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An Update in Compartment Syndrome Investigation and Treatment

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Abstract: Compartment syndrome continues to be prevalent in the practice of orthopaedic surgery today. The limb- and life-threatening complications of untreated compartment syndrome however, have decreased in frequency. This is mainly because of a better understanding of the pathogenesis and early recognition and treatment of the condition. This article presents a comprehensive review of the pathogenesis, diagnosis, and treatment of compartment syndrome of the extremity. The recent research experience at the University of Pennsylvania is also discussed, as well as my preferred techniques of diagnosis, surgical decompression, and wound closure. With prompt recognition and treatment, the potentially crippling complications of untreated compartment syndrome can be prevented.

Introduction

Since the original description of post-ischemic contracture by Volkman [5] more than a century ago, much has been written about ischemic compartment syndromes. In his now classic article, Volkman described the state of muscular contracture which he attributed to ischemia caused by trauma, swelling, and tight bandaging. Early in the 20th century, researchers began to focus their attention less on the ultimate contracture state and more on the pathophysiologic features of the inciting event.

In 1958, Ellis [4] discussed disabilities following tibial shaft injuries having a 2% incidence of ischemic contracture as a complication. This was followed by reports by Seddon [36,37] on Volkman's ischemia in the lower limb and by a further report from Owen and Tsimboukis [33], who reported on ischemia complicating closed tibia and fibular shaft fractures.

In spite of these early reports of ischemia following the treatment of injuries to the lower extremity, a heightened awareness of this syndrome is needed in managing orthopaedic trauma patients on a day-to-day basis. The financial awards that accompany legal representation of a missed compartment syndrome have been substantial [40]. This is because a missed full-blown syndrome results in a significant deficit in the involved extremity in follow-up. Various compartment syndromes have been described in the upper extremity including the shoulder and upper humeral areas as well as the forearm and hand. In the lower extremity, syndromes have been reported in the buttock and thigh area, the more common lower leg involvement, and the recently emphasized foot compartment syndromes. Recently, both teaching and descriptions of this problem have increased in academic centers, national orthopaedic meetings, and in orthopaedic publications. This has resulted in an improved understanding of the syndrome as well as improved technology regarding adjunctive measurement techniques which aid in the diagnosis of this complication.

Pathogenesis

A simple working definition for a compartment syndrome is an increased pressure within a closed osteofascial space which reduces the capillary blood perfusion below a level necessary for tissue viability. This situation may be produced by two overall mechanisms. The first is an increase in volume within a closed space, and the second is a decrease in size of the space.

An increase in volume occurs in a clinical setting of hemorrhage, post ischemic swelling, re-perfusion, and arterial-venous fistula. A decrease in size results from a cast that is too tight, constrictive dressings, pneumatic anti-shock garments, and closure of fascial defects. As the pressure increases in the tissue, it exceeds the low intramuscular arteriolar pressure causing decreased blood in the capillary anastomosis and subsequent shunting from the compartment.

The clinical conditions that may be associated with this problem include the management of fractures, soft tissue injuries, arterial injuries, drug overdoses, limb compression situations, burns, post-ischemic swelling, constrictive dressings, and tight casts.

The orthopaedic research group in San Diego [6--8,23--27] has performed excellent studies during the past several decades regarding this problem. They have found that the normal compartment pressure approaches 4 mmHg in the recumbent position.

Mubarak and Hargens [6--8,23,24,26,27] stated that an absolute pressure measurement of 30 mmHg in the compartment should be the critical pressure for occlusion of the intracompartmental microvasculature because this corresponds with normal capillary pressure. Their clinical and experimental studies have consistently implicated 30 mmHg as the critical ischemic pressure, and they have recommended fasciotomy if this pressure is reached or exceeded.

