

Acute Compartment Syndrome: Update on Diagnosis and Treatment

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Abstract

Acute compartment syndrome can have disastrous consequences. Because unusual pain may be the only symptom of an impending problem, a high index of suspicion, accurate evaluation, and prophylactic treatment will allow the physician to intervene in a timely manner and prevent irreversible damage. Muscles tolerate 4 hours of ischemia well, but by 6 hours the result is uncertain; after 8 hours, the damage is irreversible. Ischemic injury begins when tissue pressure is 10 to 20 mm Hg below diastolic pressure. Therefore, fasciotomy generally should be done when tissue pressure rises past 20 mm Hg below diastolic pressure.

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The classic descriptions of late complications of ischemic contracture of the lower and upper extremities are those of Seddon^{1,2} and Owen and Tsimboukis.³ On the basis of their retrospective reviews, they advised the necessity of early recognition of ischemia and recommended that therapy should include fasciotomy of the affected limbs. They also recognized that the classic signs of pain, pallor, paralysis, pulselessness, and paresthesia could not be relied on entirely in the clinical evaluation of patients with suspected compartment syndrome. Their descriptions of the catastrophic results of not recognizing this disorder are well known.

Most important are the functional consequences to the patient. Failure to recognize the syndrome entails the risk of malpractice litigation. Templeman et al⁴ recently reported that in a short period in Hennepin County, Minnesota, there were eight out-of-court settlements, averaging \$225,000 each, in cases involving

compartment syndrome (or alleged compartment syndrome).

The myriad causes of compartment syndrome include complications of open and closed fractures, arterial injury, temporary vascular occlusion, snake bite, drug overdose, burns, acute and chronic exertional states, and gunshot wounds. Other possible causes include leakage from venous and arterial access, pulsatile lavage, contusions in hemophiliac patients, and intraosseous fluid replacement in an infant. The occurrence of any of these factors (and undoubtedly others), especially in association with head injury, drug overdose, or other obtunded states, is indeed treacherous.⁵ Thus, a general knowledge of the pathogenesis of this state and the methods of evaluation and treatment is important.

Pathogenesis

The most common cause of compartment syndrome is muscle in-

jury that leads to edema, which usually is proportional to the tissue damage. At the time of injury, energy is dissipated into the muscle itself, causing intracellular swelling. If the patient also sustains a fracture, formation of a hematoma exacerbates the problem by increasing the volume, hence the pressure, within the closed space. Because the extremities are composed of relatively nonyielding fascial compartments, circulatory embarrassment ultimately occurs as tissue pressure rises, causing ischemia and tissue damage.

In the case of arterial injury, the muscle is deprived of its blood supply, causing intracellular injury. With reestablishment of circulation by reanastomosis or fasciotomy, reperfusion injury occurs as the muscle swells, with secondary elevation of tissue pressure. If the pressure becomes high enough, further ischemic damage occurs.

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The recent studies by Whitesides et al,⁶ Heckman et al,^{7,8} Matava et al,⁹ and Heppenstall et al¹⁰⁻¹² have established a more accurate understanding of the thresholds and parameters of ischemia. The outcome measures used include magnetic resonance determinations of pH, tissue oxygenation, and energy stores, as well as histologic findings obtained with use of histochemistry, electron microscopy, and Doppler-flow studies. In injuries that produced complete ischemia, skeletal muscle remained electrically responsive for up to 3 hours and survived for as long as 4 hours without irreversible damage. Total ischemia of 8 hours' duration produced complete irreversible changes; variable results occurred after 6 hours of total ischemia. Peripheral nerves conducted impulses for 1 hour after the onset of total ischemia and could survive for 4 hours with only neurapraxic damage. After 8 hours of total ischemia, axonotmesis was usual, and irreversible changes in the nerve commonly occurred.⁶

The occurrence of ischemia secondary to diminished blood flow or cessation of blood flow to muscle is believed to result when the perfusion gradient in the tissues of the compartment falls below a critical level. Thus, perfusion is directly related to the patient's blood pressure. Although Heppenstall et al¹⁰⁻¹² prefer to relate changes to mean arterial pressure, this value is not as easily obtained or calculated as diastolic pressure. Experimentally measured terminal arterial pressure is equal to diastolic pressure¹³; therefore, we have continued to use diastolic pressure as the critical measurement.

Experimentally, ischemia is induced in healthy muscle when the intracompartmental pressure rises to a level 10 mm Hg below diastolic pressure.^{6,7,9-12} In tissue that has been damaged by injury, the resistance to ischemia caused by increas-

ing tissue pressure is decreased, which is likely because perfusion of these tissues may not be as effective in preventing ischemia. An intracompartmental pressure of 20 mm Hg below diastolic has been documented to significantly decrease tissue perfusion in injured tissues, resulting in ischemia and ischemic changes. In both experimental animals and human subjects, it has been shown that those with higher diastolic pressures are able to withstand higher tissue pressures without ischemic damage than those with lower diastolic pressures. Conversely, hypotensive subjects do worse. Thus, it is important to establish the general circulatory status before making decisions.

