RUPTURE OF THE CALCANEAL TENDON
TREATMENT BY EXTERNAL FIXATION

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Thirty-three consecutive patients with complete ruptures of the calcaneal tendon were treated by external fixation. The patients were assessed both objectively and subjectively, and the results classified as excellent, good, fair and poor.

In 30 patients the result was excellent or good. There were no infections or re-ruptures. Two patients with fair results had sural nerve injury. The only patient with a poor result had Sudeck’s atrophy. It is felt that this operation satisfies the need for a new technique which is simple and combines the advantages of both surgical and non-surgical treatment without their major complications.

There has been considerable debate concerning the appropriate treatment of fresh ruptures of the calcaneal tendon, and that debate continues (Nistor 1981). Conservative treatment with a plaster cast in an equinus position for two months, popularised by Lea and Smith (1968), may be associated with an unacceptably high incidence of re-rupture, which was more than 30% in one series of 20 patients (Persson and Wredmark 1979) (Table I). In addition, dynamometric and calibrated load cell measurements have shown that the ultimate force of calf contraction is lower with conservative treatment than after operative repair (Jacobs et al. 1978, Nistor 1981).

Proponents of surgical repair describe a much lower incidence of re-rupture, varying from zero in 74 cases (Percy and Conochie 1978) to four out of 92 cases (Arner and Lindholm 1959). Surgical treatment may, however, be associated with significant complications, such as deep wound infection, skin slough, sinus formation, tendinocutaneous adhesions and sural nerve injury; such complications may occur in as many as one in three patients (Arner and Lindholm 1959; Nistor 1981) (Table II). Deep venous thrombosis and pulmonary embolus have been reported with both methods of treatment (Arner and Lindholm 1959; Percy and Conochie 1978; Persson and Wredmark 1979).

In a comparative study of conservative treatment versus surgical repair Nistor (1981) favoured non-surgical treatment, but other reports by Persson and Wredmark (1979) and Tom-Harald (1980) favour surgical repair. This present paper reports a new technique for the treatment of fresh ruptures of the tendon by external fixation. The method is simple and seems to combine the advantages of both surgical and non-surgical management. Tendon continuity and length are restored, and many of the complications of surgery are avoided.

Table I. Incidence of re-rupture in reported series of conservatively treated patients

<table>
<thead>
<tr>
<th>Author(s)</th>
<th>Number of cases</th>
<th>Number of re-ruptures</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gillies and Chalmers 1970</td>
<td>7</td>
<td>1</td>
<td>14</td>
</tr>
<tr>
<td>Lea and Smith 1972</td>
<td>66</td>
<td>7</td>
<td>11</td>
</tr>
<tr>
<td>Inglis et al. 1976</td>
<td>31</td>
<td>9</td>
<td>29</td>
</tr>
<tr>
<td>Lindholdt and Munch-Jørgensen 1976</td>
<td>14</td>
<td>2</td>
<td>12</td>
</tr>
<tr>
<td>Stein and Luekens 1978</td>
<td>8</td>
<td>1</td>
<td>13</td>
</tr>
<tr>
<td>Jacobs et al. 1978</td>
<td>32</td>
<td>7</td>
<td>22</td>
</tr>
<tr>
<td>Persson and Wredmark 1979</td>
<td>20</td>
<td>7</td>
<td>35</td>
</tr>
<tr>
<td>Tom-Harald 1980</td>
<td>10</td>
<td>3</td>
<td>30</td>
</tr>
<tr>
<td>Nistor 1981</td>
<td>60</td>
<td>5</td>
<td>8</td>
</tr>
<tr>
<td>TOTAL</td>
<td>248</td>
<td>42</td>
<td>17</td>
</tr>
</tbody>
</table>

TECHNIQUE OF EXTERNAL FIXATION

The basic principle of the technique is the approximation of the ruptured ends by two Kirschner arthrodesis wires: one is inserted into the calcaneum and the other into the proximal part of the ruptured tendon. The procedure has been carried out under general anaesthesia in seven patients and under local anaesthesia in 26. No tourniquet has been used.

