VOLKMANN'S ISCHAEMIA IN THE LOWER LIMB

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Although the ischaemic contracture that Volkmann described immediately suggests the familiar deformity of the forearm and hand, the first case he reported, in 1872, was of a contracture in the leg. The purpose of this paper is to review the features of fifteen cases of ischaemia in the leg following injuries or operations (the anterior tibial syndrome is excluded) that have come under the care of my colleagues and myself and to consider how important this complication may be and what might be done to prevent it. The treatment of the established condition will be discussed briefly.

**TABLE I**

<table>
<thead>
<tr>
<th>Site</th>
<th>Injury</th>
<th>Number of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Femur</td>
<td>Fracture, mid-shaft, open</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Fracture, lower third</td>
<td>1</td>
</tr>
<tr>
<td>Knee</td>
<td>Dislocation</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Fracture dislocation</td>
<td>2</td>
</tr>
<tr>
<td>Tibia</td>
<td>Fracture, shaft</td>
<td>4</td>
</tr>
<tr>
<td>(and fibula)</td>
<td>Bone shortening</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Osteotomy</td>
<td>1</td>
</tr>
<tr>
<td>Leg</td>
<td>Crushed</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Soft-tissue operation, knee and leg</td>
<td>1</td>
</tr>
</tbody>
</table>

**TABLE II**

<table>
<thead>
<tr>
<th>Category</th>
<th>Number of patients</th>
<th>Youngest</th>
<th>Oldest</th>
<th>Average age</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper limb, excluding intrinsic muscles of hand</td>
<td>52</td>
<td>2</td>
<td>37</td>
<td>12.4 m</td>
</tr>
<tr>
<td>Intrinsic muscles of hand</td>
<td>5</td>
<td>18</td>
<td>58</td>
<td>32.5 m</td>
</tr>
<tr>
<td>Lower limb</td>
<td>15</td>
<td>6</td>
<td>38</td>
<td>20</td>
</tr>
</tbody>
</table>

**Types of injury**—This collection of cases is heterogeneous (Table I); a variety of injuries of the thigh, knee and leg can give rise to this grave complication (Figs. 1 to 3). In three patients the ischaemia followed an operation on the tibia, one osteotomy to correct a deformity and two resections to shorten the bone. In another child it followed excision of haemangioma of the leg followed by confinement in plaster. In none of the three operations on bone was anything untoward noted at the time, and the exact cause of the vascular damage remains unknown. In none of the fifteen cases were the damaged vessels explored at a time when
intervention might have been effective. There were, happily, no cases of ischaemia in children with fractures of the femur treated by traction with the foot of the bed elevated (Thomson and Mahoney 1951) or by vertical suspension of the lower limbs—Bryant’s traction—although this has been described by Nicholson, Foster and Heath (1955) and by others before them. 

**Age**—Whereas Volkmann’s ischaemia in the upper limb usually occurs in children (apart from ischaemia confined to the intrinsic muscles of the hand, which affects only adults), in the lower limbs it is seen more often in adults (Table II).

**THE CONFIGURATION OF THE LESION AND THE CHANGES IN MUSCLE**

In the upper limb the mass of affected tissue usually takes the form of an ellipsoidal infarct, though there are great variations in the extent and in the intensity of the ischaemic damage. In all but one of the fifteen cases in the leg it was possible to determine the distribution of the ischaemia, and it will be noted that there is no discernible pattern (Figs. 4 and 5). All that emerges is the great vulnerability of the flexor hallucis longus and the frequent sparing of the gastrocnemius (Fig. 6).

The recognition of the shape of the lesion in the forearm (Seddon 1956) proved to be helpful in planning the treatment of established ischaemia. This has not been so in the leg.

What I have only recently appreciated is that the phenomenon of infarction—that is, circumscribed ischaemia of a mass of tissue due to a demonstrable arterial blockage—is not an invariable feature of the Volkmann lesion. It occurs when the arterial damage is relatively proximal and the reason why it attracted Tavernier, Dechaume and Pouzet’s (1936) attention and, later, mine, was that most of the patients they and I saw were children with supracondylar
fractures of the humerus. The vascular lesion was above the elbow, the infarct in the forearm. I am now doubtful whether in the handful of cases of Volkmann’s ischaemia due to fractures

![Chart](image)

**FIG. 4**
Chart, based on examination of the muscles of the leg in fifteen cases, showing the frequency of involvement of the various muscles.