We recommend that fasciotomy be performed as the intracompartmental pressure approaches 20 mm Hg below diastolic pressure in any patient who has a worsening clinical condition, a documented rising tissue pressure, significant tissue injury, or a history of 6 hours of total ischemia of an extremity. To be effective in preventing the sequelae of ischemic damage, restoration of circulation by fasciotomy must be accomplished before permanent changes occur. Prophylactic treatment is important because the results in patients with paralysis were unsatisfactory in over 80% of the cases in a meta-analysis reported by Bradley.¹⁴ Fasciotomy will not reverse the changes caused by the initial trauma, but it can prevent changes due to secondary ischemia.

In a retrospective study of closed tibial fractures, Owen and Tsimboukis³ found a 10% incidence of changes that could be attributed to compartment syndrome. In a later prospective study of consecutive closed tibial fractures, Heckman et al⁸ found a 20% incidence of impending compartment syndrome. Following the guidelines described

above, the authors performed prophylactic fasciotomy, which was successful in aborting ischemic injury to the muscle and neural tissues of the leg in all patients.

Clinical Evaluation

Pulselessness, pallor, paralysis, paresthesia, and pain have been described as the clinical hallmarks of compartment syndrome. It is critical to note that these are the signs and symptoms of an established syndrome with ischemic injury, and that fasciotomy at this stage yields dismal results. Loss of distal pulses, pallor, and diminution of capillary refill rarely occur unless there is arterial injury or unless the artery passing through an affected compartment is subjected to tissue pressures approaching the patient's systolic pressure.

Tissue perfusion in a compartment is dependent on arteriolar and capillary perfusion gradients. Therefore, compartment syndrome may occur despite the presence of peripheral pulses, capillary refill, and lack of pallor. Paralysis and sensory changes are not noted until after ischemia has been present for a period of approximately 1 hour or more. Pain and aggravation of pain by passive stretching of the muscles in the compartment in question are the most sensitive (and generally the only) clinical findings before the onset of ischemic dysfunction in the nerves and muscles. Paresthesias may be present at this time. In a normotensive patient with a diastolic blood pressure of 70 mm Hg, an increase in tissue pressure from a normal resting value of 0 to 8 mm Hg¹⁵ to a level of 30 to 40 mm Hg will result in significant discomfort and aggravation of the discomfort with passive stretching of the affected tissues. Thus, pain with passive stretching and increasing and/or unusual

pain out of proportion to that expected are clinically important symptoms.

Because pain, increased pain on passive stretching, and paresthesia are subjective symptoms, they are diagnostically useful only in conscious patients who can respond cognitively to the examination. It should also be emphasized that pain—the only early complaint of a conscious patient with impending compartment syndrome—will diminish after the pressure-induced ischemia affects the conductivity of the nerves in the compartment, and a painless state will ensue. In an unconscious or obtunded patient at risk for compartment syndrome, tissue-pressure measurements may be the only objective criteria for diagnosis. In animal experiments, loss of nerve conduction occurs within 2 hours after the onset of ischemia. Research has shown that there is little recovery if fasciotomy is done once painlessness or paralysis has occurred.¹⁴

Another important point to emphasize is that if compartment syndrome is a distinct possibility, long-acting nerve blocks, continuous epidural anesthesia, and patient-controlled intravenous opiate analgesia should be avoided if possible. If there is a reason for their use, careful monitoring is essential.

Tissue-Pressure Evaluation

Principles

The diagnosis of florid compartment syndrome can often be made without tissue-pressure measurements. If a patient sustains an injury with the potential for development of compartment syndrome, tissue-pressure measurements obtained in conjunction with the history and physical examination are often helpful in making a more accurate diagnosis.

Regardless of the technique used, tissue pressures must be measured in a defined manner to identify the area of high pressure and greatest tissue damage. Our recent study of closed tibial fractures⁸ showed that differences in tissue pressure over cephalocaudal distances as small as 5 cm were both clinically and statistically significant (Fig. 1). As the area of highest pressure cannot be reliably predicted by palpation, it is often necessary to use multiple sampling sites within a single compartment.