The patient lies prone with both ankles just overhanging the edge of the operating table. The principle of
the operation is illustrated in Figures 1 to 3. The details are described below and illustrated in Figures 4 to 7.

1. A Kirschner wire, size 0.08 and 10 to 12 cm long, is inserted through two stab wounds, one on each side of the calcaneum.

2. Two stab wounds are made, one lateral and the other medial, 2.5 cm above the gap in the tendon (point A in Fig. 1).

3. The ankle is put into an equinus position to relax the skin around the tendon. Using a small curved haemostatic clamp, the skin and subcutaneous tissue on both sides of the tendon are separated by rotating the clamp in a full circle.

4. The skin is elevated with a blunt hook (to point B in Fig. 2). A second Kirschner wire is inserted into the tendon from the lateral aspect, pushed through it and negotiated through the stab wound on the medial side (Figs 2 and 4). At this stage it can be seen that the skin around the second wire is under tension.

5. By pulling the proximal wire 1 to 1.5 cm distally, the skin is restored to its normal position. The tendon also is being pulled distally (Fig. 3).

6. Dressings are applied around the four stab wounds and the two wires are held approximated (Figs 3 and 5) while an assistant applies a strip of plaster around each side of the two wires (Fig. 6). Corks are applied to the tips of the wires and a below-knee equinus cast incorporating the wires is applied. More recently, specially designed clamps have been applied to maintain the wires in the approximated position. These clamps have two advantages: it is easier to approximate the two wires, and only a front slab of plaster is needed (Fig. 7).

**Postoperative management.** The patient is mobilised using crutches and taking no weight through the affected leg. Three to four weeks after the operation the plaster and wires are removed. A new plaster, with the foot still in equinus, is applied and this is retained for a further four to five weeks. The heel of this plaster is built up so that the patient can take full weight.

When, after a total of about eight weeks, the plaster is finally removed, the heels of both shoes are built up 2.5 cm. The patient is encouraged to mobilise the ankle gradually and to exercise the calf muscle.

**PATIENTS**

Between May 1981 and September 1983 33 patients with closed complete ruptures of the calcaneal tendon were admitted to Bradford Royal Infirmary. Twelve of the patients were women whose mean age was 46 years (range 30 to 71); the 21 men also had a mean age of 46 years (range 21 to 63). All except four of the patients were treated by the author within three days of injury. The four exceptions had been misdiagnosed by their own doctors; one was not treated until four days after the rupture, two were treated at 15 days and one at 21 days.

In hospital the diagnosis was made on clinical

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**Table II. Major and minor complications in reported series after open surgical repair**

<table>
<thead>
<tr>
<th>Author(s)</th>
<th>Number of cases</th>
<th>Major complications</th>
<th>Minor complications</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Wound infection</td>
<td>Skin slough</td>
</tr>
<tr>
<td>Arner and Lindholm 1959</td>
<td>92</td>
<td>3</td>
<td>11</td>
</tr>
<tr>
<td>Percy and Conochie 1978</td>
<td>74</td>
<td>9</td>
<td>7</td>
</tr>
<tr>
<td>Nistor 1981</td>
<td>45</td>
<td>2</td>
<td>—</td>
</tr>
<tr>
<td>TOTAL</td>
<td>211</td>
<td>14 (7)</td>
<td>18 (8)</td>
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</tbody>
</table>

Figures in brackets are percentages of the total.
grounds. None of the patients could stand on tiptoe and in all a gap could be felt between the ruptured tendon ends. Every patient had a positive Simmonds test, with no movement of the foot when the calf was squeezed. The rupture was always 2 to 5 cm above the calcaneus.

