DISABILITIES AFTER TIBIAL SHAFT FRACTURES

With Special Reference to Volkman’s Ischaemic Contracture

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In this paper I present the results of an investigation into the incidence, etiology and types of disability following tibial shaft fractures. The study has been prompted by the surprising scarcity of published reports on the functional prognosis after this type of injury. My observations indicate that the severity of the initial trauma is an important determinant of eventual function, and suggest that soft-tissue scarring, either from direct tissue damage or from ischaemic contracture, accounts for a considerable proportion of the bad results after fractures of the tibial shaft.

CLINICAL MATERIAL AND OBSERVATIONS

The series reported here comprises 343 soundly united tibial shaft fractures, occurring in 336 adult patients (over the age of sixteen) treated at the Royal Infirmary, Sheffield, in the six years 1948 to 1953. The duration of follow-up was from one to six years after bone union had been achieved.

Two hundred and twenty-eight patients (235 fractures) were personally examined and seventy-four others replied to a detailed questionnaire. Thirty-four patients had died or could not be traced directly; these were assessed from their hospital records, supplemented, when possible, by information from their family doctors or from other hospitals.

The results of this investigation were as follows. Two hundred and ninety-five limbs (86 per cent) had excellent functional and anatomical results. Nineteen limbs (5.5 per cent) had half an inch to three-quarters of an inch of shortening (measured radiographically). This resulted in no more than a mild "dipping" gait, often unnoticed by the patient. Eight limbs (2.5 per cent) had limitation of full knee flexion. This caused inconvenience in kneeling tasks, but was not otherwise disabling. Twenty-one limbs (6 per cent) had limitation of ankle movement or foot movement, or both. This was invariably a source of disability, with pain in the foot and leg on walking and impairment of such activities as running, dancing, and walking on irregular surfaces. Of eleven patients in this group who were engaged in heavy industry before injury, only two had been able to return to their former employment.

No example of significant varus or valgus deformity occurred in this series. Posterior angulation, occurring in three patients, was symptomless.

INVESTIGATION OF POSSIBLE CAUSES OF POST-TRAUMATIC JOINT STIFFNESS

Injury to the affected joint—There were eight patients in the whole series with associated fractures or soft-tissue injuries involving the knee joint, and these were the only patients found to have persistent limitation of knee movement. In contrast, ankle injuries complicated only two of the fractures that were followed by ankle stiffness. Moreover, a further eighteen patients with associated malleolar fractures regained full movement.

Severity of initial injury—Table I demonstrates the incidence of joint stiffness in fractures graded into minor, moderate and major severity of injury according to a simple classification previously described (Ellis 1958). The incidence of ankle and foot stiffness in these three grades

* Being the substance of part of the work accepted as a thesis for the degree of M.Ch. (Oxon).
was 1 per cent, 5 per cent and 22 per cent respectively. There was no correlation between knee stiffness and severity of injury; the condylar fractures involving the knee joint were all of moderate severity only.

**TABLE I**

**INCIDENCE OF JOINT STIFFNESS RELATED TO SEVERITY OF INJURY**

<table>
<thead>
<tr>
<th>Severity of injury</th>
<th>Number of fractures</th>
<th>Cases of knee stiffness</th>
<th>Cases of ankle and/or foot stiffness</th>
</tr>
</thead>
<tbody>
<tr>
<td>Minor</td>
<td>98</td>
<td>—</td>
<td>1 (1%)</td>
</tr>
<tr>
<td>Moderate</td>
<td>200</td>
<td>7 (4%)</td>
<td>10 (5%)</td>
</tr>
<tr>
<td>Major</td>
<td>45</td>
<td>1 (2%)</td>
<td>10 (22%)</td>
</tr>
<tr>
<td></td>
<td>343</td>
<td>8 (2%)</td>
<td>21 (6%)</td>
</tr>
</tbody>
</table>

**Method of treatment**—Table II shows the incidence of ankle and foot stiffness in 222 fractures treated by immobilisation alone, in 112 fractures treated by calcaneal traction and plaster immobilisation, and in nine treated by screw fixation. The fractures in each group have been subdivided according to the severity of the initial injury.