In contrast, proximal vascular injury with reperfusion produces a diffuse ischemic process with a more uniform elevation of tissue pressure throughout the compartment. Greater uniformity is also likely when the cause of the syndrome is externally applied pressure, as in crush syndrome. After

traumatic injury, ischemic involvement may produce an irreversible segmental injury to nerve or muscle, resulting in dysfunction of the more distal parts of the limb. This has been described classically in both the upper and lower extremities by Seddon.^{1,2}

Our prospective study of tibial fractures⁸ also documented the relationship between the site of injury, the compartment involved, and the distance from the fracture site in the leg. The deep posterior and anterior compartments were most commonly involved, and the highest tissue pressure was usually at the level of the fracture or within 5 cm of the fracture. Tissue pressure invariably decreased when sampled at increasing distances proximal and distal to the site of the highest recorded pressure; this decrease in pressure was statistically significant. Thus, the

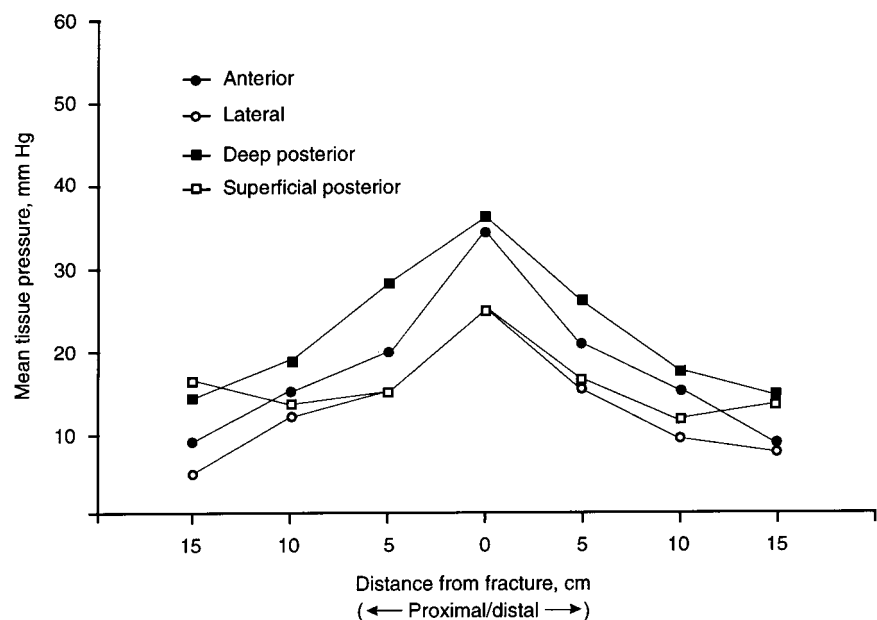


Fig. 1 Mean maximum tissue pressures measured in each compartment at the level of fracture and at 5-cm increments proximal and distal to it in 25 consecutive closed tibial fractures. There was an increased risk of higher pressure, and thus of compartment syndrome, in the anterior and deep posterior compartments at the level of fracture. (Reproduced with permission from Heckman MM, Whitesides TE Jr, Grewe SR, et al: Compartment pressure in association with closed tibial fractures: The relationship between tissue pressure, compartment, and the distance from the site of the fracture. *J Bone Joint Surg Am* 1994;76:1285-1292.)

pressure might be high enough to justify fasciotomy at one location, while only 5 cm proximal or distal to that site the pressure was not high enough to indicate the need for fasciotomy. Therefore, failure to measure tissue pressure more widely in the area of injury may result in serious underestimation of the maximum pressure present.

On the basis of these observations, we suggest that to reliably determine the location of the highest tissue pressure in patients with tibial fractures, measurements should be obtained in the leg, at a minimum, in both the anterior and the deep posterior compartments (Fig. 2) at the level of the fracture as well as at locations proximal and distal to the zone of the fracture. The highest pressure noted should serve as the basis for determining the need for fasciotomy.

When tissue pressure is rising toward the critical level for fasciotomy, careful follow-up is required. Repeated physical examination and pressure readings should be performed every 1 to 2 hours, accompanied by monitoring of other vital signs and symptoms. The pressure-measuring device can be

reinserted through the same needle-puncture site, or an indwelling monitor can be used to repetitively sample in an area of high pressure. We have observed patients in whom tissue pressures have remained at subcritical levels and then risen to critical levels over a period of 2 to 4 days; most frequently, however, the period was less than 24 hours. Therefore, these readings should be obtained until fasciotomy is needed or until the pressure has decreased to a safe level, accompanied by improving clinical signs and symptoms.

Techniques of Tissue-Pressure Measurement

A number of methods of tissue-pressure measurement have been described, some of which are still in use. These methods include the infusion technique (modified by Whitesides from older techniques for measuring subcutaneous edema pressure used in the early 1900s) and techniques involving the use of the Wick catheter (designed by Hargens), the Howmedica Slit catheter (designed by Rorabeck), and the Stryker STIC Device (designed by Stryker and Whitesides).

