LATE SURGICAL DECOMPRESSION FOR COMPARTMENT SYNDROME OF THE FOREARM

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Two cases are reported of the late diagnosis of compartment syndrome secondary to alcohol and drug overdose. Surgical decompression at two and a half days and at six days, respectively, produced worthwhile recovery. Other reports are reviewed and a case is made for the value of decompression even when performed late, and for delayed and minimal excision of apparently necrotic muscle.

Local compartment syndromes, which may occasionally lead to crush syndrome, can result from lying on a limb for a long period as a result of overdose of alcohol or of drugs (Conner 1971; La Force 1971; Schreiber, Liebowitz and Bernstein 1972; Dolich and Aiache 1973; Gaspard and Kohl 1975; Mubarak and Owen 1975; Newmeyer and Kilgore 1976; Mubarak et al. 1978; Owen et al. 1979; Gelberman et al. 1981). Some reports suggest that recovery is less likely if decompression is delayed for more than 12 hours after onset (Rorabeck and Macnab 1976; Sheridan and Matsen 1976; Echtermeyer et al. 1982). The patient suffering from an overdose may, however, for obvious reasons, present with a compartment syndrome which has been present for several days.

This paper reports two cases in which worthwhile function was regained after late decompression. This was done for both forearms of one patient two and a half days after onset, and for one arm of the other patient after six days. The results of fasciotomy and conservative resection of necrotic muscle are reported at 17 and 12 months respectively after operation.

CASE REPORTS

Case 1. A 42-year-old alcoholic woman was admitted 54 hours after consuming an entire bottle of rum. She had been lying comatose with her arms under her body for 20 hours, and had lost all power of movement and all sensation in her hands. She had been incontinent while unconscious, and because of this lay on an electric storage radiator to dry her clothes. She again fell asleep with her arms under her and slept for a further 12 hours. Because she was unable to use her hands after this it was a further 22 hours before she was rescued from her flat and brought to hospital.

On admission she could move her fingers very slightly, but had almost total anaesthesia in the distribution of all three major nerves to both hands. There were blisters resembling burns on her face and on both forearms and hands. Treatment by broad-spectrum antibiotics was started, and a compartment syndrome of both forearms was diagnosed.

At operation, bilateral open volar fasciotomy was carried out; it extended from the antecubital fossa into the hand, and included division of the bicipital aponeurosis, the carpal ligament and the fascia over the ulnar nerve at the wrist. A semi-closed fasciotomy was considered to be adequate on the dorsal surface, since the volar skin incision had been left open, thus ensuring that the skin could not form a limiting membrane.

When the volar fascia was first incised, swollen muscle and much serosanguineous fluid under pressure was released. All the muscle of the left forearm was pale and woody. Its colour improved slightly after fascial release, but the muscle neither contracted on pinching nor bled on cutting. In the right forearm, some contraction did occur on pinching the grossly oedematous muscle. Collections of fluid under pressure were found between the deep muscle bellies, and further fascial release over individual muscle bellies was performed. The ulnar and the median nerves were explored and decompressed. No devitalised muscle was excised at the primary operation because all the muscle looked uniformly bad. The wounds were covered with loose moist dressings only.

Infusion of large volumes of plasma and crystalloids was required after operation in order to keep pace with the large volume of fluid which drained from the wounds. An attempt was made to maintain a high volume of urine output in order to reduce the chance of renal failure secondary to myoglobinuria. Despite this, two days after operation, the patient developed anuria; this was treated successfully by the infusion of high doses of frusemide.

The wounds were inspected every few days and on each occasion only muscle which was brown and totally avascular was excised. Most of the muscle regained a good circulation, though at first it was rubbery, homogeneous and did not contract on stimulation. No contraction at all was demonstrable in the right forearm.
after the initial operation. The wounds were eventually covered with skin grafts; these were applied to the right arm eight days after decompression, and to the left arm after 17 days. Both hands were held in a functional position by volar splints and passive movements were started.

Some movement and sensation had started to return to the right hand before the patient left hospital after 57 days. Four months after operation, an adduction contracture of the right thumb developed rapidly over a period of three weeks. Useful sensation and movement began to appear after five months and, eight months after injury, the patient was able to return to work as a typist.

Seventeen months after decompression there was normal sensation apart from some blunting at the tips of the middle and index fingers of the left hand. The range of movement is shown in Figures 1 to 4. Power within this range of movement was normal, but contracture of the first dorsal interosseous muscle had produced an adduction deformity of the right thumb. Surgical release of this contracture is being considered.

**Case 2.** A 62-year-old man was found unconscious at home having taken an overdose of amylobarbitone. He had vomited and on admission he had an aspiration pneumonitis for which treatment in the intensive care unit was needed. His right arm was swollen and blistered, and there were raised erythematous patches on the right side of his trunk and head. Broad-spectrum antibiotics were given.

After 24 hours, it was noted that he was unable to move his right hand. His forearm was tense and purpuric, his hand was floppy and there was blunting of sensation in the fingers. Passive extension of the wrist caused pain in the forearm. He was transferred to a psychiatric ward.

Six days after admission he was referred for an orthopaedic opinion. On examination at that stage, there was gross pitting oedema of the right forearm, with anaesthesia distal to the wrist, and absence of motor function of both median and ulnar nerves in the forearm and hand.