![Diagram](image)

**FIG. 5**
Diagram showing vulnerability to ischaemia of muscles of the leg. The flexor hallucis longus is the most frequently damaged, but the anterior muscles, which are superficial, suffer considerably.

of the bones of the forearm the tissues affected would, if they could have been cleanly excised, have shown the regular form of an ellipsoid. In the leg an ellipsoidal infarct was exceptional because the vascular damage and the ischaemia occurred most often (in this small series) in

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the same segment of the limb—as in the forearm fractures. In the upper limb (Seddon 1964) twenty-nine out of forty-seven (62 per cent) single injuries—multiple injuries are excluded—were well proximal to the site of ischaemia. By contrast, in these fifteen cases of ischaemia of the leg, the arterial injury was in the thigh in only two, at the level of the knee in three;

![Image](image_url)

FIG. 6
Although all muscles below the knee, except the gastrocnemius, were paralysed in a patient who had suffered a dislocation of the joint, with injury to the popliteal artery, necrosis was limited to the belly of flexor hallucis longus just proximal to the ankle. This was excised when the calcaneal tendon was lengthened.

nine were in the leg and in one case the level was uncertain. An ellipsoidal infarct was identified in only three cases, all with proximally situated vascular lesions. It may be possible to explain the result of an arterial injury below the knee, as in the case illustrated in Figure 3, in which the ischaemia was largely confined to the distribution of the damaged anterior tibial artery; but this is unusual. When the leg itself is injured almost anything can happen. Thus,
there is far less scope than in the upper limb for basing the treatment of the muscle contracture and of any concomitant nerve lesion on the simple notion of a well defined infarct.

The various degrees of ischaemia of muscle do not call for detailed comment, but in all there is early and gross swelling of the muscle, which, if not relieved by prompt incision of the overlying deep fascia, especially in the anterior and lateral compartments of the leg, may rapidly complete the destruction of the muscle. Late calcification of necrotic muscle, which has been described many times, was seen in three cases (Fig. 7), but not the liquefaction and cyst formation described by Gallie and Thomson (1960) in ischaemia of the leg.

DEFORMITY

As in the upper limb the deformity is fairly well accounted for by the distribution of the ischaemic muscle damage. Involvement of the calf muscle, more particularly the soleus, explains the equinus; clawing of the toes is due to contracture of their long flexors; and inversion of the foot is produced by shortening of the tibialis posterior (Fig. 8). Limitation of plantar-flexion of the ankle may be produced by contracture of the tibialis anterior, but no significant deformity has been noted as a consequence of ischaemia of the peronei or the extensors of the toes.

IN Volvement OF NERVES

It has been established (Tavernier, Dechaume and Pouzet 1936; Holmes, Highet and Seddon 1944) that nerve involvement is due to the ischaemia itself, not to fibrotic muscle compressing the nerves. When it was possible to expose the posterior tibial nerve during exploration of the calf, or to examine all the nerves in amputation specimens, it was found that the damage was due to ischaemia (Fig. 9). It was particularly notable that the posterior tibial nerve showed no sign whatever of strangulation by scar tissue even when the surrounding long flexor muscles were extensively involved (Fig. 10).

When the vascular injury is caused by dislocation or fracture-dislocation of the knee the displacement may also damage the tibial and peroneal nerves by traction. It may then be

TABLE III

<table>
<thead>
<tr>
<th>Nerves Involved in Fourteen Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
<tr>
<td>-----------------------------------</td>
</tr>
<tr>
<td>Number</td>
</tr>
<tr>
<td>Recovery</td>
</tr>
<tr>
<td>Partial recovery</td>
</tr>
<tr>
<td>Recovering</td>
</tr>
<tr>
<td>No certain knowledge</td>
</tr>
</tbody>
</table>

Ischaemic nerve damage was present in twelve cases; in seven both nerves were involved; in two the peroneal alone; in three the tibial alone. There were also two cases in which both nerves were damaged by traction.

impossible, except by exploration, to decide whether the paralysis and deformity of the leg following a gross injury of the knee are due to ischaemia at all. Two of my patients with injuries
The characteristic equinovarus deformity. The line indicates the area of cutaneous sensory loss. This boy's leg had been crushed by a block of concrete and the incision was made to evacuate a large haematoma. He was subsequently found (Fig. 10) to have extensive necrosis of the soleus and the flexor hallucis longus.

