We made a prospective study of 208 patients with tibial fractures treated by reamed intramedullary nailing. Of these, 11 (5.3%) developed dysfunction of the peroneal nerve with no evidence of a compartment syndrome.

The patients with this complication were significantly younger (mean age 25.6 years) and most had closed fractures of the forced-varus type with relatively minor soft-tissue damage. The fibula was intact in three, fractured in the distal or middle third in seven, with only one fracture in the proximal third. Eight of the 11 patients showed a ‘dropped hallux’ syndrome, with weakness of extensor hallucis longus and numbness in the first web space, but no clinical involvement of extensor digitorum longus or tibialis anterior. This was confirmed by nerve-conduction studies in three of the eight patients.

There was good recovery of muscle function within three to four months in all cases, but after one year three patients still had some residual tightness of extensor hallucis longus, and two some numbness in the first web space. No patient required further treatment.

Patients and Methods

We studied prospectively 208 consecutive isolated tibial diaphyseal fractures during the first year after closed intramedullary nailing. The mean age of the patients was 32.1 years (13 to 76); there were 164 men and 44 women. A total of 187 fractures was closed and 21 open. All the patients had a careful preoperative assessment of their neurological status within 24 hours of admission by a consultant surgeon.

Neurological abnormality was defined as a persistent sensory or motor deficit for over 48 hours, and all such observations were recorded before and after operation by a research physiotherapist (EW). All the patients had routine monitoring of pressure in the anterior compartment using a slit catheter. The threshold for fasciotomy, irrespective of the neurological status, was an absolute pressure greater than 40 mmHg or a differential pressure, the diastolic minus that of the compartment, of less than 30 mmHg.

All the fractures were treated by a standard method of closed, reamed, intramedullary nailing within 48 hours of admission. Traction was through an os calcis pin or by taping the foot to the fracture table. No tourniquets were used, and the pressure in the anterior compartment was monitored before and for at least 24 hours after operation.

All patients were reviewed jointly by a consultant surgeon and the research physiotherapist two and six weeks after surgery and at regular intervals up to one year, with careful assessment of any residual neurological deficit at each visit.

We analysed risk factors for dysfunction of the peroneal nerve in relation to age, gender, the mechanism of injury, the location of tibial and fibular fractures, displacement,
configuration, and soft-tissue injury. To compare patients with palsy with the total group, we used Student’s t-test (equal variances not assumed) for continuous data and Fisher’s exact test for categorical data.

**Results**

Of the 208 patients, 11 (5.3%) developed dysfunction of the peroneal nerve with no evidence of a compartment syndrome, either clinically or from monitoring of pressure. Details of the group are shown in Table I, including the type of fracture and mechanism of injury.

The affected patients were significantly younger with a mean age of 25.6 years (18 to 33; Student’s t-test, p = 0.003). Two of the fractures had been sustained in road-traffic accidents and one was a grade-I open fracture as classified by Gustilo and Anderson. The other eight had minimal soft-tissue injuries of type C0 or C1 of Oestern and Tscherne.

Compared with those without nerve deficit, Fisher’s exact test was not significant. The fractures in the 11 patients were predominantly of the ‘bending’ forced-varus AO type A or B and were significantly different by the Fisher’s exact test (p = 0.025, odds ratio = 8.6, 95% CI 1.1 to 68.4). There was a proximal fibular fracture in only one patient (case 7) and in three cases the fibula was intact with no significant difference from the uninvolved patients by Fisher’s exact test. There were no significant differences in gender or location and displacement of the fracture.

Three patients (cases 1 to 3) showed neurological deficits before operation: one had dysfunction of both superficial and deep branches, one had involvement of the deep branch only and the third showed isolated paralysis of extensor hallucis longus, with loss of sensation in the first web space. None of the three patients had any evidence of compartment syndrome either before or after operation.

Of the eight patients who developed a neurological deficit after operation, one had deep peroneal palsy, and seven had a dropped great toe with sensory loss in the web space only. There had been no technical difficulties at operation; five had traction through the os calcis and three by taping to the fracture table. All eight nails had been locked distally, with proximal locking by a transverse screw in six and an anteroposterior locking screw in two.

All 11 patients had monitoring of compartment pressure for 48 to 72 hours after operation, with no evidence of increase. Despite this, persistence of neurological signs led to exploration of the anterior compartment in three patients (cases 1, 4 and 11). The compartment in each was not tight, showing minimal haematoma and bruising of the muscles of the anterior compartment around the site of the fracture.