Currently, the most commonly used commercially available monitor specifically designed for tissue-pressure measurement is the Stryker STIC Device. Any clinical electronic arterial-pressure-monitoring device can be adapted to monitor tissue pressure with the use of a stopcock and extension tubes. Repeat measurements are difficult to obtain at multiple areas in the same compartment with an indwelling device. Therefore, needle methods are more appropriate for multiple-site and repeated measurements. Properly used, all of these methods are accurate and equally measure the same phenomenon, provided an appropriate zeroing technique is used. Because of the possible failure of electronic devices, a nonelectronic method (such as the infusion technique) should be considered as a backup.

Infusion Technique

The necessary equipment is inexpensive and readily available in hospitals, emergency rooms, and doctors' offices. The equipment includes (1) a mercury or accurate aneroid manometer or an electronic arterial-pressure monitor with transducers; (2) two plastic intravenous extension tubes; (3) two 18-gauge needles, preferably 1.5 inches in length; (4) one 20-mL syringe; (5) one three-way stopcock; (6) and one vial of bacteriostatic normal saline.

The steps in the technique are as follows:

(1) The extremity to be evaluated is cleaned and prepared so that pressure measurements can be obtained both proximal and distal to the level of injury.

(2) The vacuum in a sterile bottle of saline is broken with an 18-gauge needle so that fluid can be withdrawn easily (Fig. 3, A).

(3) A 20-mL syringe is attached to a three-way stopcock. One intravenous extension tube is attached,

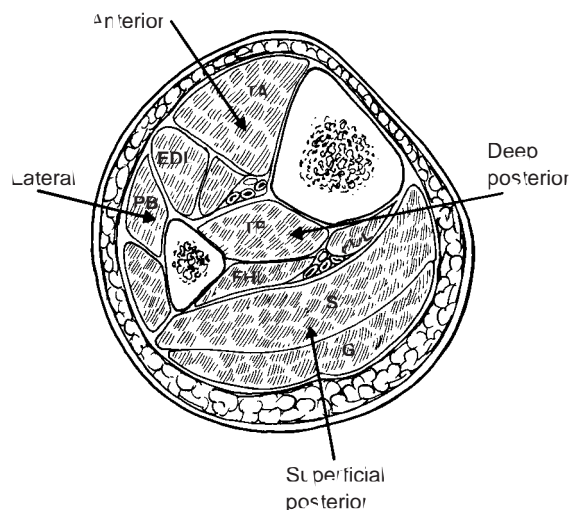


Fig. 2 Cross section of the proximal half of the leg shows the direction of needle insertion for testing each compartment. EDL = extensor digitorum longus; FDL = flexor digitorum longus; FHL = flexor hallucis longus; G = gastrocnemius; PB = peroneus brevis; S = soleus; TA = tibialis anterior; TP = tibialis posterior.

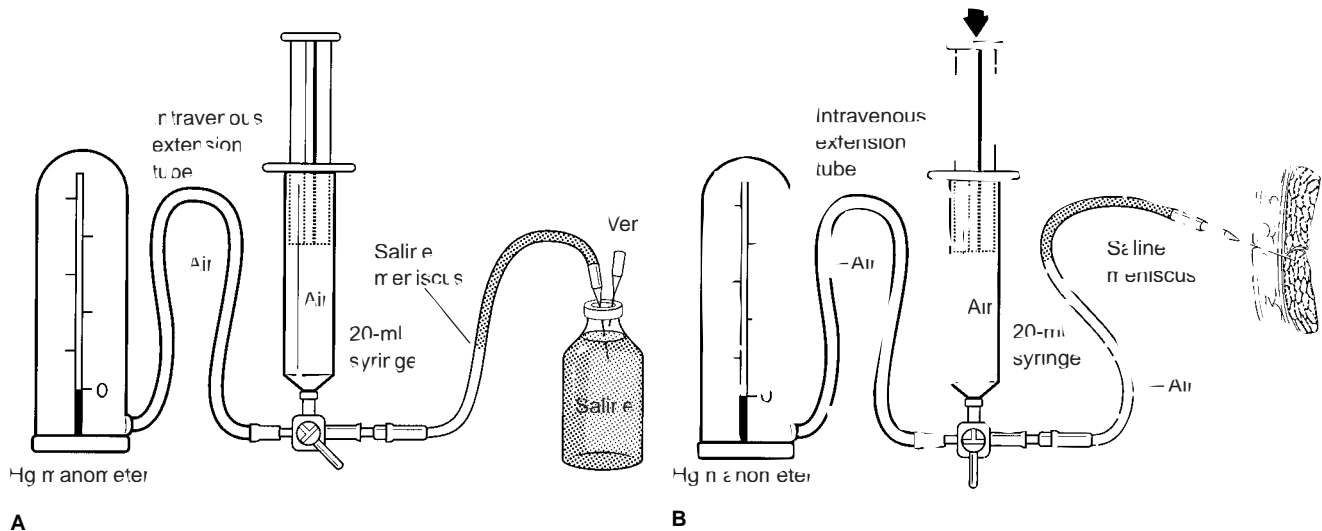


Fig. 3 A, Assembled equipment for Whitesides infusion technique just prior to placement of needle into area to be tested. Valve is in closed position. B, Configuration of equipment ready to test. The valve has been turned to an open position, making a "T" of open tubing.

and this is then attached to the second 18-gauge needle. The third, unused port of the stopcock is temporarily closed off.