**TABLE II**

**INCIDENCE OF ANKLE AND FOOT STIFFNESS RELATED TO THE METHOD OF TREATMENT USED**

<table>
<thead>
<tr>
<th>Severity of injury</th>
<th>Method of treatment</th>
<th>Number of fractures</th>
<th>Incidence of ankle and/or foot stiffness</th>
</tr>
</thead>
<tbody>
<tr>
<td>Minor</td>
<td>Immobilisation</td>
<td>98</td>
<td>1 (1%)</td>
</tr>
<tr>
<td></td>
<td>Traction</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>Screw fixation</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Moderate</td>
<td>Immobilisation</td>
<td>104</td>
<td>6 (6%)</td>
</tr>
<tr>
<td></td>
<td>Traction</td>
<td>88</td>
<td>4 (5%)</td>
</tr>
<tr>
<td></td>
<td>Screw fixation</td>
<td>8</td>
<td>—</td>
</tr>
<tr>
<td>Major</td>
<td>Immobilisation</td>
<td>20</td>
<td>3 (15%)</td>
</tr>
<tr>
<td></td>
<td>Traction</td>
<td>24</td>
<td>6 (25%)</td>
</tr>
<tr>
<td></td>
<td>Screw fixation</td>
<td>1</td>
<td>1 (100%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>343</td>
<td>21 (6%)</td>
</tr>
</tbody>
</table>

In the fractures caused by moderate violence the incidence of joint stiffness was the same whether or not traction had been employed, but in those caused by severe violence the incidence of joint stiffness was higher in the group treated by traction (25 per cent compared with 15 per cent). However, two very severe injuries are included in the fractures treated by traction, whereas there was no case of such severity in the major fractures treated by immobilisation: in both cases it was feared at first that immediate amputation might be required, but both limbs survived, eventually uniting after many months of immobilisation, and represented two of the poorest functional results in the study.

The figures for fractures treated by screw fixation are too small to be of significance; it is of interest, however, that the only patient with a poor functional result in this group sustained the one fracture of major violence treated by this method.
Infection—Five of the 105 compound fractures became infected. One of these was followed by persistent ankle and foot stiffness but this was in a fracture of major severity. Two of the 112 fractures treated by calcaneal traction were complicated by infection of the pin track, but both patients regained full joint movement.

Infection did not, therefore, account for the high incidence of persistent joint stiffness in fractures of major severity in this series.

Ischaemic contracture—Of the 225 injured limbs that were re-examined, nine were found to have one or more of the following contractures: clawing of the toes, seven examples; hallux flexus, six examples; equinus deformity, three examples. Brief details of these nine patients are given in Table III, and typical examples are illustrated in Figures 1 to 3. These contractures varied in severity from mild clawing of the toes to disabling deformities which resembled the descriptions of fully established ischaemic contractures of the lower limb (due to a variety of causes) reported by Burdzik (1953), Felländer (1949), Horwitz (1940), Jones and Cotton (1935), Miller, Markin and Grossman (1952), and Salembier (1954). Seven of these nine limbs had severe limitation of ankle and foot movement and comprised a third of the cases in the series with this disability.

Six of the nine contractures followed major fractures and one other patient developed progressive contractures after two successive moderate fractures of the same leg. Six of the fractures were treated by calcaneal traction, one by internal fixation and one by plaster immobilisation alone. The patient with two successive fractures had had the first treated by traction, the second conservatively.

ARTERIOGRAPHIC STUDIES IN THE ACUTE PHASE OF TIBIAL FRACTURES

Because of the occurrence of these contractures after fractures of the tibial shaft, it was decided to study the vascular pattern in the injured limb immediately after the injury.
Percutaneous femoral arteriography was carried out in patients with freshly sustained tibial shaft fractures, using 20 millilitres of 50 per cent Pyosil. Rapid serial radiographs were taken, the plates being passed beneath the leg through a special cassette (Jones and Steiner 1949).

Although satisfactory arteriograms were obtained in only five cases, it is of interest that three of these showed definite disturbances of major arteries. Two such disturbances occurred in fractures of minor severity, were not followed by any clinical evidence of ischaemic change, and were presumably transient. These arteriograms are reproduced in Figures 4 and 5.

