateral incision was made over the lateral epicondyle. The common extensor origin was separated from the extensor carpi radialis longus anteriorly and from the anconeus posteriorly. Using an osteotome, the common origin was then detached from the epicondyle along with a thin section of bone. The tendon was then divided close to its bony attachment and allowed to retract distally, and the resulting specimen consisting of the cortical bone of the epicondyle with its tendinous insertion was sent for histologic examination.

Specimens were fixed in formalin-saline. They were then embedded in wax, sectioned, and stained with hematoxylin and eosin, periodic acid Schiff, alcian blue (pH 2.5), and Weigert's resorcin-fuchsin. The specimens occasionally required decalcification. Nine pairs of control specimens (similar to those taken at operation) were obtained from cadavers aged 22–81 years with no known history of tennis elbow and examined histologically using the same method. Pairs of specimens were obtained for each decade of age.

One patient was lost to follow-up, and another died from an unrelated cause. The remaining patients (20 elbows) were seen at follow-up examination 2 (1/2–4) years postoperatively. Symptoms were given one of four grades depending on the degree of pain present: none, mild (full activity with occasional ache), moderate (pain on moderate or heavy use), or severe (pain at rest or on minimal use). Elbows were examined for tenderness, range

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of movement, and grip strength was measured using a grip-strength sphygmomanometer adaptor.

Of the 20 patients reviewed, 5 were completely cured, 12 had improved, 2 had no change, and in 1 the elbow symptoms were worse after operation (Table 1). Five elbows had residual tenderness over the lateral epicondyle, and in three elbows there was mild limitation of extension of less than 10°. Grip strength was expressed as the ratio of the value for the affected to that for the unaffected arm for the 16 cases that were unilateral. The mean ratio was 0.92 (0.4–1.73). The grip strength ratio was not correlated with the patients' symptoms, and it was therefore felt that grip strength measured in this way was not a very useful indicator.

Table 1. Clinical features of elbows in each pathologic grouping

| Case | Age | Sex | Duration symptoms (yr) | No. inj. | Symptoms | |
|---|---|---|---|---|---|---|
| | | | | | Preop | Postop |
| Vigorous reactions | | | | | | |
| 1 | 47 | F | 1 | 3 | 3 | 0 |
| 2 | 42 | F | 2 | 4 | 3 | 0 |
| 3 | 51 | M | 3 | 5 | 3 | 2 |
| 4 | 43 | M | 2 | 1 | 3 | 3 |
| 5 | 44 | F | 2.5 | 6 | 3 | 0 |
| 6L | 50 | F | 2.5 | ++ | 3 | 1 |
| 6R | 50 | F | 1.5 | ++ | 3 | 2 |
| 7 | 30 | F | 2.5 | 3 | 3 | 1 |
| Active reactions | | | | | | |
| 8 | 51 | F | 1 | 1 | 3 | 1 |
| 9 | 55 | M | 0.5 | 8 | 3 | 2 |
| 10 | 56 | F | 2 | – | 3 | 1 |
| 11 | 51 | M | 10 | 6 | 3 | 3 |
| 12 | 59 | M | 2.5 | 3 | 3 | 0 |
| 13 | 34 | M | 1.5 | 3 | 3 | 2 |
| 14 | 48 | F | 6 | 4 | 3 | 1 |
| Degenerative/fibrofatty | | | | | | |
| 15 | 40 | F | 2.5 | 2 | 3 | 2 |
| 16 | 44 | F | 0.5 | 5 | 3 | 0 |
| 17R | 28 | M | 1 | 2 | 3 | 1 |
| 17L | 28 | M | 1 | 2 | 3 | 2 |
| 18 | 39 | M | 1 | 3 | 2 | 2 |

Symptoms: 0 none, 1 mild, 2 moderate, 3 severe.

## Results

### *Control specimens*

Figure 1 shows normal cortical bone at the epicondyle with a clear interface with the fibrocartilaginous zone followed by a smooth transition to tendon fibers. These findings were largely the same throughout the age range of the controls, with the exception that there were mucopolysaccharides in older specimens.

### *Operation specimens*

None of the operation specimens were normal. The appearances were variable, but there were three main types of appearances.