(4) The 18-gauge needle at the end of the extension tube attached to the stopcock is then inserted into the bottle of saline, and the tip is placed beneath the level of the saline. Saline is aspirated without bubbles into approximately half the length of the extension tube. The three-way stopcock is then turned to the off position, blocking loss of saline during the transfer of the needle from the saline bottle to the patient's tissues.

(5) The second extension tube is connected to the three-way stopcock at its remaining open port, and the other end is then connected to the manometer. One can accomplish this with a similar apparatus when no stopcock is available by using intravenous tubing with access ports, multiple hemostats, and ingenuity.

(6) With the stopcock still closed to the extension tube containing the saline, approximately 15 mL of air is aspirated into the syringe, and the syringe is reattached. The saline-

containing needle is then inserted through the skin and fascia into the muscle to be tested.

(7) The stopcock is turned so that the syringe is open to both extension tubes, forming a T connection with a free column of air extending from behind the column of saline into the syringe as well as into the manometer (Fig. 3, B). This creates a system that allows air from the syringe to flow into both extension tubes as pressure within the system is increased in the process of measurement.

(8) The portion of the tube containing the top of the column of saline to be observed is then placed carefully at the same level as the tip of the needle in the patient. Any raising or lowering of this portion of the tubing will give an artificially low or high reading. The plunger of the syringe is then slightly depressed to inject a minute amount of saline to ensure that the system and the tip of the needle are clear of any obstruction.

When the pressure in the tissue exceeds that in the air column in the T-shaped system, the column of

saline in the tubing normally forms a convex meniscus away from the patient, due to capillary attraction. As the plunger of the syringe is slowly depressed, the column pressure is gradually raised, increasing the pressure in the system. The saline meniscus will be seen to change from convex to flat when the air pressure in the system equals the interstitial pressure in the patient's tissue. If the air pressure is raised higher than the interstitial pressure in the tissue, the saline meniscus will change from convex to concave. If the system is left in this state, saline will be injected.

The measurements should be recorded from the manometer when the saline meniscus is flat—that is, when the pressure in the tissue at the tip of the needle and the pressure in the column of air behind the saline are equal. Care should be taken not to read the pressure when saline is being injected into the muscle (Fig. 3, A), as this will result in an erroneously high reading.

(9) After recording the pressure at one site, the system is equilibrated

while withdrawing on the syringe plunger until a reading of 0 mm Hg is present on the manometer. This prevents saline from being lost from the system as the needle is withdrawn from the tissue. Other measurements may be performed by reinserting the same needle in this or other areas. If a new sterile needle is attached, however, the column of saline must be advanced until saline exudes from the tip of the needle.

Medical Treatment

Animal studies have shown that muscle tolerance of ischemia may be extended by hypothermia, anticoagulants, and corticosteroids. Clinically, anticoagulants and corticosteroids cannot produce their desired pharmacologic effects unless perfusion has been reestablished, giving access to the muscle tissue. Hypothermia, however, may have a helpful clinical application.

When reperfusion cannot be achieved in a timely manner, hypothermia can be used in a protective fashion until revascularization or fasciotomy can be performed. For example, a patient with an arterial injury might have other injuries that are so critical that delay of revascularization is appropriate. The ischemic extremity might be cooled in the interim, while awaiting revascularization. If the limb is revascularized and the compartments receive circulation after a 4- to 8-hour period of ischemia, tissue pressure should be evaluated immediately, and fasciotomy should be done if appropriate.

Surgical Treatment

Vascular repair and fasciotomy to reestablish circulation are the only reproducible methods of therapy when ischemia is present. Because of space constraints, our discussion

of surgical treatment will be limited to the leg and forearm.

Leg

The leg may be considered as generally comprising four compartments: anterior, lateral, superficial posterior, and deep posterior. The tibialis posterior muscle sometimes occupies a separate fascial compartment of its own.

Of anatomic interest is that the soleus muscle takes origin from the tibia and fibula for the entire proximal half of the leg. Thus, underneath this "soleus bridge" the deep posterior compartment and its contents are not subcutaneous. For fasciotomy of the deep posterior compartment, the soleus origin must be detached from either the tibia or the fibula. The anterior compartment is easily palpable on the anterolateral side of the leg, and the lateral and superficial posterior compartments are also totally subcutaneous. Therefore, measuring the tissue pressure in the anterior, lateral, and superficial posterior compartments is very direct. The pressure in the deep posterior compartment can be easily measured in the distal half of the leg medial and posterior to the tibia, as it is subcutaneous. Proximal to this area, the pressure is most easily measured through the soleus origin from the tibia (Fig. 2).