Matson [17--20] on the other hand, noted that as a compartment syndrome develops, capillary pressure must rise as the venous pressure rises within the compartment, inferring that normal baseline capillary pressure levels are not relevant to the process of microvascular occlusion. In a clinical study, he found that only patients with intracompartmental pressures of 45 mmHg or greater could be identified as having true compartment syndromes.

Whitesides [42--44] has found that a fasciotomy is required when the intracompartmental pressure approaches 20 mmHg below the diastolic pressure in any patient who has a worsening clinical condition, a documented rising tissue pressure, significant tissue injury, or a history of six hours of total ischemia of an extremity. Experimentally, ischemia has been induced in healthy muscle when the intracompartmental pressure rises to a level of 10 mmHg below diastolic pressure. In tissue that has been damaged by injury, the resistance to ischemia caused by increasing tissue pressure is decreased because perfusion of these tissues may not be as effective in preventing ischemia. In extremity injuries that result in complete ischemia, skeletal muscle has remained electrically responsive for up to three hours and survived for as long as four hours without irreversible damage. On the other hand, variable results occur after six hours of total ischemia and complete, irreversible changes occur after eight hours. It has been demonstrated that peripheral nerves are able to conduct impulses for one hour after the onset of total ischemia and can survive for four hours with only neuropraxic damage. However, eight hours of total ischemia result in axontomesis and irreversible changes in the nerve.

Summary of the University of Pennsylvania Studies

The investigative studies with regard to limb ischemia by the Department of Orthopaedic Surgery utilizing 31 phosphorus magnetic resonance spectroscopy (^{31}P-MRS) began in 1980 [2,11,14,32,34,35,39]. This resulted in a collaborative effort with Dr. Britton Chance in the Department of Biophysics and Biochemistry. The technique involved the noninvasive, nondestructive assay of cellular energy metabolism in relationship to adenosine triphosphate (ATP). Using this technique we were able to very accurately measure intracellular pH. In this manner, phosphorus spectroscopy was used as a metabolic study in comparison to proton imaging which was used as an anatomic studies typical of magnetic resonance imaging (MRI) investigations.

The initial study that was performed involved the investigation of muscle ischemia at various tourniquet times to provide an improved understanding of the safe limits of tourniquet application. This study was performed by Alex Sapega [34] when he was an research resident in our department. He demonstrated that safe tourniquet times were one and one-half hours followed by decompression of the tourniquet for a five to seven minute time interval, and reinflation for another one and one-half hours.

This was followed by a study of electromagnetic stimulation (EMS) of human muscle in training athletes [39]. Tetanic EMS provided a similar metabolic demand to that of conventional resistance exercise, and therefore, some similar training effects were anticipated. Non-tetanic EMS provided an entirely different acute metabolic demand, possibly allowing relatively increased blood flow during contractions.

The investigative studies relating to compartment syndrome considered the relationship between direct compartment pressure measurements and the systemic blood pressure. This involved the use of the mean arterial blood pressure which is estimated as the diastolic pressure plus one-third of the pulse pressure. We then coined the term Delta P (DeltaP) which is the mean arterial blood pressure minus the compartment pressure. This gave an index of net muscle perfusion. In essence, lower DdeltaP meant less muscle blood flow.

A study was then undertaken to compare the tolerance of skeletal muscle to ischemia with tourniquet application with acute compartment syndrome [12]. It was important to perform this study because several discussions of ischemic injury in compartment syndrome have frequently used tourniquet-induced ischemia as a patholphysiologic reference. Because there was no evidence that ischemia was the only cause of muscle cell injury in compartment syndrome, we felt that such references were misleading. It was our purpose to determine the differences, if any, in the metabolic and ultrastructural response of skeletal muscle in compartment syndrome compared with tourniquet-induced ischemia. It was noted that during ischemia, the cellular levels of phosphocreatine decreased at an identical rate in both groups. In contrast, the levels of ATP diminished rapidly in the animals with compartment syndrome, but remained unchanged in the tourniquet group. Ischemic muscle necrosis was more severe in dogs with a compartment syndrome than in tourniquet-induced ischemia. In the tourniquet group, the phosphocreatine, ATP, and pH were all normal within 15 minutes after release of the tourniquet, but these values remained depressed even two hours after fasciotomy in the group with compartment syndrome. Electromyographic (EM) studies revealed more severe cellular degeneration in the group with the compartment syndrome. This study unequivocally proved that elevated tissue pressure appears to act synergistically with ischemia to produce more severe cellular deterioration than does ischemia alone.

