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## Ultrasound-guided autologous blood injection for tennis elbow

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**Abstract Objective:** To assess the efficacy of autologous blood injection under sonographic guidance for the treatment of lateral epicondylitis.

**Design and Patients:** Thirty-five patients (23 men, 12 women, mean age 40.9) with refractory lateral epicondylitis (mean symptom duration 13.8 months) underwent sonographic evaluation prior to dry-needling the tendon and injection with autologous blood. Patients were reviewed, and measures of Nirschl and Visual Analogue Scores (VAS) were taken pre-procedure and post-procedure, at 4 weeks and 6 months.

**Results:** Following autologous blood injections, significant reductions were reported for Nirschl scores, which decreased from a median (inter-quartile range) pre-procedure score of 6 (6–7), to 4 (2–5) at 4 weeks ( $p<0.001$ ), and to 0 (0–1) at 6 months ( $p<0.001$ ). Similarly, significant reductions were reported for VAS scores from a median (inter-quartile range) pre-procedure score of 9 (8–10), to 6 (3–8) at 4 weeks ( $p<0.001$ ), and to 0 (0–1) at 6 months

( $p<0.001$ ). Sonography demonstrated a reduction in the total number of interstitial cleft formations and anechoic foci; a significant reduction in tendon thickness from a mean (SD) of 5.15 mm (0.79) at baseline to 4.82 mm (0.62) at 6 months post-procedure ( $p<0.001$ ) was observed. Hypoechoic change significantly reduced from a median (inter-quartile range) of 7 (6–7) at baseline to 2 (1–3) at 6 months post-procedure ( $p<0.001$ ). Neovascularity also significantly decreased from a median (inter-quartile range) of 6 (4–7) at baseline to 1 (0–3) at 6 months post-procedure ( $p<0.001$ ), although sonographic abnormality remained in many asymptomatic patients.

**Conclusions:** Autologous blood injection is a primary technique for the treatment of lateral epicondylitis. Sonography can be used to guide injections and monitor changes to the common extensor origin.

**Keywords** Ultrasound · Autologous blood · Lateral epicondylitis · Dry needling · Treatment · Tendon healing

### Introduction

Tennis elbow or lateral epicondylitis are terms used to describe a syndrome of pain involving the common extensor origin of the forearm musculature where it arises from the lateral epicondyle. It was first reported in the literature in 1873 by Runge, and later in 1896 by Bernhardt [1]. It is generally a self-limiting condition often seen in tennis

players, athletes and tradesmen. The diagnosis is clinical, usually requires no imaging, and most patients respond to conservative treatment. However, in a number of cases the condition is refractory to treatment, and intervention for resolution of symptoms is necessary.

Lateral epicondylitis is thought to be secondary to degeneration of the common extensor origin. It is now accepted that it is not an inflammatory condition but a

fibroblastic and vascular response, pathologically known as angiofibroblastic degeneration although more commonly referred to as tendinosis [2]. Treatments such as steroid injections have been focused on a presumed inflammatory process that does not exist in tendinosis. While it is recognised that steroid injections may provide symptomatic relief, there is no evidence that steroids promote healing. Other treatments such as forms of immobilisation may even cause deleterious effects rather than cure [3, 4]. Various surgical treatments have been described which result in an improvement in outcome, but morbidity such as neurovascular injury, fracture and ligament rupture has been reported, as has failure [5].

More recently, an injection of autologous blood has been reported for the treatment of lateral epicondylitis [3]. It is hypothesized that transforming growth factor- $\beta$  and basic fibroblast growth factor carried in the blood will act as humoral mediators to induce the healing cascade [6]. We describe a modification of this technique using sonographic guidance, and report our results. Ultrasound can document the pathology prior to the injection and accurately identify the site for injection, and thereafter monitor tendon healing. We postulate that this should lead to an improvement in patient outcome following autologous blood injection.

### Materials and methods

Thirty-five consecutive patients with refractory lateral epicondylitis were recruited into the study following informed consent and institutional review board approval. There were 23 men and 12 women, with a mean age of 40.9 years (age range 26–62). The mean time of symptoms was 13.8 months (range 6–48 months).