We initially used fibulectomy as a means of performing four-compartment fasciotomy of the leg. We now prefer a perifibular approach through a single incision, although this is more difficult than a two-incision method. The benefits of the single incision are that it leaves only one wound to repair and that this incision is usually more distant from the most commonly occurring fracture wounds. Consequently, it is worthy of consideration despite its increased difficulty.

The two-incision method includes an anterolateral approach,

with the incision done longitudinally to expose the contents of the anterior compartment and the lateral compartment, taking care to preserve the superficial peroneal nerve. The medial incision is made in a longitudinal manner just posterior to the tibia. The superficial posterior compartment is then opened, and the soleus is detached from its tibial origin to expose the deep posterior compartment in the proximal half of the leg. In the distal half of the leg, the deep compartment is subcutaneous and can be approached directly.

The perifibular approach is carried out through a straight lateral incision beginning just posterior and parallel to the fibula from the level of the fibular head to a point above the tip of the lateral malleolus. At the proximal end of the incision, the common peroneal nerve should be exposed and/or protected. The dissection is then deepened to incise the fascia between the soleus and the flexor hallucis longus distally and is extended proximal to the soleus origin from the fibula (Fig. 4). This allows access to the entire length of the superficial posterior and deep posterior compartments. The incision should be made long, as decompression of the deep posterior compart-

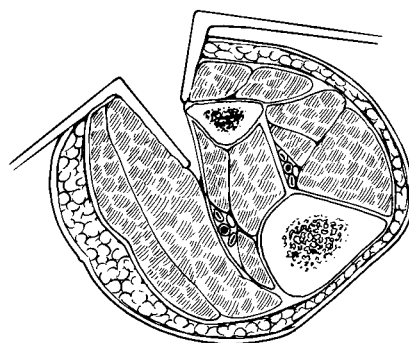


Fig. 4 Approach for fasciotomy of the deep posterior compartment with use of a perifibular technique.

ment is more difficult with this dissection than with dissection from the medial side. The anterior edge of the incision is then retracted to expose the anterior and lateral compartments, taking care to avoid the superficial peroneal nerve as it exits the fascia of the lateral compartment and runs anterior in the distal third of the leg. At the end of this dissection, the tibialis posterior muscle and others should be checked to ensure that any less common anatomic arrangement of the compartment is not missed.

The fracture can then be stabilized. A locked unreamed intramedullary nail or an external fixator is generally used. Our preference is for intramedullary fixation, as it allows easier care of the wounds and the leg. The wounds are left open, and a large, nonrestrictive, bulky dressing is applied. There is usually a significant amount of drainage. Delayed primary closure may be attempted 3 to 7 days after fasciotomy if closure can be done without tension. Alternatively, a split-thickness skin graft may be placed for muscle coverage.

Although subcutaneous fasciotomy may be appropriate in chronic exertional compartment syndromes involving only the anterior or lateral compartment, it cannot be used for decompression of the deep posterior compartment because this compartment is subcutaneous only in the distal half of the leg. In our experience, whenever a fasciotomy is done prophylactically with tissue pressure rising toward the critical threshold, closure of the incision is not possible. We therefore believe that subcutaneous fasciotomy cannot be used safely in the treatment of acute compartment syndromes of whatever etiology.

Forearm

Anatomically, the forearm can be divided into three compartments: dorsal, volar, and "mobile wad" (Fig. 5). The mobile wad consists of

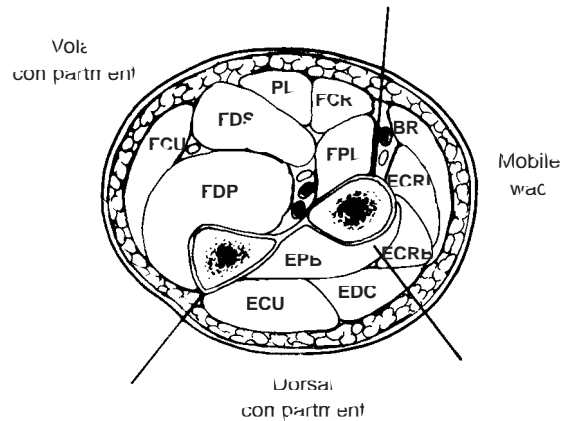


Fig. 5 Cross section of the midportion of the forearm shows location of the various compartments. BR = brachioradialis; ECRB = extensor carpi radialis brevis; ECRL = extensor carpi radialis longus; ECU = extensor carpi ulnaris; EDC = extensor digitorum communis; EPB = extensor pollicis brevis; FCR = flexor carpi radialis; FCU = flexor carpi ulnaris; FDP = flexor digitorum profundus; FDS = flexor digitorum superficialis; FPL = flexor pollicis longus; PL = palmaris longus.