Section of an ischaemic nerve, not subjected to compression by overlying fibrotic muscle. There is dense endoneural and perineural collagenisation.
at the knee presented identical clinical pictures: equinovarus deformity; paralysis of all muscles in the leg except the gastrocnemius, though that was weak; and profound disturbance of sensibility in the distribution of the peroneal and tibial nerves. They were investigated electrically—direct nerve stimulation, strength-duration curves of the accessible muscles and electromyography—and by arteriography. The results were inconclusive and it was not until the calf was explored that ischaemia of flexor hallucis longus was discovered in one case (Fig. 6), whereas in the other there was no trace of ischaemic damage in any muscle. Yet in the ischaemic case funicular biopsy of the peroneal nerve, carried out at the same time as the exploration of the calf, showed that that nerve had been damaged by traction, not by ischaemia. Thus in these uncommon injuries the diagnosis depends ultimately on exploration of the leg which has to be undertaken anyway for elongation of the shortened calcaneal tendon.

![Fig. 10](image)

**Excision of necrotic muscle from a boy's leg (see Fig. 8).** The calf muscle was turned proximally and its deep part, the soleus, was found to be necrotic. So was the flexor hallucis longus; the rake retractor is pulling the friable fibres apart. The ischaemic posterior tibial nerve lies beneath the instrument.

The frequency of nerve damage is depressingly high and, as is shown by Table III, the prognosis is poor. There is no place here for endeavouring to repair the posterior tibial nerve by grafting because, as has been shown by Clawson and Seddon (1960), the results even of suture of the posterior tibial nerve after ordinary injury are so indifferent as to make the operation valueless.

A curious feature of ischaemic nerve damage in the leg is the frequency of pain; it was present in six of the fifteen cases. I have never encountered this in ischaemia in the upper limb except in one case due to the hypoxaemia of barbiturate poisoning. The pain may be deep-seated, constant and burning, or it may be felt only when the foot is handled or comes into contact with the ground, then resembling the hyperpathia not uncommonly seen after ordinary injuries of the sciatic nerve if they are partial or in process of recovery after suture. One patient complained of the pain only when at rest. In one case the pain was so intense as to be the chief factor determining amputation of the leg, after which it ceased completely.

**OTHER TISSUES**

Elsewhere (Seddon 1964) I have described ischaemic changes affecting tissues other than muscle and nerve. The only comment called for here is that the fibrosis at the periphery of the ischaemic muscle may involve the long flexor tendons behind the ankle and in consequence elongation of the calcaneal tendon alone may not suffice to correct the equinus deformity. The tendons of the long flexors of the toes and perhaps of tibialis posterior have then to be
dealt with and, because they are involved in a mass of scar tissue, the only satisfactory treatment is excision. The ischaemic muscle itself (Fig. 6) should also be excised; otherwise there will be recurrence of the contracture.

**TREATMENT**

The treatment of established ischaemia in the leg is simple because there is so little to be done—far less than in the upper limb.

The deformity must be corrected. This requires: 1) lengthening of the calcaneal tendon; 2) lengthening, or excision if involved in scar tissue, of the tendons of such other muscles as are producing deformity, and removal of any muscle bellies that are totally destroyed (in the case shown in Figure 10 a large necrotic mass was removed); and 3) if clawing of the toes persists after the tendons of their long flexors have been dealt with it will require local correction. The indications for amputation are pain, ulceration or scarring and intractable deformity (Table IV). It was required in four of the fifteen cases; the results were satisfactory.

**TABLE IV**

**AMPUTATIONS**

<table>
<thead>
<tr>
<th>Age at time of amputation (years)</th>
<th>Interval between injury and amputation (years)</th>
<th>Reasons for amputation</th>
<th>Length of below-knee stump (inches)</th>
</tr>
</thead>
<tbody>
<tr>
<td>8</td>
<td>2</td>
<td>Pain: +, Deformity: +, Scars: +, Ulceration: +</td>
<td>7</td>
</tr>
<tr>
<td>21</td>
<td>2</td>
<td>Pain: +, Deformity: +, Scars: +, Ulceration: +</td>
<td>5</td>
</tr>
<tr>
<td>27</td>
<td>8</td>
<td>Pain: +, Deformity: +, Scars: +, Ulceration: +</td>
<td>6</td>
</tr>
<tr>
<td>34</td>
<td>1</td>
<td>Pain: ++, Deformity: +, Scars: +, Ulceration: +</td>
<td>6</td>
</tr>
</tbody>
</table>

**PREVENTION**

Perusing reports of small collections of cases of ischaemia of the leg throws light on its etiology and pathology, but gives no indication of its frequency. Measures taken to prevent any sort of disaster must bear some relation to its rate of occurrence. How often does ischaemia of the leg occur in the day-to-day treatment of injuries of the lower limb?