Inclusion criteria included symptoms of at least 6 months following presentation to a consultant specialist, failure of conservative treatment including rest, physiotherapy and steroid injection, and confirmation of the diagnosis on MR imaging. Seven patients with intraarticular pathology (two), chondral defects (two) or lateral collateral ligament tears (two) were excluded from the study following the MR study. Full-thickness tears of the common tendon origin were also excluded from the study. Furthermore, patients who had had a steroid injection or other intervention in the preceding 3 months were excluded from the patient cohort.

Patients were asked to rate their pain on a visual analogue scale (VAS) 0 to 10, with 0 representing no pain and 10 the worst pain they had ever experienced. In addition they were categorised according to the Nirschl staging 0–7 (Table 1) [7].

All patients were examined with a 15L8W transducer on a Siemens (Acuson) Sequoia ultrasound machine (Siemens Medical Solutions, Mountain View, CA, USA). Sonographic evaluation of the extensor tendon origin was performed, and the diagnosis of tendinosis was confirmed according to four sonographic criteria (Fig. 1). These were tendon size,

**Table 1** Nirschl score

Phase 1	Mild pain with exercise, resolves within 24 hours
Phase 2	Pain with exercise, exceeds 48 hours
Phase 3	Pain with exercise, does not alter activity
Phase 4	Pain with exercise, alters activity
Phase 5	Pain with heavy activities of daily living
Phase 6	Pain with light activities of daily living, intermittent pain at rest
Phase 7	Constant pain at rest, disrupts sleep.

echotexture, partial discrete fibril discontinuity (tears) and neovascularity [8]. Tendon size was measured by placing one marker on the tendon surface and another marker on the cortical bony interface of the lateral epicondyle, at the tendon midportion. For reproducibility, this was performed 5 mm from the joint margin. Echotexture was evaluated by identifying areas of hypoechoic change within the tendon which had otherwise preserved its fibrillar pattern. A semi-quantitative score of 1–10 was awarded, with 0 representing a normal echogenic tendon and 10 representing diffuse hypoechoic change seen throughout the entire common tendon origin. Discrete tears within the tendon were identified as focal areas of anechoic change with no fibres intact, or as distinct hypoechoic planes of fibril discontinuity. The number and size of these tears was recorded. In addition, neovascularity was assessed by placing the color Doppler sensitivity at threshold and turning back the gain. A percentage estimate of the maximum number of color pixels occupying the tendon origin was made, and given a score of between 1 and 10. For subsequent visits, the color Doppler parameters were recorded and maintained for the follow-up examination.

Two ml of autologous blood were drawn from the contralateral antecubital fossa of the patient. This was gently



**Fig. 1** Patient positioning: patient places arm on workbench in neutral position, and probe is placed over lateral epicondyle

shaken to prevent the blood from clotting. Two ml of bupivacaine (0.25%) were infiltrated along the surface of the tendon using a 23G needle. After a suitable interval of several minutes to allow the anaesthetic to work, the needle tip was positioned into the site of maximal tendon injury and the tendon was dry-needled for a period of 1 min to create fenestrations and cause fibril disruption and internal bleeding (Fig. 2). Thereafter, the blood was slowly injected into the site of tendinosis and fibril discontinuity. The time from blood aspiration to injection was approximately 5 min.

Patients were told to continue activities of normal daily living but to avoid any activities that were likely to cause symptoms. They were rescheduled for a follow-up appointment 4 weeks later, at which time a second sonographic evaluation was made, followed by a second blood injection being given. Patients then returned at 12 weeks after the initial injection for clinical evaluation and sonographic assessment, and were given the option of a third blood injection as felt warranted by the patient. Final sonographic and clinical follow-up was performed at 6 months. All ultrasound assessment and injections were performed by a solitary musculoskeletal radiologist of more than 10 years experience (DC).

The VAS and Nirschl scores were recorded prior to the procedure, and recorded again at the 4-week sonographic assessment and 6-month post-injection follow-up.