three muscles: the brachioradialis, the extensor carpi radialis longus, and the extensor carpi radialis brevis. The dorsal compartment contains the extensor pollicis brevis, the extensor carpi ulnaris, and the extensor digitorum communis. These muscles are innervated by the posterior interosseous nerve and receive their blood supply from the posterior interosseous artery and through interosseous perforators arising from the anterior interosseous artery. The volar compartment contains the muscles responsible for flexion, pronation, and supination of the wrist, hand, and fingers: the flexor pollicis longus, the flexor carpi radialis, the flexor carpi ulnaris, the flexor digitorum superficialis, the flexor digitorum profundus, and the palmaris longus. These muscles are innervated by the median and ulnar nerves and receive their blood supply from the radial, ulnar, and anterior interosseous arteries.

In evaluating any patient for a suspected compartment syndrome of the forearm, the pressures in all compartments should be measured. Compartment syndrome is most common in the volar compartment but may also develop in the deep aspects of the posterior compartment, even when the superficial aspects of

the posterior compartment are not involved. Therefore, both the deep and superficial aspects of the posterior compartment should be specifically tested individually.

In some cases, tissue pressures in the mobile wad and dorsal compartments will significantly decrease after volar release, making a dorsal fasciotomy unnecessary. Therefore, fasciotomy is first done volarly.

Volar fasciotomy begins with an incision above the elbow laterally in the manner of Henry and is then extended distally and transversely across the antecubital fossa to the ulnar aspect of the proximal portion of the forearm (Fig. 6, A). It is continued distally along the ulnar side to the level of the wrist and then medially onto the volar aspect of the wrist, paralleling the thenar crease. The laceratus fibrosus is routinely released at the level of the elbow to decompress the median nerve and other structures. The fascia of the forearm is then opened from proximal to distal, exposing the ulnar and median nerves and opening the superficial and volar muscles as well as the intervening neurovascular structures. The pronator teres and flexor digitorum superficialis may have to be released distally to complete decompression of the median nerve in some cases. Epimysiotomy is per-

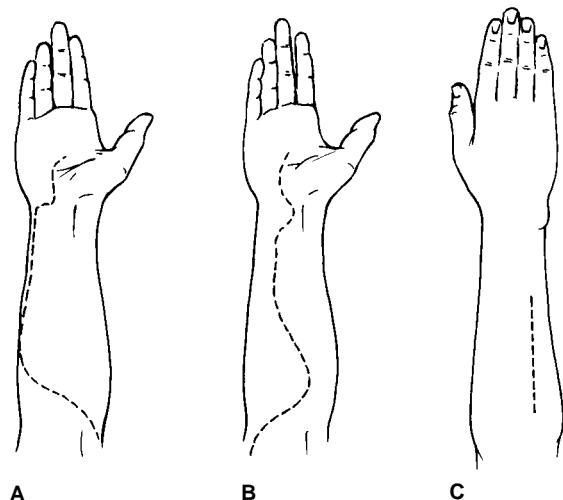


Fig. 6 Incisions for forearm fasciotomy. **A**, Volar approach of Henry extended into the carpal tunnel. **B**, Curvilinear volar approach from above the elbow into the carpal tunnel. **C**, Dorsal approach.

formed on any enveloped muscles. A carpal tunnel release is done to ensure decompression, especially if carpal canal tissue pressures are elevated. Alternatively, a curvilinear volar approach to the forearm can be used (Fig. 6, B).

After completion of the volar decompression, pressure measurements are obtained in all compartments, including the dorsal compartment. In cases in which the dorsal pressures remain elevated, a dorsal incision is made in a line with the lateral epicondyle of the humerus and the distal radioulnar joint. The incision should extend at least to the junction of the middle and distal thirds of the forearm, as most of the musculature is proximal to that point (Fig. 6, C). The extensor retinaculum of the wrist should not be disrupted. The underlying fascial incision should be in line with the skin. Tissue pressures at this point should again be obtained in the deep dorsal compartment and the mobile wad. Rarely will one then have to make a third incision in the brachioradialis to make certain that it is decompressed.

An alternative approach for decompression of the forearm is that of McConnell combined with exposure of the median ulnar nerves as de-

scribed by Henry (Fig. 6, A). The advantage of the ulnar-side approach we have described over this approach is that the flexor tendons and median nerve are left with good soft-tissue coverage in the area where necrosis is most likely to occur. Either of these approaches, if completed appropriately, will result in adequate decompression. In no case should subcutaneous fasciotomy be performed alone because, without direct visualization of the fascia, superficial nerves and veins will be injured and inadequate decompression of the median and ulnar nerves will likely occur.