Another study [14] was undertaken to quantitate the cellular biochemical limits of muscle viability in a canine compartment syndrome during six hours of ischemia. We intended to provide information about the metabolic insult which occurs during a compartment syndrome as a function of time and perfusion pressures rather than as an isolated (critical) compartment pressure above which a fasciotomy is required.

The data from that study indicated the following.

1. Lower DeltaP results in a drop in the intracellular phosphocreatine ratio and pH.
2. At lower DeltaP, there is a decline in percentage recovery post-fasciotomy.
3. Blood pressure is extremely important for muscle survival and periods of hypotension may result in increased muscle damage at lower compartment pressures.

This study proved that lowering or raising the systemic blood pressure may affect the level of direct compartment pressure that results in ischemic compromise. This study further supported the notion that blood pressure was important in the development of compartment syndrome [42--44].

An experimental dog study as well as a clinical patient study [13] were utilized to study muscular energy metabolism with this new technique. The basic dog study investigated both traumatized and non-traumatized muscle with an induced compartment syndrome. The dog study revealed that the lowest DeltaP at which a normal cellular metabolic state can be maintained is approximately 30 mmHg in normal muscle and 40 mmHg in moderately traumatized muscle. The clinical patient series demonstrated that the threshold for cellular metabolic derangement in skeletal muscle subjected to increased tissue pressure was more closely associated with the difference between mean arterial blood pressure (MABP) and compartment pressure than with the absolute compartment pressure alone. We felt that it was imperative to interpret compartment pressure measurements in light of the degree of soft tissue trauma sustained and the patient's blood

pressure, as well as the clinical signs and symptoms.

A study was undertaken to explore the relationship between muscle tissue gases (pO_2 and pCO_2), blood flow, cellular pH and the bioenergetic (phosphorylation) state in experimentally induced compartment syndromes of two different degrees of severity, that is a DeltaP of 40 and a DeltaP of 20 mmHg. Hindlimbs subjected to compartment pressures of DeltaP = 40 mmHg demonstrated a 50% decrease in muscle oxygen tension and blood flow, but no evidence of even a partial shift to anaerobic metabolism was observed. In a DeltaP = 20 mmHg group, a minimal degree of anaerobic metabolism was detected in the presence of tissue oxygen tensions and blood flows approaching what have been cited in the literature as the critical anaerobic threshold levels for these parameters. Evidence of irreversible cellular changes were seen with the electron microscopic analysis of these specimens in the DeltaP = 20 group. Blunt muscle trauma before compartment pressurization sensitized the affected muscle making it more prone to metabolic and physiologic alteration by increased interstitial pressure. Compartment pressures within 20 mmHg MABP (DeltaP = 20) reduced tissue perfusion and oxygenation to compromise normal aerobic cellular energy metabolism in muscle. In this setting, cell death ensues. Surgical decompression at/or below a DeltaP of 40 mmHg appears to be a safe clinical guideline, with a wide margin of safety for both normal and modestly traumatized muscle. In severely traumatized limbs or in cases of unremitting and clear-cut symptoms of compartment syndrome, fasciotomy may be justified regardless of the tissue pressures documented.

A study was undertaken to establish a correlation between the presence of exertional pain and elevated compartment pressure and was further designed to answer the following questions.