Eight specimens showed vigorous reactive changes with new bone formation and remodeling. There was widespread mucopolysaccharide infiltration and new bone formation at the bone-tendon junction. There was evidence of both osteoblastic and osteoclastic activity indicating bone remodeling (Figures 2, 3).

Seven specimens had less florid changes, but had signs of an active mesenchymal response—the most common feature being an excess of mucopolysaccharide at the tendinous insertion.

In five specimens the appearances were more degenerative in nature, with areas of fibrofatty change and elastosis (Figure 4).

Of the 2 patients lost to clinical review, the histologic examinations revealed vigorous reactive changes in 1 and fibrofatty change in the other. No preparations showed evidence of vascular ingrowth or inflammatory cell infiltration. Granulation tissue was not seen, nor were tendon tears observed—neither gross nor microscopic ones.

There was no correlation between the histologic appearances and the outcome of surgical treatment, various degrees of recovery occurring in each group, nor was any relationship to steroid treatment apparent (Table 1). There was a preponderance of females in the group with vigorous histologic reactions, and interestingly, the group with degenerative changes tended to be somewhat younger with shorter clinical histories, though the number of patients was too small to draw any firm conclusions from these observations.

## Discussion

Previous studies have examined the histologic appearance of the common extensor origin. Bosworth (1955) found degenerative changes in the orbicular ligament, and Goldie (1964) found granulation tissue in the subtendinous space of the extensor origin. More recent papers have localized the

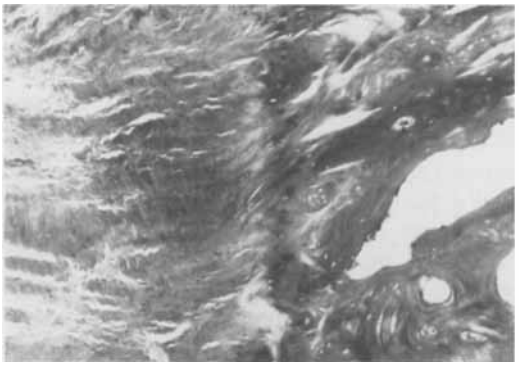

Figure 1. Control specimen. Bony tendinous junction showing wavy tendon fibers (left) and mature lamellar bone (right) with fibrocartilage between. HE, ×40.

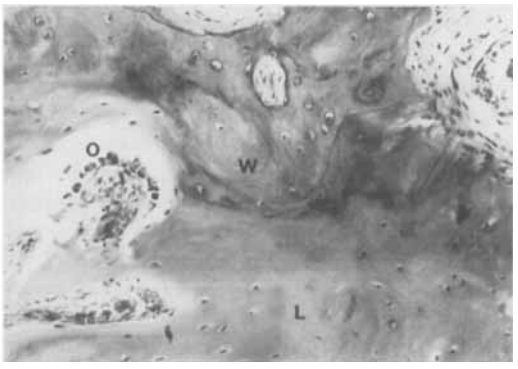


Figure 2. Lamellar (L) and woven (W) bone at the tendinous insertion. A rim of active osteoblasts to the left (O). HE, ×200.

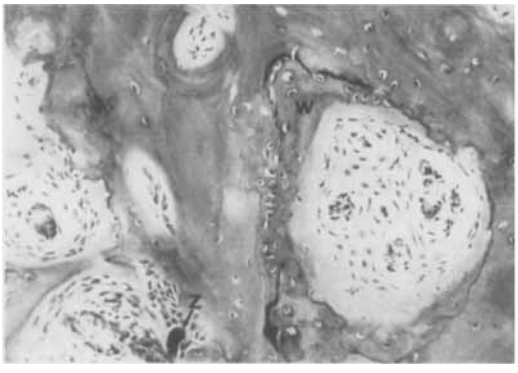


Figure 3. Active new bone formation (W) and osteoclastic activity (Z) at the tendon insertion. HE, ×200.