Postoperatively, the arm is immobilized in a bulky, noncompressive dressing with a plaster splint and with appropriate splinting of the hand. Delayed skin closure or split-thickness grafting should be done after appropriate reduction of edema. This is important to provide soft-tissue coverage of the exposed muscles and especially the tendons and nerves.

Missed Compartment Syndrome

Unfortunately, compartment syndrome is sometimes diagnosed too

late for intervention, or the patient does not seek care. Thus, at times one has to make a decision about appropriate therapy when a compromised result is inevitable. It is unusual to see untreated severe compartment syndrome that results in total death of the limb and dry gangrene. More often, the patient presents with various stages of muscle infarction, muscle contracture, secondary deformity, lack of motion, neurologic involvement with paralysis of distal muscles, and loss of sensation. Rarely is infection a problem. However, if fasciotomy is done after the injury to muscle is essentially total, secondary infection may supervene, as the necrotic muscle is a perfect culture medium. Gram-negative sepsis, including clostridial infection, has been reported, and amputation has occasionally been the result.

The timing of surgical therapy is very important and often difficult. The instructions in regard to the forearm that Seddon gave many years ago are still appropriate. Reconstructive surgery should be carried out before there has been too long a period of constriction about the nerves, but should be done only when it can be determined which muscles should be excised and which transfers can be made. The same is true of the leg.

Difficulty occurs when massive changes due to missed compartment syndrome have become obvious. There is an impulse to do a fasciotomy even though it is obvious that irreversible changes have become present in the muscle as well as the nerve. If the surgeon is certain the muscle can be debrided accurately and adequate neurolysis can be performed, intervention may be appropriate. However, fasciotomy will not help and may make the situation worse because the muscle is necrotic and the risk of infection is high. In addition, there are strong reasons in late circumstances not to carry out fasciotomy or decom-

pression in the anterior compartment of the leg. The scar contracture that occurs effectively works as a "checkrein" to counteract the foot-drop, whereas debridement of the muscle removes this checkrein and will not improve sensation in the foot.

Complications of Compartment Syndrome

Myonecrosis

Unfortunately, patients do present after an ischemic insult of 8 or more hours' duration. It can be expected that myonecrosis will occur on revascularization. Fasciotomy and debridement of the muscles, as well as neurolysis, may become necessary.

Myonecrosis is often a problem after a crush injury. Cellular damage results in edema when circulation is reestablished. This causes increased interstitial pressure and, if the pressure becomes critical, further ischemia. Myoglobin may be absorbed (clinically evidenced by myoglobinuria). Because renal damage may result, diuresis should be promoted to increase tubular flushing and eliminate the proteinaceous material. This can be done with the use of mannitol, diuretics, and intravenous fluids. If this therapy is inadequate, surgical debridement of injured muscle to lower the myoglobin burden and decrease renal damage may be appro-

priate. Excision of necrotic muscle can also decrease secondary neural compression and muscle contracture (the cause of much secondary joint deformity and malfunction).

Complications in the Leg

In reports of late follow-up of closed tibial-shaft fractures, ischemic contracture or neural injury occurred in up to 10% of patients.^{2,3} Clinically affected patients may complain of burning pain and anesthesia of the extremity and may present with ulcerations of the skin, deformity, and difficulty in ambulation. How the extremity is affected is dependent on the compartments involved in the ischemic event. In cases involving the deep posterior compartment, findings may range from simple clawing of the toes to more extensive equinus and cavovarus deformities. Sensory changes are reflected in insensitivity of the sole and the plantar aspect of the toes. Infarction in the anterior compartment results in a foot-drop; as the contracture of the muscles increases, this will diminish because of a checkrein effect.

Complications in the Forearm

Although classically Volkmann's contracture of the forearm results from an ischemic injury after a supracondylar fracture of the humerus, this contracture occurs more commonly after injuries to the forearm. The deep

flexor muscles of the forearm are most severely involved; the more superficial ones are progressively less involved.¹ As the condition progresses, fixed contractures and deformities become increasingly more severe, depending on which nerves and muscles are affected and to what degree.

Summary

Recent studies have better elucidated the parameters of tissue ischemia and the location of insult to muscle in injured extremities. This information is helpful in the decision-making process necessary to carry out effective, prophylactically timed fasciotomy in an attempt to avoid the deleterious effects of untreated compartment syndrome. Pain out of proportion to injury is the only early symptom of impending acute compartment syndrome. If ignored or covered up by injudicious opiate administration, general anesthesia or other anesthetic procedures (such as peripheral nerve or epidural blocks), or an obtunded state of any cause, the opportunity to treat effectively in a prophylactic manner and obviate a catastrophic state may be missed. Understanding the physiology of compartment syndrome and the parameters of tissue ischemia is essential to effecting timely intervention and producing a more salubrious result.

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