Sporting activities were responsible for the injuries in 16 patients. The mechanism seemed to be a push-off type of injury in 15 patients, a landing injury in 6, a pot-hole injury in 3 and direct trauma in 3. In six patients there was no evidence of indirect or direct trauma—the rupture appeared to be truly spontaneous; they gave no history of pain or discomfort in the region of the calcaneal tendon before the rupture, but two were receiving systemic steroids for bronchial asthma. Four of the patients with a history of injury also had been on systemic steroids.

The non-dominant side was injured in 19 patients, (18 left and 1 right) whilst the dominant side was injured in 14 patients (12 right and 2 left).

Three patients participated in competitive sports; two were at maximal fitness and one was out of training. Thirteen were regular amateurs in various sports and all these were out of training at the time of rupture. The remaining patients ruptured during ordinary daily activities; the two oldest each had a limp before the rupture.

Assessment. All the patients were evaluated at three-monthly intervals up to one year, then annually. The mean follow-up period was 2.4 years (range nine months to four years). The state of the skin was noted and the tendon palpated; the gait pattern and the ability to stand on tiptoe were observed. The study included measurement of the range of movement, the circumference of the calf, the width of the tendon and the power of plantarflexion as compared with the uninjured side. The range of movement was measured radiographically using the angles subtended by the long axis of the tibia and the calcaneus; three lateral radiographs of the ankle were taken with the foot in neutral position, in full plantarflexion and in full dorsiflexion respectively.

Plantarflexion power was measured with a specially constructed dynamometer, with an adjustable footplate to accommodate different sizes of foot. The patient was in the sitting-position with the knee at a right angle and straps around the thigh, calf and leg (Fig. 8). The ankle was aligned at 15° of dorsiflexion when the ball of the foot was resting on the footplate. The peak torque and power were measured; the patient was allowed to familiarise himself with the dynamometer, then the test...
was repeated three times for each leg and the highest score was used. Twenty-eight normal individuals with no known injury to their lower limbs were measured to establish the power difference between the dominant and non-dominant leg: the dominant was stronger by a mean of 5%, using the paired t-test.

The patients were asked about their sporting activities before and after treatment and whether they had any complaints; they were also asked to evaluate their own results as excellent, good, fair or poor.

Objectively the results were graded as follows.

**Excellent.** The patient had no symptoms and had returned to normal activities including sport; there was no more than 5% loss or increase in the range of movement; the power of the injured leg was within 5% of normal.

**Good.** The patient had no symptoms and had returned to normal activities excluding sport; there was no more than 10% loss in the range of movement; loss of power was less than 15%.

**Fair.** The patient had minor complaints, but had returned to normal activities excluding sport; there was more than 10% loss of the range of movement and more than 15% loss of power.

**Poor.** The patient had a significant complaint, or infection, or marked loss of movement and power, or a limp, or had re-ruptured.

### RESULTS

All the patients except one had returned to their original level of daily activities. The three patients who had engaged in competitive sport before injury all returned to competition at between seven and nine months from the date of injury.

No patient has so far re-ruptured and skin problems have been trivial—one patient had a small granuloma on the lateral aspect of the lower calf four weeks after fixation, but four weeks later this had healed uneventfully. There was no evidence of pin-track infection, skin slough or adhesions between the tendon and the skin. Numbness along the distribution of the sural nerve was observed in two patients: one recovered completely after about six months; the other did not recover and felt minimal discomfort, but he returned to his original activities.

Thickening of the tendon at the site of the rupture was noted in all patients; it was 5 to 9 mm thicker than on the uninjured side. In five patients the tendon at three months from operation felt hard where the Kirschner wires had been; in all five the consistency had returned to normal when the patients were assessed at one year. The circumference of the calf was reduced by an average of 1.2 cm (range 0 to 2 cm). In many patients the medial head of gastrocnemius looked flattened (Fig. 9).