#### Statistical analysis

The Kolmogorov-Smirnov test was used to assess the normality of all the data, for the ultrasound findings and the VAS and Nirschl scores.

Ultrasound findings data that did not follow a normal distribution was analysed using the Wilcoxon Signed Ranks test. Data that satisfied the normality assumption was analysed using a Paired Sample *t*-test. Comparisons were made between the tendon thickness, hypoechoic and

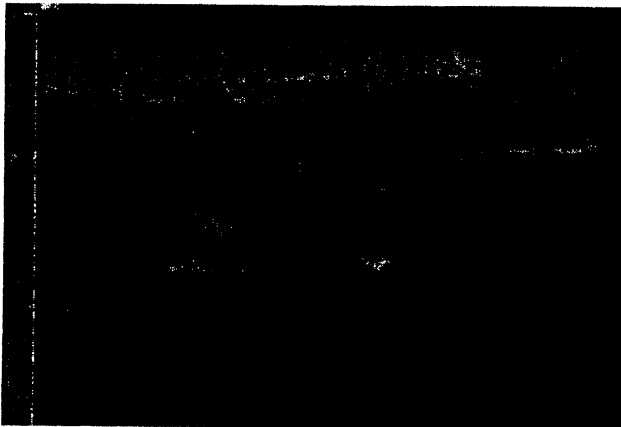


Fig. 2 Needle inserted into site of tendinosis. The tendon origin was dry-needled for 60 s prior to autologous blood injection

neovascularity scores before the autologous blood injection (baseline) and 6 months after the autologous blood injection (post-procedure).

The data from the VAS and Nirschl scores did not follow a normal distribution, and was therefore analysed using the Wilcoxon Signed-Ranks test to assess differences between pre- and post-procedural measures of Nirschl and VAS pain scores, taken at 4 weeks and 6 months post-procedure.

All statistical analysis was performed using SPSS for Windows, version 12.0 (Chicago, IL, USA) and *p* values of <0.05 were considered statistically significant.

#### Results

Between Jan 2004 to May 2005, 54 patients were referred for autologous blood injection, having met the inclusion criteria. There were 12 patients excluded following MRI and/or ultrasound; seven patients were lost during follow-up. Thirty-five patients (23 men, 12 women) completed the course of treatment, and were followed up for a period of up to 6 months following the initial injection. The mean symptomology was 13.83 months, and the range was 6–48 months.

Twenty-six patients had two autologous blood injections, while nine patients had three injections (mean no. of injections 2.26). Thirty-two (91.4%) patients expressed satisfaction with both the procedure and the outcome, and 31 (88.6%) said they would be prepared to undergo the procedure again.

Two patients felt the procedure had failed, and subsequently went on to have a lateral release and tendon repair at surgery. One of these patients had reported improvement at 4 weeks with a VAS of 4 and Nirschl score of 2. Prior to the intervention their VAS score was 10 and the Nirschl score was 7. Unfortunately, at six months their VAS score was 9 and the Nirschl score was 7. One patient reported a 50% reduction in their symptoms following three autologous blood injections, and continues to be managed conservatively.

In the remaining group of patients the median (IQR) VAS pain score at pre-procedure was 9 (8–10) with a range of 6–10, which had decreased to 6 (3–8) at 4 weeks, with a range of 0–9. Statistical analysis found the difference to be significant ( $z=4.88$ ,  $p<0.001$ ). In addition, statistical analysis between the VAS pain score at pre-procedure and at 6 months, which had a median (IQR) of 0 (0–1) and a range of 0–10, revealed a significant decrease ( $z=5.16$ ,  $p<0.001$ ).