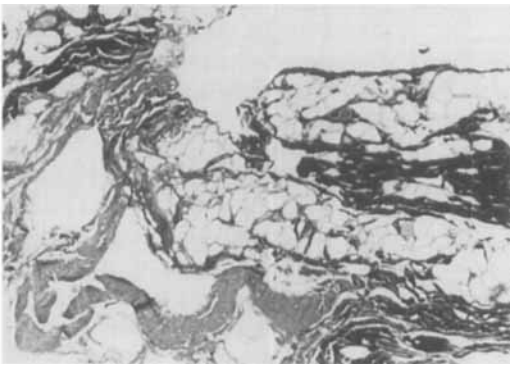

Figure 4. Elastotic change staining deep black in the fibro-fatty tissue next to the insertion of the tendon. Weigert's resorcin fuchsin, ×80.

lesion to tears in the extensor carpi radialis brevis tendon (Coonrad and Hooper 1973, Nirschl and Pettrone 1979). In his extensive study of the subject, Goldie (1964) examined 58 operative specimens histologically, of which 45 included the tendinous insertion. His control specimens had similar appearances to those in our study; but in the operative cases, he found no specific abnormalities of the tendinous insertion. The majority of changes in his study were in the aponeurosis and consisted of cellular and vascular invasion, particularly in the subtendinous space, with breakage of some collagen fibers and fibrinoid degeneration in some cases. Coonrad and Hooper (1973) found calcification at the epicondylar origin in 2 of their 39 patients, but they reported tears or scar tissue in the aponeurosis accompanied by round cell infiltration and degeneration in the majority. Similarly, Nirschl and Pettrone (1979) found the tendon to be invaded by granulation tissue, referred to as angiofibroblastic hyperplasia, but they made no mention of the osteotendinous junction. The study of Uhthoff et al. (1978) noted mesenchymal cell and vascular infiltration in the tendon tissue, but no inflammatory cells. Five specimens showed soft-tissue calcification near the insertion, but no new bone formation. In contrast to previous studies, we found extensive changes at the osseotendinous junction in the form of bone formation and mucopolysaccharide infiltration, but little evidence of granulation tissue at this site.

The presence of tears in the aponeurosis of the extensor tendons has been widely quoted as an underlying feature in tennis elbow. In the studies both of Coonrad and Hooper (1973) and of Nirschl and Pettrone (1979), the majority of patients operated on had grossly visible tears or scar tissue indicative of previous tears. We found no macroscopic tears or other gross changes in our series, in

common with several other studies (Goldie 1964, Boyd and MacLeod 1973, Posch et al. 1978, Calvert et al. 1985). Nirschl and Pettrone (1979) suggested that this may be due to failure to visualize the tendon of the extensor carpi radialis brevis adequately. Our operative method, however, like that of Calvert et al. (1985), separates the extensor carpi radialis longus from the common origin, and should have demonstrated any such changes. It would seem that macroscopic tears and gross degenerative changes are present only in some cases of tennis elbow and may be a reflection of patient selection or referral methods.

On the other hand, Goldie (1964) noted microscopic tears in the absence of naked-eye changes in many of his cases as did Uhthoff et al. (1978). We were looking specifically at the bone-tendon insertion, and we did not see any microscopic tears in the small piece of tendon included in the average specimen, though it is possible these were present elsewhere in the tendon.

The changes found in our study were clearly different from the control specimens and suggest that many patients with tennis elbow have microavulsion fractures at the lateral epicondyle in addition to possible microscopic tears in the tendon proper. The tendon, insertion and bony epicondyle may therefore be seen as a functional unit, the parts of which are subject to tissue damage, either together or separately. We would agree with Uhthoff et al. (1978) that the symptoms of tennis elbow are then due to the resulting repair reaction rather than to degenerative changes. Five specimens in our study did have degenerative changes, and some of these were in a slightly younger age group. However, in view of the findings in the other cases and the lack of degenerative changes in our controls at any age, it seems reasonable to assume that such changes were a secondary rather than a primary event.

In conclusion, our study demonstrates the involvement of the bone-tendon junction in the pathogenesis of tennis elbow. This appeared to be principally a repair process in response to tissue damage. The process is of an indolent character in no way resembling an acute or chronic inflammatory response. The patients studied had severe symptoms requiring surgical treatment, but there was no correlation between the pathologic changes and the outcome of surgery.

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