1. Is ^{31}P-MRS an effective noninvasive tool for the diagnosis of chronic exertional compartment syndrome?
2. What is the role of ischemia in secondary metabolic compromise and the pathophysiology of exertional compartment syndrome?
3. What affect does fasciotomy have on the metabolic balance of the compartment musculature as determined by ^{31}P-MRS?

A total of 26 patients were enrolled in the initial study. Resting levels of high-energy compounds were assessed before insertion of a Slit catheter. Resting compartment pressures were recorded. A tourniquet was applied to the proximal thigh. While in the MRS, patients were asked to complete three consecutive 20-second isometric contractions of the affected compartment with a thigh tourniquet inflated to 150 mmHg higher than the resting blood pressure. This was performed to establish the ability of the patient to deplete phosphocreatine. Recovery was then permitted following tourniquet deflation and was documented by the return of phosphocreatine to resting levels. The patient was then removed from the magnet and was positioned on a treadmill. Treadmill speed was increased to the usual running pace of the patient. When the patient experienced pain, exercise was continued until the pain became intolerable. At that time the tourniquet was inflated to "freeze" the bioenergetic state and the patient was immediately repositioned in the MRS. A one-minute scan was performed and then the tourniquet was deflated and measurements were taken until resting ratios had been reattained. Diagnosis of compartment syndrome was made when the post-exertional compartment pressure failed to return to resting levels within six minutes of the termination of exercise.

The findings in this study verify that ischemia can and does occur in chronic exertional compartment syndrome but only in those situations in which the

intracompartmental pressures are exceptionally high (160 mmHg). It would seem, therefore, that the technique of dynamic metabolic monitoring provided by this technique was quite sensitive in reflecting tissue ischemia adequate to induce anaerobic metabolism. Furthermore, this premise seems to hold true whether the disturbance is increased intracompartmental pressure, vascular insufficiency, or enzyme deficiency. Our data also indicate that in most cases of chronic exertional compartment syndromes, the pain of the syndrome is not caused by the significant ischemia. We can only postulate that the increased pressure attained during exercise is responsible for stimulation of the pacinian corpuscles within the fascia and periosteom, thereby producing pain. This may also account for the relief of pain associated with fasciotomy. Furthermore, in the laboratory diagnosis of chronic exertional compartment syndrome, the rate of return to resting compartment pressure following exercise seems to be more accurate than reliance on the resting pressure alone.

A final study [2] involved the affect of antecedent ischemia on the tolerance of skeletal muscle to increased interstitial pressure. The results indicated that muscle subjected to six hours of antecedent ischemia demonstrated a lower tolerance to increased tissue pressure than otherwise normal muscle. In addition, once the pressure threshold of post-ischemic muscle was crossed, there was a more rapid rate of high-energy phosphate depletion than seen in normal muscle pressurized to the same degree below its DeltaP threshold. This has obvious implications for fascial release following arterial reconstruction in a traumatized extremity.

McQueen and Court-Brown [21,22] have made a prospective study of 116 patients with tibial diaphyseal fractures who had continuous monitoring of anterior compartment pressures for 24 hours. Their criterion for fasciotomy was a differential pressure (diastolic minus compartment pressure) of less than 30 mmHg. In the first 12 hours of monitoring, 53 patients had direct compartment pressures of more than 30 mmHg, and 30 had pressures of more than 40 mmHg; 4 had pressures of more than 50 mmHg. Only one patient had a differential pressure of less than 30 mmHg and he had a fasciotomy.

In the second 12-hour period, 28 patients had direct pressures of more than 30 mmHg and seven of more than 40 mmHg. Only two had differential pressures of less than 30 mmHg and they had fasciotomies. None of the total patient population of 116 tibial fractures had any sequelae of a compartment syndrome 6 months later. In our clinical patient series at the Hospital of the University of Pennsylvania (HUP) [13] we also had patients with elevated direct pressures greater than 300 mmHg but with DeltaP greater than 40 who did not receive a fasciotomy and did not develop compartment syndrome sequelae. Therefore, McQueen's data are very much in agreement with Whitesides and our recommendations regarding the relationship of systemic blood pressure and direct compartment pressure in recommending when to perform a fasciotomy. We reference mean arterial pressure and they reference diastolic blood pressure.