Fasciotomy was carried out by the same technique as that used for the first case. The muscle was found to be pale and swollen and did not contract when pinched. Its colour improved a little after decompression, except for a dead area about 2 cm square in the superficial flexors. No muscle was excised at the first operation. After fasciotomy the arm was elevated; a high volume of urine output was maintained, and in this case there was no evidence of renal failure. The wounds were inspected at intervals under general anaesthesia. At the first two such operations any muscle which had not regained a blood supply was excised; it was usually easy to distinguish avascular from healthy muscle. After 18 days the wound was closed by secondary suture and treatment by splints and passive physiotherapy was started. Function of the hand recovered slowly; active flexion was first noted only after four months.

One year after operation there was reduced sensation distal to the metacarpophalangeal joints in the index and middle fingers, and distal to the proximal interphalangeal joints in the ring and little fingers, with loss of all sensation in the pulps of all four fingers. Sensation in the
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There Gelberman deciding middle policy. In this view, has been reported in the literature of functional and abnormality of the carpi 1975; Matsen and Veith 1981). Some have advised early excision of necrotic muscle (Dolich and Aiache 1973; Mubarak and Owen 1975), and the last-named authors report one case in which debridement of all of the volar muscles of the forearm led to the expected serious disability. The opposite policy was followed in a case reported by Conner (1971). Apparently total necrosis of the flexor digitorum superficialis and flexor carpi ulnaris-muscles was seen at fasciotomy; no muscle was excised and, one year later, no significant functional abnormality could be detected.

My view, in common with that of other authors (Sanderson et al. 1975; Gelberman et al. 1981; Matsen and Veith 1981), is that any excision of muscle should usually be delayed at least until the first dressing, about three days after decompression. Even at this operation any muscle of doubtful viability should be left for later reassessment. Some evidence in support of this opinion is discussed below.

Le Gros Clark (Clark WEL 1946), Clark and Wajda (1947), Vracko and Benditt (1972), and Sanderson et al. (1975) all demonstrated that regeneration of skeletal muscle could occur along the intact basement membrane which remained after the induction of ischaemic necrosis. It is of some interest that Clark reported severe impairment of this regeneration if the involved limb was immobilised, thus supporting the use of vigorous post-operative physiotherapy. Sanderson et al. in 1975 reported that regenerating muscle fibres do not mature and will atrophy if they do not regain a nerve supply. This emphasises the importance of minimising ischaemic damage to nerves by full exploration and decompression.

Time must be allowed for blood supply to the muscle bellies to recover. If the basement membrane is undamaged and some islands of undegenerated cells remain, the return of blood supply may lead to considerable regeneration. Excision of all apparently necrotic muscle would prevent this normal repair mechanism.

Fasciotomy, even at a late stage, is worthwhile. Dolich and Aiache (1973) quote complete recovery in one patient who had a fasciotomy one month after the onset of a compartment syndrome. Eaton and Green (1975) found that fasciotomy was effective after 16 weeks, while Spinner et al. (1972) had good results up to three weeks after onset, especially in children. This last group also report progressive deterioration of the histological picture with increasing periods of unrelieved compression, and conclude that spontaneous recovery without fasciotomy is unlikely.

Adduction contracture of the thumb, as a late and residual complication of compression syndrome, is well recorded (Conner 1971; Newmeyer and Kilgore 1976; Quiqley, Popich and Lanz 1981). It is the result of a compartment syndrome affecting the first interosseous compartment of the hand and may go unnoticed because the early clinical signs are masked by anaesthesia from nerve involvement in the volar compartment of the forearm. The prevention of this contracture requires either blind prophylactic decompression of thenar, hypothenar and interosseous compartments as practised by Quiqley et al. (1981) or the measurement of pressure in all six intrinsic compartments of the hand, in all established cases of compartment syndrome of the forearm, with a view to selective decompression.

It is now well recognised that good distal pulses and capillary return do not exclude the diagnosis of compartment syndrome. Most cases reported in the literature have had palpable pulses (Schreiber et al. 1972; Eaton and Green 1975; Mubarak and Owen 1975; Newmeyer and Kilgore 1976; Mubarak et al. 1978). To induce a compartment syndrome, pressure within tissues need only rise sufficiently to reduce capillary flow to the point when normal physiological function cannot be maintained. A pressure as low as 33 mmHg may cause problems (Matsen and Veith 1981), a level much below systolic arterial pressure.

Elevation of the limb before fasciotomy is contra-indicated. It is the raised tissue pressure that obstructs venous return from within the compartment and as lifting
the limb does not influence this venous obstruction it therefore cannot improve venous return. Elevation will, however, reduce the arterial perfusion pressure, and exacerbate the ischaemia. This was demonstrated experimentally by Matsen, Krugmire and King (1979).

Raised erythematous patches and bullae containing serosanguineous fluid are common features of compartment syndromes (Conner 1971; Schreiber et al. 1972; Dolich and Aiache 1973; Newmeyer et al. 1976; Owen et al. 1979), especially after prolonged periods of compression. In the past, they were erroneously considered to be drug reactions to an overdose of barbiturate (Gröschel, Gerstein and Rosenbaum 1970). A more plausible view is that they are caused by cutaneous ischaemia secondary to local pressure (Newmeyer and Kilgore 1976; Owen et al. 1979).

**Conclusions.** Early diagnosis and early decompression give the best chance of complete recovery, but late decompression is still worthwhile. Early or over-zealous excision of muscle should be avoided because the potential for revascularisation cannot be assessed at the first operation, and apparently necrotic muscle may regain its blood supply and show a strong capacity for regeneration. Continuity of a muscle belly may be important if it is to regain function after decompression. Passive and active physiotherapy are essential both to prevent joint stiffness and possibly to promote muscle regeneration.

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**REFERENCES**