**Gait.** Most of the patients walked normally and could stand on tiptoe at six months. At nine months all the patients but one could stand on tiptoe. This patient, a woman aged 60 on systemic steroids, had been kept in plaster for three months as the tendon did not seem sufficiently healed at two; she developed Sudeck’s atrophy, continued to limp and was unable to stand on tiptoe at one year. All the other patients, apart from two who had limped for other reasons before rupture, could walk normally at one year.

**Range of movement.** A full range of movement was regained in 11 patients. A change of 5% or less was found in 20 patients (Figs 10 and 11); in 9 there was a decrease in plantarflexion and in 11 a decrease in dorsiflexion. In two patients the change in the range of movement was more than 10%. One woman aged 33 lacked 10° of dorsiflexion and 5° of plantarflexion. The patient with Sudeck’s atrophy lacked 15° of dorsiflexion and 25° of plantarflexion. It is important to note that none of the patients had an increased range of movement.

**Plantarflexion power.** In 25 patients the power was measured at one year, using the uninjured side as a control and taking into account the fact that the dominant

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**Fig. 9**
A typical patient six months after operation. Figure 9—Standing on tip-toe; note the flattening of the medial head of gastrocnemius.

**Fig. 10**
Range of movement of the uninjured side.

**Fig. 11**
Range of movement on the injured side.
limb was stronger than the non-dominant by an average of 5%. Normal power was regained in three patients. The remaining 22 patients had less power on the injured side: in 19 patients power was reduced by 5% to 10%; two patients lost 13% and 15% respectively; and in the patient who developed Sudeck’s atrophy, the injured side was 50% weaker than the uninjured side.

**Table III. Results in present series**

<table>
<thead>
<tr>
<th></th>
<th>Objective</th>
<th>Subjective</th>
</tr>
</thead>
<tbody>
<tr>
<td>Excellent</td>
<td>30</td>
<td>31</td>
</tr>
<tr>
<td>Good</td>
<td>—</td>
<td>1</td>
</tr>
<tr>
<td>Fair</td>
<td>2</td>
<td>—</td>
</tr>
<tr>
<td>Poor</td>
<td>1</td>
<td>1</td>
</tr>
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</table>

**Subjective assessment.** Table III shows the overall results. Subjectively 31 of the 33 patients considered the result excellent. One patient, a woman aged 33, considered her result was merely good because of stiffness of the ankle and shoe problems; she had, however, returned to her original sport. The woman of 60 on systemic steroids considered her result poor because of limitation of movement and residual swelling at the end of the day as a result of Sudeck’s atrophy.

**DISCUSSION**

Surgeons are divided as to what constitutes the best treatment for subcutaneous rupture of the calcaneal tendon. Nistor (1981) in his randomised study of 105 patients favoured conservative treatment despite an 8% re-rupture rate, an increased range of dorsiflexion of more than 10° in 23% of patients, and as much as 50% loss of power in 7% of his 60 patients treated conservatively. Of his 45 patients treated surgically 4% had infection and 4% re-ruptured; 20% had a sural nerve injury and in 44% the site of the tendon repair was tethered to the overlying skin (this seldom restricts movement seriously).

Deep sepsis, the prevalence of which has been reported to be as high as 9 out of 74 cases (Percy and Conochie 1978), is a serious problem leading to sloughing of the skin over the site of the repair; tendon healing is jeopardised with an increased incidence of re-rupture, while rehabilitation is delayed until the skin has healed.

Rupture of the calcaneal tendon nearly always occurs 3 to 5 cm above its insertion into the calcaneus. This site is ideal for external fixation. It is, however known to have a poor blood supply (Lagergren and Lindholm 1959), and this is associated with histopathological changes of the collagen fibres, decomposition and decreased staining of the cell nuclei (Arner and Lindholm 1959). These degenerative changes explain the tendency to infection and the liability to re-rupture.

**Conclusion.** In the treatment of ruptures of the calcaneal tendon external fixation satisfies the demand for a technique associated with a low local complication rate. The results of 33 cases so treated suggest that this new technique combines the advantages of both surgical and non-surgical treatment.

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**REFERENCES**