The Nirschl score at pre-procedure had a median (IQR) of 6 (6–7) and a range 4–7, which at 4 weeks significantly decreased ( $z=4.64$ ,  $p<0.001$ ) to a median (IQR) of 4 (2–5) and a range of 0–7. The difference was found to be statistically significant. The analysis between the Nirschl score at pre-procedure, and the Nirschl score at 6 months, which had a median (IQR) of 0 (0–1) and a range of 0–7,

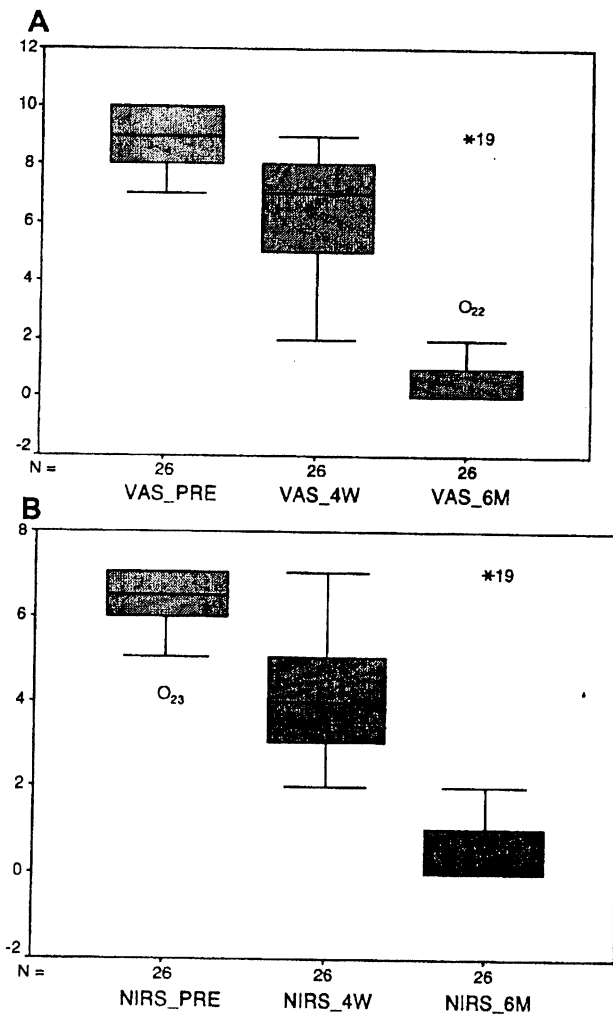
revealed a statistically significant difference ( $z=5.05$ ,  $p<0.001$ ).

Figure 3a displays the VAS pain scores and Fig. 3b displays the Nirschl scores diagrammatically in Box and Whisker plots, for the three time periods in which patients attended follow-up. The midline inside the box represents the median score, the box itself represents the lower and upper quartiles (inter quartile range), and the whiskers represent the remainder of the range, with the outliers indicated by a circle or asterisk.

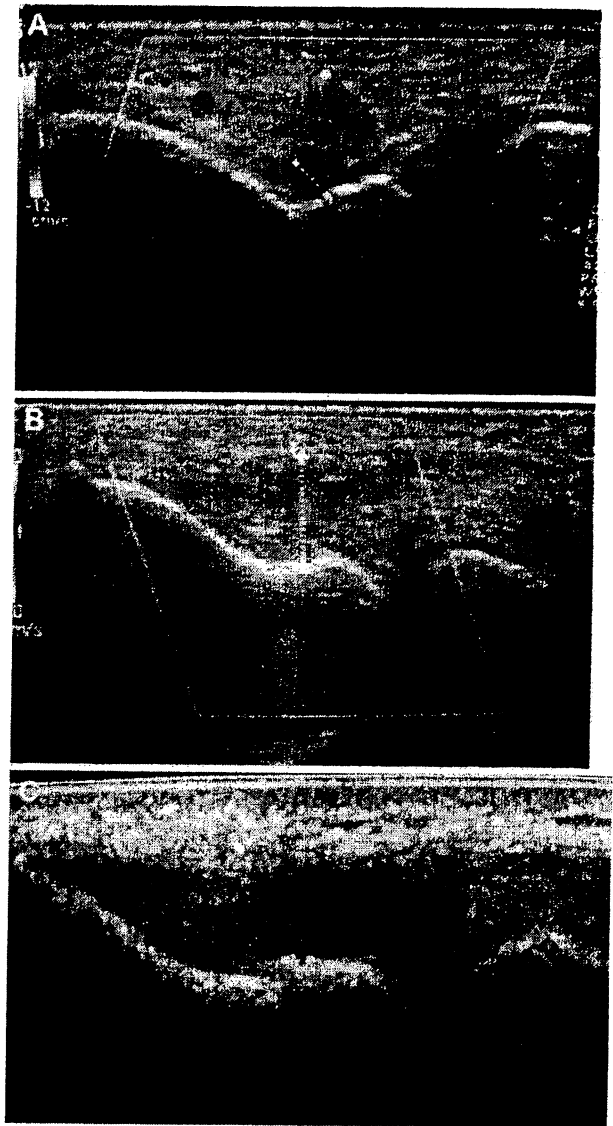
There was not any occurrence of infection, neurovascular damage or tendon rupture in the group of patients following their autologous blood injections.