* deficit present before operation
† extensor hallucis longus
Three other patients (cases 3, 6 and 8) had MRI of the calf after nailing. These confirmed fracture haematoma, with evidence of general muscle injury in the anterior compartment, not confined to the extensor hallucis longus, and similar changes in the lateral and posterior compartments.

After operation, patients with common or deep peroneal nerve palsies were provided with foot-drop splints and all were mobilised, fully weight-bearing, within three days. There was some recovery of extensor function during the first six weeks, with complete recovery of muscle power at ten to 18 weeks. Three patients (cases 5, 6 and 10) had some tightness of extensor hallucis longus after recovery of power, but without significant functional impairment. Sensory loss in the first web space recovered completely in seven patients by ten to 18 weeks and by 40 weeks in two (cases 4 and 8), but was incomplete at one year in two (cases 5 and 9).

Studies on nerve conduction in three patients with dropped hallux and delayed recovery (cases 3, 7 and 8) at four to six weeks showed denervation of extensor hallucis longus and extensor digitorum brevis but normal evoked potentials in the tibialis anterior, extensor digitorum longus, and the muscles of the lateral and posterior compartments. There were abnormal sensory potentials from the first web space.

Discussion

Dysfunction of the peroneal nerve after intramedullary nailing of the tibia is uncommon; most reviews either fail to refer to this complication or report a low incidence. More careful evaluation has shown an incidence of up to 30%, but about 90% of cases are due to transient neurapraxia. In one of these series, tibial nailing was delayed for an average of ten days after injury. Our patients had their nailing within 48 hours of injury, with an incidence of dysfunction of the peroneal nerve of 5.3%, which is similar to that reported for persistent palsies in other series.

Our patients with this complication were significantly younger, and usually had closed fractures of the forced-varus type, with relatively minor soft-tissue injury.

Three of our 11 patients had preoperative deficits caused by traction or contusion. As previously reported the preoperative nerve palsies have a good prognosis.

Palsies after intramedullary nailing are iatrogenic and therefore potentially preventable. Previous studies suggest that risk factors include the use of the ‘90/90’ position, calcaneal traction, reaming and the subclinical compartment syndrome; but minimising these risk factors does not abolish all neurological complications.

We found that seven of the eight patients with postoperative defects had complete sparing of tibialis anterior and extensor digitorum longus.

Dropped hallux has been described after high tibial osteotomy, but has rarely been reported in isolation after tibial fracture. The relatively high incidence (up to 12%) after high tibial osteotomy has been blamed on tight bandaging, the level of division of the fibula, the level of the tibial osteotomy, the use of external fixation devices, a compartment syndrome and damage to the anterior tibial artery. The common and deep peroneal nerves give multiple branches to the tibialis anterior and extensor digitorum longus below the neck of the fibula. The deep peroneal nerve is close to the fibula in the anterior compartment, but the branches to tibialis anterior and extensor digitorum longus are anterolateral and therefore at less risk during manipulation of the fibula. The extensor hallucis longus, which is smaller with a more distal origin, is usually innervated by a tortuous single branch which originates more distally.

This suggests that the dropped hallux syndrome is due to a lesion of the distal part of the deep peroneal nerve. This was supported by studies on nerve conduction in three of our patients.

The distal part of the deep peroneal nerve could be selectively injured by traction, by a subclinical compartment syndrome or during the insertion of a proximal transverse screw. Traction neurapraxia seems most likely in view of the early recovery, and the significantly greater proportion with a forced-varus fracture of the tibia and an intact or distal fracture of the fibula. It has been shown that compartment pressures may vary with distance from the fracture with peak pressures usually close to it. Although none of our patients had evidence of an acute compartment syndrome, it is possible that the more distal origin and innervation of extensor hallucis longus and the tortuous distal course of the deep peroneal nerve put these structures at greater risk to the pressure effects of hyperextension of the anterior compartment. The residual tightness of this muscle in a few of our patients also suggests the possibility of a subclinical compartment syndrome not detected by monitoring.

The diagnosis of dropped hallux is clinical, although we have identified some preoperative risk factors. Monitoring of the anterior compartment, using a silt catheter close to the fracture site, is mandatory for patients who develop the syndrome. In the presence of normal pressures, exploration of the compartment is not, at present, justified. More work is required on reducing operative risk factors, and on the anatomy and neurophysiology of the deep peroneal nerve in the anterior compartment.

No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

References


