COMMON PERONEAL NERVE DYSFUNCTION
AFTER HIGH TIBIAL OSTEOTOMY

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We assessed 16 patients before and after high tibial osteotomy by electrophysiological recordings, creatine phosphokinase levels, radiographs and intracompartmental pressure monitoring. We found mild electrophysiological abnormalities pre-operatively in 12 of the 16 patients, but postoperatively these had deteriorated in 11 of the 14 patients studied. Creatine phosphokinase levels, compartment pressure and radiological deformity were not predictive of the development of postoperative common peroneal nerve palsy.

Patients who also had a proximal fibular osteotomy had greater electrical abnormalities postoperatively and two of them developed common peroneal palsy. Proximal fibular osteotomy appears to be a causative factor in the development of common peroneal nerve palsy; more work is needed on the blood supply of the nerve.

High tibial osteotomy for the treatment of arthritic deformity of the knee was first reported in the English literature by Jackson in 1958. The operation has evolved since then because of the incidence of non-union and poor function after procedures at or below the level of the tibial tubercle (Jackson and Waugh 1974). Laterally based wedge osteotomy above the tubercle is now recommended, and non-union and infection are more common when internal or external fixation is used.

The cause of postoperative weakness of foot dorsiflexion and common peroneal palsy has not been clearly established. It has been ascribed to overnight plasters, ischaemic damage from high compartment pressure or damage to the anterior tibial artery. We have tried to elucidate the nature of this complication.

PATIENTS AND METHODS
We studied 16 patients having high tibial osteotomy in the Leicester General Hospital between January 1986 and July 1988. Long-leg weight-bearing radiographs were taken pre-operatively and an electrophysiological study was performed 24 hours pre-operatively and repeated 6 to 12 weeks later to quantify nerve and muscle function. A creatine phosphokinase (CPK) sample was taken just before operation and at 6, 12, 24, and 48 hours postoperatively.

All the operations were performed by one surgeon (RC) using a modified Gariépy procedure (1964). Proximal fibular osteotomy was required in nine patients. Slit catheters were placed in the anterior and deep posterior compartments for the continuous monitoring of pressures (Barnes et al 1985). The osteotomy site was immobilised solely by a long leg plaster. Patients were monitored closely in the early postoperative period for signs of compartment syndrome or common peroneal nerve palsy. Postoperative radiographs were taken to determine the tibiofemoral angle. Electrophysiological studies. A Medelec Mystro apparatus was used with standard recording techniques (Lenman and Ritchie 1970). Motor nerve conduction velocities (NCV) of the tibial nerve and of the common peroneal nerve (CPN) were calculated from the measured latency differences. Muscle compound potential amplitudes were also measured; tibial NCV was used as each patient’s control. Sensory NCVs and sensory nerve action potentials were assessed in the sural and superficial peroneal nerves. Electromyographic recordings were obtained from tibialis anterior, extensor hallucis longus, peroneus longus and gastrocnemius.

Each of these measurements was graded on a 4-point scale (0 being normal, 3 most abnormal) and total scores were calculated for the tibial and common peroneal nerves and, separately, for the deep and superficial branches of the common peroneal nerve. Statistical
analysis was carried out using non-parametric methods: binomial, Mann-Whitney and Spearman's rank correlation. The levels of significance and standard errors are given for two-tail tests.

RESULTS

Of the 16 patients, 12 were men and four women. Their ages ranged from 28 to 83 years (mean 64) though only one was under 50. The indications for surgery were severe osteoarthritis in 15 and marked genu varum with knee pain in one patient. Ten osteotomies were performed on the left side and six on the right. The pre-operative tibiofemoral angle varied from 3° to 12° of varus (mean 6.1). Postoperative angles were from 0° to 4° of valgus (mean 2.3). Tourniquet time was 15 to 44 minutes (mean 25). Fibular head or neck osteotomy was performed in nine patients (Table I).

**Creatine phosphokinase.** Pre-operative levels were available for 15 patients, being normal in 13 and raised in two (range 45 to 344 IU/L; mean 103). Postoperative values were available for the same 15 patients. Two patients with normal pre-operative values had gross elevation after operation. Of the two patients with abnormal pre-operative levels, one had no change after operation (248 to 259 IU/L) and the other had a large elevation from 344 to 641 units. Patients 9 and 16, who developed common peroneal nerve palsies after operation, both had normal CPK levels before and after operation (Table I).

**Compartment pressure.** Compartment pressures were measured in 13 patients. Of these, five showed significantly raised pressures (Gibson et al 1986). Patients 9 and 16, who both developed common peroneal palsy, had peak pressures of 50 mmHg and 20 mmHg respectively. The highest pressures recorded were in case 5 (60 mmHg) and case 6 (70 mmHg), but neither suffered electrophysiological or clinical deterioration.

**Electrophysiology.** All 16 patients had the pre-operative study and 14 the postoperative. One patient died of unrelated cardiac failure and one refused the second study. The mean interval between the two tests was 63 days (range 44 to 82).

Only four of the 16 patients had entirely normal pre-operative findings (Table I). However, the abnormalities were minor and included subclinical L5-S1 radiculopathy in five patients and mild peripheral nerve dysfunction in four. In three patients the findings were consistent with either a proximal or distal lesion.