Diagnosis

In the past, the five "P"s (pulselessness, pallor, paralysis, paresthesia and pain) have been advocated as clinical signs of a developing compartment syndrome. In fact, these particular symptoms and signs are present during a well established compartment syndrome and the results of appropriate fasciotomies have not been consistently successful in maintaining limb function under these circumstances.

It has been demonstrated that pulses may be present during the development of a compartment syndrome. Because tissue perfusion in a compartment is dependent on arteriolar and capillary perfusion gradients, a compartment syndrome may result in the presence of peripheral pulses. If the pulses are absent, this indicates that there has been a significant decrease in the arteriolar and capillary perfusion gradients, which indicates a significant lack of flow through the muscle.

Pallor may not be present during a developing compartment syndrome as there is still enough flow to the distal portion of the extremity such that it is not present. The same is true for capillary refill. As long as there is some flow through the muscle, then capillary refill may not be grossly changed at the same time that muscle is undergoing significant changes during the developing compartment syndrome.

In a similar manner, paresthesia does not develop until there is significant compromise of flow, because nerve tissue is even more sensitive to ischemia than muscle. In fact, it has been demonstrated that nerve function ceases following two hours of total ischemia. It has also been demonstrated that pain corresponding to passive stretching of the muscles in a compartment in question is the most sensitive clinical finding in a developing compartment syndrome. This is one of the earliest signs of compartment syndrome that may be elicited in the examination of a particular patient. Obviously, palpation of the compartments is very important in demonstrating whether tension is present within the compartment. The opposite limb, if it is uninjured, may be a good reference point in determining whether there is fullness and tension in the compartment. Paresthesia develops as the compartment syndrome progresses.

Pain out of proportion to what is expected with the type of injury that the patient has sustained is another leading hallmark of a developing compartment syndrome. In fact, pain in the extremity, in addition to increased pain with motion of the muscles within the compartment, is the leading symptom and sign in a developing compartment syndrome. Obviously, if the extremity has been casted, then it is vitally important that the cast be bivalved and the surrounding soft dressings under the cast be removed so that all external compression of the compartment may be removed. This also allows the examiner to palpate the compartments for tension.

Compartment Pressure Measurements

The significance of measurement of compartment pressures is extremely important in a patient with a head injury. If the patient is unable to respond to pain and there is fullness of the compartment it is mandatory to either measure the compartments to rule out developing compartment syndrome or to perform a fasciotomy. The unconscious patient with a full compartment is the one true indication for the absolute need for compartment pressure measurements. In general, compartment pressure measurements are not as important as the clinical manifestations which have been outlined above. Rather, they are an adjunct in the evaluation of the patient. In addition to the patient with a head injury, they are important in patients who demonstrate some signs of developing compartment syndrome, but in whom it is difficult to determine whether a full-blown syndrome is present.

The San Diego group did extensive work measuring normal compartment resting

pressures which are less than 10 mmHg and advocated possible fasciotomy when patients reached direct compartment pressures of 30 or 40 mmHg. Matson, on the other hand, found a large series of patients with direct pressure measurements of 45 mm or less who did not develop a compartment syndrome, whereas patients with compartment pressures of more than 45 mmHg did develop compartment syndromes. Whitesides [42--44] has consistently advocated the diastolic pressure as a reference point in determining whether a fasciotomy is required. His recommendation is that a fasciotomy should be performed when tissue pressures rise to 20 mmHg below diastolic pressure. The studies of Whitesides and Matson coincide with our own in that we are advocating fasciotomies in relation to the systemic blood pressure as well as the direct compartment pressure.