Tendon thickness decreased marginally from a mean (SD) of 5.15 mm (0.79 mm) to 4.82 mm (0.62 mm) at

baseline (range=3.7–6.4) to post-procedure (range=3.7–5.8) respectively, representing a significant 6.4% reduction ( $t=4.987$ ,  $df=34$ ,  $p<0.001$ ). Hypoechoic change decreased between baseline—with a median (IQR) of 2 (1–3) and a range of 0–4—and 6 months, with a median (IQR) of 7 (6–7) and a range of 2–9. The difference was statistically



**Fig. 3** a Box and Whisker plot of VAS pain scores pre- and post-procedure at 4 weeks and 6 months. b Box and Whisker plot of Nirschl scores pre- and post-procedure at 4 weeks and 6 months



**Fig. 4** a Sonography of lateral epicondylitis in a 28-year-old tennis player. The tendon size is measured, the number of interstitial scars counted and neovascularity assessed prior to autologous blood injection. b Scan 4 weeks post-autologous blood injection shows a thickened and hypoechoic tendon, with resolution of the interstitial cleft and diminution of the neovascularity. c Scan at 6 months. Tendon remodelling with reduction in size and hypoechoic change. Interstitial clefts have resolved and there is some restoration of fibrillar pattern

significant ( $z=5.18$ ,  $p<0.001$ ). In addition some restoration of the echogenic fibrillar pattern was observed (Fig. 4).

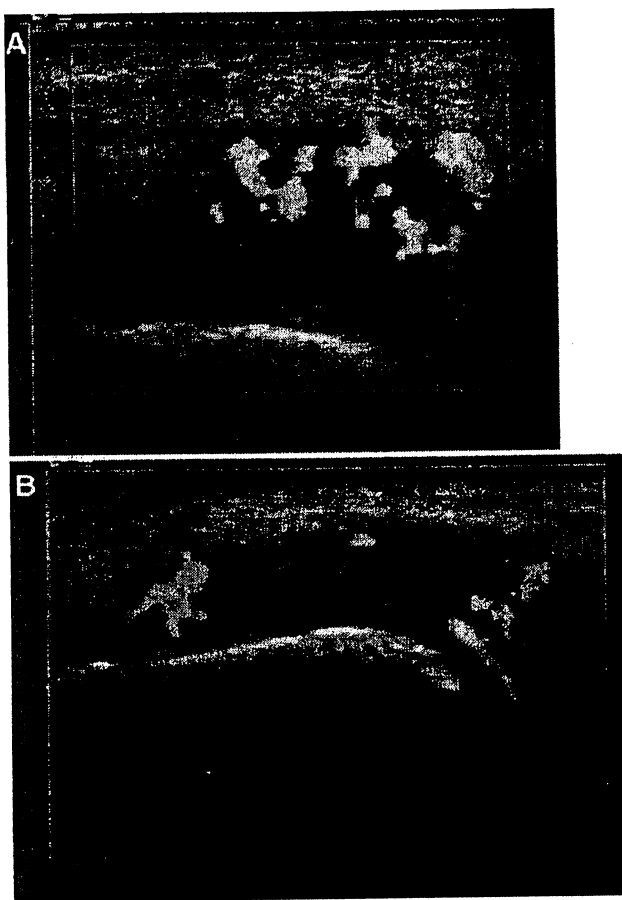
At baseline, 33/35 patients demonstrated pathological vascularity of the tendon origin, which was no longer present at the 6-month follow-up in 19/35 patients. Furthermore, there was a reduction in the extent of neovascularity in 12 patients, although this had not completely resolved (Fig. 5). In four patients there was no change in the colour Doppler score. Overall, in all the patients (including two of the three failures) the number of blood vessels occupying the tendon decreased markedly over the 6-month period; this difference was statistically significant ( $z=5.03$ ,  $p<0.001$ ). At baseline the median (IQR) was 6 (4–7) with a range of 2–9, and at post-procedure the median (IQR) was 1 (0–3) with a range of 0–4.