In this connection a paper by Ellis (1958) is particularly valuable. He studied the disability occurring after fractures of the tibial shaft in adults treated in the excellent fracture service at Sheffield. There were 336 patients, 343 fractures and nine cases of ischaemic contracture. In the period covered by the survey there had also been six fractures with such severe circulatory disturbance that amputation had been necessary, although two of the bony injuries were what would ordinarily be called minor affairs. Thus disability due to vascular injury occurred in 4.3 per cent of fractures of the tibia and fibula—a slightly higher figure in terms of patients—of which 2.6 per cent were due to ischaemia that passed unnoticed. These figures can hardly be ignored and would be cause for serious concern if they related to supracondylar fractures of the humerus in children. Admittedly the hand is more important that the foot and ischaemia of the leg is not always very crippling. But a disorder that may lead to amputation is no light matter.

There are two problems here, that of the overt vascular catastrophe and the ischaemia coming on insidiously. If an obviously ischaemic leg is not otherwise damaged beyond hope of salvage, speedy exploration of the damaged vessel is called for and, if feasible, its repair. As Kirkup (1963) and other authors whom he quotes have shown, these major vascular injuries can be tackled successfully (he was referring to injury of the femoral artery complicating fracture of the femoral shaft) if recognised immediately and operated on as soon as the patient’s general condition permits.
In cases of the kind just described gangrene rather than ischaemic contracture is the penalty of delay, or of ineffective intervention.

The vascular damage that leads to ischaemic contracture can easily escape detection and, as in the upper limb, it has to be looked for. Elsewhere (Seddon 1964) I have reviewed the signs of impending ischaemia in the upper limb. Neither pain, pallor, cyanosis, pulselessness, paralysis, nor contracture was noted in over half the cases that were carefully documented. The most reliable sign of all is painful limitation of extension of the fingers. The treatment of many injuries of the lower limb includes fixation in plaster-of-Paris and this restricts examination of the foot. Nevertheless, if only the possibility of ischaemia is borne in mind it should be possible to detect its early manifestations and then intervene promptly as has been done so often and so successfully in the upper limb.

I have never been in charge of an accident or fracture service and it would be presumptuous for me to describe a programme of attack that I have not had the opportunity of carrying out. My personal experience has been limited to a few cases of major vascular catastrophe with threatened gangrene. However, it may confidently be affirmed that for injuries below the knee with impending ischaemia generous incision of the deep fascia, particularly that of the anterior and lateral compartments, and evacuation of any haematoma that may be present deep to the muscles thus exposed will be high on the list of necessary measures. They are regularly employed by one of my colleagues, K. I. Nissen (1965), who in all cases of this sort favours skeletal traction during the period of uncertainty.

The avoidance of Bryant’s traction or traction on an inclined plane in children seems desirable in view of the accidents that have been reported, some of which involved the normal leg. Yet I am bound to record that both these methods of treatment have been used at the Royal National Orthopaedic Hospital over a period of thirty-four years, and are still being used, without one single vascular catastrophe. In operations on the tibia, particularly at its upper end, the risk of vascular damage is considerable (Fig. 11) and scrupulously careful technique is essential.

When the vascular lesion is at or above the knee there is now no doubt that the main vessel should be dealt with if less radical measures have failed. There is less certainty about direct attack on vessels below the knee.

**SUMMARY**

1. Volkmann’s ischaemia of the lower limb is more common in adults than in children and occurs with sufficient frequency after injuries of the femur, knee and leg to warrant a more determined effort to prevent it.
2. The first and most essential step is to recognise the early signs of ischaemic damage. Incision of the deep fascia may then save the threatened underlying muscle, though it may also be necessary to seek for and evacuate a haematoma beneath the muscle. When the femoral or popliteal artery is injured, exploration and repair may be imperative.
3. The treatment of established ischaemic contracture is by whatever measures are necessary to correct the deformity. These are lengthening of shortened tendons, or excision of them if they are involved in dense fibrosis at the periphery of the ischaemic mass; and excision of all totally destroyed muscle. Amputation may be necessary.

I am greatly indebted to my colleagues at the Royal National Orthopaedic Hospital for allowing me to study six of their patients who are included in this survey. Mr K. M. Bryant has also been kind enough to allow me to see and to refer to one patient under his care.

REFERENCES