### TABLE III
**Details of Nine Cases of Ischaemic Contracture**

<table>
<thead>
<tr>
<th>Case number</th>
<th>Age of patient (years)</th>
<th>Site</th>
<th>Severity</th>
<th>Primary treatment</th>
<th>Time in plaster (weeks)</th>
<th>Ischaemic manifestations</th>
<th>Subsequent treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>16</td>
<td>Lowest third</td>
<td>Major (thrombosed posterior tibial artery)</td>
<td>Screw fixation; plaster</td>
<td>12</td>
<td>Wasting of calf muscles; equinus; limited extension of toes; mild hallux flexus</td>
<td>Lengthening of tendo calcaneus</td>
</tr>
<tr>
<td>2</td>
<td>41</td>
<td>Middle third</td>
<td>Major</td>
<td>Calcaneal traction; plaster</td>
<td>12</td>
<td>Stiff subtalar joint and forefoot; hallux flexus; clawed toes</td>
<td>—</td>
</tr>
<tr>
<td>3</td>
<td>59 (second fracture a year later)</td>
<td>Uppermost third</td>
<td>Moderate</td>
<td>Calcaneal traction; plaster</td>
<td>11</td>
<td>Hallux flexus</td>
<td>Keller's operation</td>
</tr>
<tr>
<td>(b)</td>
<td></td>
<td>Lowest third</td>
<td>Moderate</td>
<td>Plaster alone</td>
<td>13</td>
<td>Stiff, cold, equinus foot; clawed toes</td>
<td>Foot fusion; later below-knee amputation</td>
</tr>
<tr>
<td>4</td>
<td>20</td>
<td>Middle third</td>
<td>Major</td>
<td>Calcaneal traction; plaster</td>
<td>14</td>
<td>Clawed toes</td>
<td>—</td>
</tr>
<tr>
<td>5</td>
<td>35</td>
<td>Middle third</td>
<td>Moderate</td>
<td>Calcaneal traction; plaster</td>
<td>14</td>
<td>Limited ankle extension; hallux flexus</td>
<td>—</td>
</tr>
<tr>
<td>6</td>
<td>37</td>
<td>Middle third</td>
<td>Moderate</td>
<td>Plaster alone</td>
<td>30</td>
<td>Equinus deformity; stiff subtalar and forefoot joints; clawed toes</td>
<td>—</td>
</tr>
<tr>
<td>7</td>
<td>32</td>
<td>Lowest third</td>
<td>Major</td>
<td>Calcaneal traction; plaster</td>
<td>18 then further 22 after refraction</td>
<td>Stiff ankle; subtalar and forefoot joints; hallux flexus; foot blue and swollen</td>
<td>Keller's operation</td>
</tr>
<tr>
<td>8</td>
<td>36</td>
<td>Middle third</td>
<td>Major (posterior tibial vessels ligated)</td>
<td>Calcaneal traction; plaster</td>
<td>34</td>
<td>Wasting of calf muscles; stiff ankle, subtalar and forefoot joints; hallux flexus; clawed toes; leg becomes cyanosed when dependent</td>
<td>Hallux fusion</td>
</tr>
<tr>
<td>9</td>
<td>47</td>
<td>Middle third</td>
<td>Major</td>
<td>Calcaneal traction; plaster</td>
<td>23</td>
<td>Clawed toes</td>
<td>—</td>
</tr>
</tbody>
</table>

The third example was in a compound fracture caused by major violence; here arteriography demonstrated virtually complete obliteration of the vascular tree below the popliteal artery (Fig. 6). Exploration revealed a complete tear of the anterior tibial artery and subsequently the patient developed typical ischaemic contractures, with clawing of the toes, a plantarflexed ankle and a stiff foot (Fig. 7). The fracture in this case was treated by immobilisation in a heavily padded, widely split plaster.
ARTERIAL DAMAGE RESULTING IN GANGRENE

During the period under review six tibial shaft fractures necessitated amputation for gangrene. Four of these followed gross injuries with extensive arterial damage, but two followed relatively minor fractures. In one, the posterior tibial artery was torn midway between a minor, midshaft tibial fracture and an associated dislocation of the knee. The other was of particular interest in that, although the fracture was a closed, midshaft fracture with an intact fibula, the posterior tibial artery was found at exploration to be ruptured at the level of the fracture.

One may sum up this study of vascular damage and ischaemic contracture in tibial fractures as follows. 1) Nine patients had clinical features of ischaemic contracture and seven of these had severe ankle and foot stiffness, accounting for one-third of the total examples of this disability in the present study. 2) Arteriography in acute fractures confirmed that vascular trauma at the initial injury may lead to contractures and associated foot and ankle stiffness. Moreover, it demonstrated that fractures of relatively minor severity may be associated with at least transient arterial disturbance. 3) Consideration of the cases of gangrene in this series showed, again, that serious arterial damage may accompany relatively minor fractures.
DISCUSSION

Anatomical deformity after a tibial shaft fracture is clearly a consequence of inability to reduce the fracture or to hold it reduced until sound union has occurred. In contrast, the etiology of persistent joint stiffness, a far more potent cause of disability, is less easily explained. Some examples are accounted for by direct damage to the joint itself at the time of initial injury, a factor that explained all the cases of limited knee movement in this study. Others may follow severe infection of a wound involving the joint, although there was no example of this in the series.