There was also a total reduction in the number of interstitial clefts and anechoic foci from 63 to 37, which

were all felt to represent intrasubstance tears. An interesting observation was an alteration in the appearance of the anechoic foci and interstitial clefts, with blurring of the peripheral margins and filling of these spaces with echogenic foci. Although we have no histological correlation, we postulate that this might represent granulation/immature scar tissue.

Although there was a general improvement in all of the sonographic parameters, not one of the treated tendons returned to a normal sonographic appearance, although some had a "near normal"-like appearance (Fig. 4c).

Twenty-five of the patients reported temporary pain and/or stiffness following the injections. This tended to be most pronounced following the first injection and usually resolved within a day or two. Three patients required the use of short-term narcotics. One patient returned with diffuse symptoms from the axilla to the wrist, which showed no sonographic abnormality on follow-up but was treated with short-course oral corticosteroids.



**Fig. 5** a Autologous blood injection in 34-year-old tradesman with lateral epicondylitis. A transverse sonogram demonstrates tendon thickening, hypoechoic clefts and neovascularity. b Sonography at 6 months shows a decrease in tendon size, but there is persistent neovascularity and hypoechoic clefts. Nevertheless, the patient was symptom-free

## Discussion

The primary abnormality of tennis elbow involves the origin of the extensor carpi radialis brevis, and less commonly the anterior aspect of the extensor digitorum tendon [8]. The cause is generally considered to be repetitive microtrauma sustained during supination of the forearm and dorsiflexion of the wrist. Repetitive microtrauma results in tendon degeneration. A chronic cycle of tendon degeneration and repair ensues, with weakening of the common extensor origin and with potential for rupture. Histopathological correlation of over 600 cases of tennis elbow demonstrated disrupted collagen fibres, increased cellularity and neovascularisation in the common extensor tendons [9, 10].

Tendon healing occurs in three overlapping phases [11]. There may be a brief period of acute inflammation causing pain for a few days after tendon injury, but this is not felt to be the cause of patient's pain [12, 13]. Erythrocytes and neutrophils enter the injury site, and phagocytosis of necrotic material occurs. Vasoactive and chemotactic factors are released, leading to initiation of angiogenesis, tenocyte proliferation and recruitment of more inflammatory cells [14]. Tenocytes gradually migrate to the injury site, and type-III collagen synthesis commences. After 6 weeks, the remodelling phase begins with decreased collagen and glycosaminoglycan synthesis, and the repair tissue changes from cellular to fibrous. Collagen fibres become aligned according to the direction they are stressed [15]; thereafter, there is a gradual change from fibrous tissue to scar-like tendon tissue over the course of 1 year, during which tenocyte metabolism and tendon vascularity decline [16].

The mechanism of short term relief following steroid injection or needling is not understood. It is however postulated that fenestration of an area of tendinosis with needling may promote beneficial bleeding into new channels

created through mucoid degeneration. The mechanical disruption may initiate healing response in the tendon [2, 17].

Neovascularisation of the tendon has been postulated as a cause for symptoms in patients with tendinosis. The paratenon is highly innervated and vascularised when compared with the tendon, and is more likely to be affected by neurotransmitters such as substance P causing mast-cell degranulation and secretory activity. Neural activity may be amplified when mast cells are released, having an impact on vascular elements and fibroblasts. This process may explain the neovascularisation of the tendon [17]. Our study showed that there was a decrease in the number of blood vessels in the common extensor origin following autologous blood injection, with complete resolution in a small number of patients. However, many patients had persistent blood vessels remaining in the tendon origin at the 6-month follow-up scan, despite resolution of their symptoms. Clearly the cause of patients' symptoms are more complex than can be attributed to neovascularity alone.

