THE RELIEF OF TRAUMATIC ARTERIAL SPASM IN THREATENED VOLKMANN'S ISCHAEMIC CONTRACTURE

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The etiology of Volkmann's contracture has been much debated. Incorrect bandaging (Volkmann 1881), venous occlusion (Murphy 1914, Brooks 1922, Jepson 1926), subfascial haematoma (Moulonguet and Senèque 1928, José Jorge 1925), arterial spasm (Griffiths 1940) are some of the causes that have been suggested. Leriche (1928) likened the changes in the muscles to those seen in cardiac and pulmonary infarction. In the forearm, muscle necrosis may affect the extensors as well as the flexors; this distribution can be explained by attributing the necrosis to arterial occlusion (Seddon 1956).

Massart (1935) observed that the muscles were infiltrated by a sero-haemorrhagic exudate and that they appeared ready to burst. Personal observations at operation on patients suffering from limb injuries with vascular complications suggest that the presence of oedema alone in a neighbouring fascial compartment may be an important etiological factor.

CASE REPORTS

Case 1—A man aged twenty-five years fractured the middle of the shaft of his left tibia and fibula in a motor cycle accident. On admission to hospital four hours later the foot was blue and cold. The popliteal region and calf were swollen, and neither the popliteal pulse nor the foot pulses were palpable.

The fracture was reduced, and six and a half hours after the accident a spinal anaesthetic was given, but the circulation in the foot did not improve; therefore the popliteal fossa was explored through a longitudinal incision. No bleeding was seen from the skin or deeper structures, and the popliteal artery could not be identified although the vein was traced throughout the length of the popliteal fossa. The incision was extended down to the level of the ankle, and, on division of the deep fascia of the calf, the muscles bulged out. The popliteal artery dilated at once and the operation field began to bleed. The foot regained its normal colour, and the pulses became palpable. The fascia and skin edges were three inches apart, and since they could not be sutured without interfering with the circulation the wound was packed with vaselined gauze. A well padded plaster was applied and bivalved. The systolic blood pressure did not fall below 125 millimetres of mercury during the operation. Two weeks later secondary suture was carried out, and the limb subsequently recovered its full function.

Neither haematoma nor damaged tissues were seen at any stage of this operation. The fracture haematoma remained deep to the fascia on soleus; yet the uninjured gastrocnemius was oedematous. Immediately the tension in this oedematous muscle was released the spasm in the popliteal artery disappeared.

Case 2—A girl aged seven years fell on her outstretched hand and sustained a supracondylar fracture of the left humerus. On examination the fingers were cold and the radial pulse was not palpable. Four hours after the injury gentle manipulation under general anaesthesia failed to reduce the fracture, and, because of the circulatory impairment, exploration was carried out immediately.

The cubital fossa was exposed and found to contain a haematoma, and the neurovascular bundle had been impaled by the distal end of the proximal humeral fragment. Distal to the spike the artery was threadlike and did not pulsate; proximal to the spike it was normal. The neurovascular bundle was dissected free, a local periarterial sympathectomy was performed
and a 2·5 per cent solution of papaverine sulphate was applied to the artery. These measures failed to relieve the spasm. The deep fascia enclosing the flexor muscles of the forearm was then incised longitudinally and the muscles burst out, having been under tension from oedema. There was no sign of injury distal to the elbow and no spread of haematoma into the forearm muscles. Warm packs were applied, and, after twenty-five minutes, the arterial spasm disappeared, the wound began to bleed and the radial pulse became palpable at the wrist. The fracture was reduced and as the skin edges could not be approximated a split-skin graft was applied. The systolic blood pressure did not fall below 115 millimetres of mercury during the operation, and no change (of blood pressure) occurred at the time that the spasm relaxed. The limb was immobilised on a plaster slab and later made a complete recovery.

Case 3—A girl aged sixteen injured her left forearm, causing separation of the lower radial epiphysis with slight displacement. On examination, within two hours of the injury, the fingers were blue and cold, and the radial pulse was not palpable.

The wrist was manipulated under general anaesthesia and a plaster slab applied, but there was no improvement in the circulation. A brachial plexus block was followed by return of the radial pulse. Four hours later the hand again became blue and cold and the radial pulse impalpable.

Under general anaesthesia a longitudinal incision was made on the flexor aspect of the forearm. When the fascial compartment of the flexor group was incised the muscles bulged out and the circulation in the hand recovered immediately. There was a haematoma in the distal third of the flexor muscles, all of which were oedematous. An attempt was made to suture the fascia, but the radial pulse was obliterated when the sutures were tightened; therefore the wound was packed with vaselined gauze. Secondary suture, two weeks later, was followed by complete recovery.

In this case the arteries were not seen, and therefore the existence of the spasm was not proven. However, the transient improvement in the circulation of the hand which followed brachial plexus block suggests that the impairment of the blood supply was due partly to arterial spasm.

DISCUSSION

There is considerable evidence to show that incision of the fascia enclosing the flexor muscles of the forearm may prevent irreversible ischaemic changes (Murphy 1914, José Jorge 1925, Jepson 1926, Moulonguet et Senèque 1928, Massart 1935). In this paper an attempt is being made to relate this with the view that Volkmann's ischaemic contracture is due to arterial occlusion (Griffiths 1940, Seddon 1956).

In Cases 1 and 2 tension in muscle groups distal to arterial spasm was due to oedema and was not associated with haematoma, sero-haemorrhagic exudate or other signs of injury. In Case 3, although haematoma was present, oedema was a prominent feature. The oedema causing tension in these muscles might be due to arterial spasm and not to direct injury. It is possible that soon after the appearance of spasm the anoxia from diminished blood flow (Cohen 1940) causes increased capillary permeability and exudation of fluid. Tension mounts in the flexor muscles of the forearm, or the calf muscles of the legs, because they are enclosed by unyielding fascia.

There is a tendency for an artery in spasm to relax spontaneously provided the systemic blood pressure remains within normal limits (Kinmonth 1952); therefore when spasm persists without obvious local cause it is desirable to look further afield. My observations suggest that tension from oedema in a neighbouring fascial compartment may be the stimulus that maintains the spasm. Thus a vicious circle is set up, in which the increased tension maintains the arterial spasm, and the consequent ischaemia aggravates the oedema and hence the tension in the fascial compartment.

Spasm of an artery is occasionally seen when it is being handled at operation, and may spread for several centimetres (Rob and Eastcott 1957). It may be by such a mechanism that
tension on an artery, or its branches, causes or maintains spasm of the main vessel several centimetres away.

If this supposition be correct, two fascial compartments may have to be explored, because the cubital or popliteal fossa can be opened without releasing tension in the neighbouring ischaemic muscle group.

SUMMARY

1. Three cases of traumatic arterial spasm are reported.
2. In each case there was increased tension in a neighbouring myofascial compartment.
3. The cause of this tension was oedema, possibly supplemented in one case by haematoma.
4. Release of tension by splitting the sheath was followed by relaxation of the artery.
5. It is suggested that tension in a fascial compartment may provide the stimulus that maintains arterial spasm and that the consequent ischaemia aggravates the oedema, so that a vicious circle is established.
6. It is further suggested that if spasm persists in spite of the usual measures, including exploration of the artery, the distal myofascial compartment should be decompressed. Division of the deep fascia of the cubital or the popliteal fossa is not enough.
7. Such persistent arterial spasm is uncommon, and further observations are needed to define the significance of increased tension in a distal myofascial compartment.

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