The part played by prolonged immobilisation in the causation of persistent limitation of joint movement is still debated. Hugh Owen Thomas and Robert Jones both stressed that immobilisation of an undamaged limb does not result in persistent stiffness, provided the joint is not kept in a position of strain, and the experimental investigations of Scaglioni and Casuccio (1936) support this view. However, in the case of an injured limb it has been suggested that prolonged immobilisation allows the organisation of traumatic exudate into firm adhesions, with resultant persistent stiffness. There is no available reported study in which the incidence of joint stiffness after tibial fracture treated by internal fixation and immediate mobilisation have been contrasted with exactly comparable cases kept immobilised until union has been achieved. Until such an investigation has been carried out it is impossible to say whether immobilisation of the injured limb, in itself, causes persistent disability, or whether any correlation between prolonged immobilisation and joint stiffness is accounted for entirely by the severity of the injury, a factor which is associated both with the incidence of joint stiffness and with delay in fracture healing (Ellis 1958).

It has been stated that damage to joints, with consequent stiffness, may occur after capsular strain or pin-track infection when skeletal traction has been employed (Wardle 1933, Jones 1936). In the present investigation there was no evidence that skeletal traction was responsible for persistent knee stiffness, because this occurred in only one patient of the 112 treated by this method. Moreover, in this solitary case there had been associated direct injury to the knee. When the incidence of ankle and foot stiffness was considered, a comparison of fractures of similar extent of injury treated by immobilisation alone and by calcaneal traction revealed little difference between comparable cases in the two groups. In contrast, there was a marked increase in the incidence of persistent joint stiffness after fractures of increasing severity of injury in both series of cases (Table II).

It has been suggested that joint stiffness after a fracture may result from associated soft-tissue injury with subsequent scarring and adhesion formation (Doran 1944, Nicoll 1941, Smillie 1951). This is supported by our findings that the incidence of joint stiffness can be correlated with the severity of the initial trauma.
I believe these soft-tissue injuries to be of two types: the direct injury, which follows severe destructive violence to the tissues, with subsequent scarring; and the indirect injury resulting from vascular damage with subsequent ischaemic changes.

In the present study no less than a third of the patients with persistent ankle and foot stiffness had clinical evidence of ischaemic contractures. The rarity of previous reports of contractures after tibial fractures, compared with the numbers found in this series, suggests that this complication has frequently been overlooked in the past.

The vascular damage in association with tibial fractures may occur in a number of ways. There may be direct injury to major arteries (Fig. 6), or the vessels may go into spasm (Cohen 1944). Such spasm, if it persists for several hours, may result in irreversible changes in the muscles (Harman 1947). Even if the spasm is then relieved, and the arterial supply of the limb appears to be normal again, permanent damage may already have taken place. Should the main arteries of the limb escape injury or spasm there may still be damage to small peripheral vessels. Clark (1945) demonstrated that the vascular supply to muscles is by an end-artery system and local vascular trauma may, therefore, be followed by areas of muscle infarction—an occurrence which has been confirmed in crush injuries by Bowden and Gutmann (1949). Muscle ischaemia may not only follow discrete arterial injury; there is also evidence that infarction and ischaemic changes may result from venous damage (Adams, Denny-Brown and Pearson 1953; Brooks 1922, Middleton 1930).

What of the occasional cases of persistent joint stiffness that occur after relatively minor injuries? I suggest that unrecognised vascular damage or spasm may, on occasion, complicate even minor fractures, and the resulting muscle ischaemia may produce sufficient scarring to cause the persistent limitation of joint movement that follows a small proportion of these injuries. If fibrosis is extensive, frank contractures will be evident, but it is suggested that subclinical examples of the consequences of muscle ischaemia may occur—that is to say, scarring sufficient to limit movement but not to produce deformity.

On the basis of the present study, therefore, I advance the following hypothesis: that persistent joint stiffness after a fracture, with no evidence of direct damage to the affected joint, is caused by replacement fibrosis consequent upon soft-tissue trauma. This damage may result from direct injury, from the ischaemia of associated vascular lesions, or from a combination of both.

SUMMARY

1. A study of the late results of 343 soundly united tibial shaft fractures was carried out. Limitation of ankle and/or foot movement occurred in twenty-one patients (6 per cent) and was found to be the most important cause of disability. Knee stiffness (2.3 per cent of cases) and shortening of up to three-quarters of an inch (5.5 per cent of cases) caused little functional impairment.

2. Stiffness of the foot and ankle was correlated with the severity of injury, occurring in 1 per cent of minor, 5 per cent of moderate and 22 per cent of major injuries.

3. One-third of the patients with limitation of foot and ankle movement had clinical evidence of ischaemic contracture.

4. It is argued that, in the absence of direct injury to the joint, persistent joint stiffness is caused by replacement fibrosis of soft tissues. This may result either from direct tissue injury or from associated vascular damage.

I would like to thank Mr F. W. Holdsworth for permission and encouragement to investigate this group of patients, all of whom were under his care.
REFERENCES