Other researchers have demonstrated that the size of an abnormality as assessed on ultrasound does not necessarily correlate with increased pain or symptoms [18–20]. We observed reductions in tendon thickness, hypoechoic changes and incomplete resolution of intrasubstance tears following autologous blood injection. However, our study demonstrated that patients can be symptom-free following autologous blood injection, and yet still show these abnormalities on sonography. Unfortunately, the study does not explain how an autologous blood injection improves symptoms.

The study by Edwards and Calandruccio showed that 22/28 patients responded to autologous blood injections, with average Nirschl Scores decreasing from 6.5 to 2.0 with a mean follow-up of 9.5 months. Their technique differed from ours in that they mixed the autologous blood with local anaesthetic before injecting along the undersurface of the extensor carpi radialis brevis tendon without guidance. In contrast, we injected 0.25% bupivacaine along the superficial surface of the tendon, to have an anaesthetic effect before dry-needling and injecting the blood into the areas of tendinosis under guidance. Dry-needling has the advantage of fenestrating the tendon, causing further fibril disruption and local bleeding before the autologous blood injection. We believe that a targeted approach is more likely to produce better results. Furthermore, we routinely performed two injections, with an option of a third, and monitored any changes in the tendon with ultrasound. We have observed that patient benefit occurs mostly between 2–4 weeks following the injection, and that the pain relief appears cumulative following each injection. We postulate that the optimal time interval between injections should be about 4–5 weeks, and if a patient is likely to respond then should do so by the completion of three injections. We

routinely inform all patients that the treatment is a 3-month healing process.

In our group of 35 patients there were two clear failures to the treatment, and a third patient who no longer had pain at rest but was unable to lift heavy objects or perform DIY activities. Two of these patients are tradesmen, and the third incurred his injury following a fall. There was no improvement in the symptoms following the injection. There were no identifiable features at baseline ultrasound to suggest a poor outcome in this group. Furthermore, there were no differential signs to suggest which patients might require three injections rather than two. We excluded patients with intraarticular pathology or substantive tears of the tendon. Like most procedures, we believe that patient selection is critical to the success of the procedure. At our institution, patients with partial or full-thickness tears of the CEO or involvement of the lateral collateral ligament are referred directly to surgery.

There are a number of limitations to our study. The most obvious is that all sonographic observations were made by a solitary radiologist, and hence we have no measure of either intra- or inter-observer variability. There is no control group, and the subject bias inherent in our study was unavoidable. At the study outset, we opted to use colour Doppler, although it could be argued that power Doppler may be more sensitive and less susceptible to flow direction. Our follow-up period of 6 months is moderate, but we do not know if this group of patients is predisposed to recurrent injury and/or symptoms. Furthermore, although we believe that dry-needling and autologous blood injected into a focus of tendinosis incites a healing response with the formation of scar tissue, we have no histopathological correlation.

A limitation of the study design is that it does not allow for comparison with ultrasound-guided dry-needling alone. Unfortunately, the dry-needling technique for treatment of tendinopathy has not been reported in the literature. However, the senior author has significant experience with the dry-needling technique. We estimate that ultrasound-guided dry-needling alone results in satisfactory outcomes in approximately 60% of patients, although this has not been documented or reported. Clearly, more research is required. The authors also have experience with autologous blood injections in the rotator cuff, common flexor origin, patellar tendon and Achilles tendon, all of which show encouraging results.

In summary, we feel the combined action of dry-needling and autologous blood injection under ultrasound guidance is an effective way in which to treat patients with refractory lateral epicondylitis, as demonstrated by a significant decrease in pain and fall in Nirschl scores. Further research using autologous blood for the treatment of lateral epicondylitis and other tendinopathies is required.

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