The mean score in the tibial nerve territory was 1.5 ± 0.4 pre-operatively and 1.9 ± 0.45 postoperatively. The common peroneal nerve mean score pre-operatively was 4.1 ± 0.8 rising to 7.5 ± 1.55 postoperatively. This increase was consistent in 11 patients, although it just failed to reach statistical significance.

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**Table I.** Details of operation and results in 16 patients having high tibial osteotomy

<table>
<thead>
<tr>
<th>Case</th>
<th>Sex</th>
<th>Side</th>
<th>Age (yr)</th>
<th>Pre-op varus</th>
<th>Post-op varus</th>
<th>CPN score*</th>
<th>Anterior compartment pressure (mmHg)</th>
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<td>L</td>
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<td>4</td>
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<tr>
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<td>R</td>
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<td>16†</td>
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<td>L</td>
<td>60</td>
<td>5</td>
<td>3</td>
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</table>

* score for deficiency in common peroneal nerve function
† developed clinical common peroneal nerve palsy
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... (p < 0.06). There was no correlation between electrophysiological abnormalities and anterior compartment pressure, CPK level, patient age or tibiofemoral angle.

The mean common peroneal nerve score in the nine patients having a proximal fibular osteotomy was 3.0 ± 0.87 pre-operatively and 8.8 ± 2.17 postoperatively. This increase in common peroneal dysfunction (Fig. 1) was significant (p < 0.05). In the other patients, without fibular osteotomy, the levels were 5.5 ± 1.61 pre-operatively and 5.2 ± 1.65 postoperatively. Both patients who developed common peroneal nerve palsies had proximal fibular osteotomies.

DISCUSSION

Complications after high tibial osteotomy include non-union, infection, recurrent deformity and pain, compartment syndrome and common peroneal nerve lesions. Of these, weakness of foot dorsiflexion has been described as “the most puzzling and potentially serious symptom after tibial osteotomy” by Jackson and Waugh (1974). Its aetiology remains obscure though tight plaster (Coventry 1965) or tight bandages (Devas 1969) have been blamed. Jackson and Waugh (1974) postulated damage to the anterior tibial artery because of its anatomical proximity to the site of operation.

Clinically there is delayed onset of weakness of foot dorsiflexion with or without superficial peroneal sensory changes and pain on the dorsum of the foot. Delay in the onset of symptoms and signs has also been reported in 11 of 27 patients with this syndrome after blunt, penetrating or operative trauma at the knee (Berry and Richardson 1976; Stewart 1987); it was ascribed to nerve compression by either oedema or haemorrhage. The complication has also been described after total knee arthroplasty (Kaushal et al 1976; Rose et al 1982).

We found interesting electrophysiological evidence of pre-operative common peroneal nerve dysfunction in a high proportion of our patients. Spinal nerve root degeneration at L5 and S1 and the vulnerability of the CPN at the neck of the fibula are largely responsible for these findings (Berry and Richardson 1976; Stewart 1987). However, these abnormalities were not present in either of the two patients who developed postoperative palsies (CPN scores were 0 and 1 pre-operatively). This points to an intra-operative or postoperative cause.

We have previously investigated the relationship of raised compartment pressure to this complication (Gibson et al 1986). We proved a tendency to high pressures after osteotomy – but the two patients who developed the complication both had normal pressures. Our new results support our previous findings since only one of the two affected patients had raised pressures. Both of our affected patients had normal common peroneal nerve function for at least three days postoperatively (we documented normal sensation and extensor hallucis longus power). At this time, compartment pressure is returning to normal values (Gibson et al 1986) so this is further evidence against compartment syndrome as a cause.

Tourniquet-induced ischaemia has been linked to the occurrence of postoperative weakness of foot dorsiflexion (Gitlitz 1965; Patman 1975; Qvarfordt et al 1983). Prolonged tourniquet times may cause ischaemic damage, but in our series the mean tourniquet time was 25 minutes, and in the patients who developed the complication the times were 17 and 20 minutes. Prophylactic release of the common peroneal nerve during total knee arthroplasty in patients with severe flexion deformity to avoid traction injury, did not alter the incidence of postoperative nerve palsies (Rose et al 1982). This suggests that traction alone is not the cause.

Different types of operation have been associated with markedly different incidences of CPN palsy. Jackson abandoned the use of a curved osteotomy below the tubercle when he found that this was associated with a 25% incidence of weakness of dorsiflexion (23 of 92 patients). In contrast, osteotomy above the tibial tubercle did not produce the complication in one of a series of 83 patients (Jackson and Waugh 1974), though other authors using this method have reported nerve complications (Coventry 1965; Vainionpää et al 1981). The cause of this variation in incidence with surgical technique is unclear but intriguing. We found that patients who had proximal fibular osteotomy had significantly worse.

![Common peroneal nerve scores before (A) and after tibial osteotomy (B), grouped according to the use of fibular division.](image-url)
common peroneal nerve function postoperatively; two such patients subsequently developed common peroneal nerve palsies.

Conclusions. We feel that proximal fibular division may be an important aetiological factor in common peroneal palsy after high tibial osteotomy. More work is needed on the blood supply of the common peroneal nerve and the possible influence on this of tibial and of proximal fibular division.

We wish to express our gratitude to the Leicester orthopaedic consultants for allowing us to use their patients in this trial.

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REFERENCES