Our technique, the DeltaP, relates to the mean arterial pressure which is the most frequent measurement utilized in vascular studies. However, the measurements are an adjunct to the clinical evaluation of a patient with a compartment syndrome. They should be taken as a reference in addition to the clinical evaluation, which we feel is the most important determination except in a patient with a head injury.

Tissue Measurement Techniques

These are four techniques that have been utilized for tissue pressure measurements in compartment syndrome. These include the Whitesides infusion technique, the Stic technique, the Wick catheter, and finally the Slit catheter technique.

The two most common techniques employed in clinical practice include the Whitesides technique or the Stryker Stic device.

The Whitesides infusion technique was a modification of an old technique used in the early 1990s to measure subcutaneous edema [43]. The beauty of the Whitesides technique [44] is that it uses materials that are available in any hospital in-patient floor or emergency room (Figure 1).

Fig. 1. 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 (Reproduced with permission from Thomas E. Whitesides, Jr., MD).

The equipment includes a mercury manometer (or alternatively, an electronic arterial pressure monitor with associated transducers), two plastic intravenous extension tubes, two 18-gauge needles, a 20-mL syringe, a three-way stop cock, and a vile of bacterio static normal saline. The technique involves prepping the extremity to be measured in the usual fashion. Saline is obtained from a sterile bottle. A 20-mL syringe is attached to a three-way stop cock. A single intravenous extension tube is attached and is then attached to a second 18-gauge needle. The third unused portion of the stop cock is temporarily closed off. The 18-gauge needle at the end of the extension tube attached to the stop cock is then inserted into the bottle of saline with the tip placed beneath the level of the saline. Saline is then aspirated into approximately half of the length of the extension tube. Next, the three-way stop cock is turned to the off position blocking loss of saline during the transfer of the needle from the saline bottle to the extremity of the patient. The second extension tube is connected to a three-way stop cock at its remaining open port and the other end is connected to the manometer. The syringe is aspirated with 15 mL of air with the stop cock remaining closed to the extension tube containing the saline. The syringe is then reattached. The saline-containing needle is then inserted into the muscle of the extremity to be tested. The stop cock is then turned in order to open the syringe to both extension tubes forming a T connection with a free column of air extending from behind the column of saline into the syringe and the manometer. In this manner, a system is created that allows air from the syringe to flow into both extension tubes as pressure within the system is increased in the process of measurement. The portion of the tube containing the top of the column of saline which is to be observed is positioned to the same level as the tip of the needle in the patient. It is important to maintain this portion of the tubing at the same level as the tip of the needle in the patient because raising or lowering the tube will reflect an artificial low or high reading. The examiner then depresses the plunger of the saline very slightly to inject a

minute amount which will ensure that the entire system and the tip of the needle are clear of any obstruction. A convex meniscus in the saline away from the patient is formed when the pressure in the tissue exceeds the pressure in the air column in the T-shaped system which is caused by capillary attraction. As the plunger is depressed, the saline meniscus will be altered from a convex configuration to a flat configuration when the air pressure in the system equals the interstitial pressure in the examined tissue of the patient. If the examiner depresses the plunger in a more firm manner, the saline meniscus will change from a convex to a concave configuration. This will obviously result in saline being injected into the examined tissue which is not beneficial. The examiner then reads the pressure on the manometer when the saline meniscus is flat. This situation occurs 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. If multiple readings are to be performed, the system is re-equilibrated while withdrawing on the syringe plunger until a reading of 0 mmHg is present on the manometer. This is done to prevent saline from being lost from the system during the withdrawal of the needle from the tissue. The system is now ready for another reading in a different location.

The Stryker Stic system is a hand-held unit, as depicted in the illustration (Figure 2), which is easily equilibrated and measurements are made directly from a scale on the device. The advantage of this type of device is that it is very portable, small in nature, capable of being held in the palm of the hand, and relatively inexpensive. We utilize this device in our hospital complex and have been satisfied with its performance.

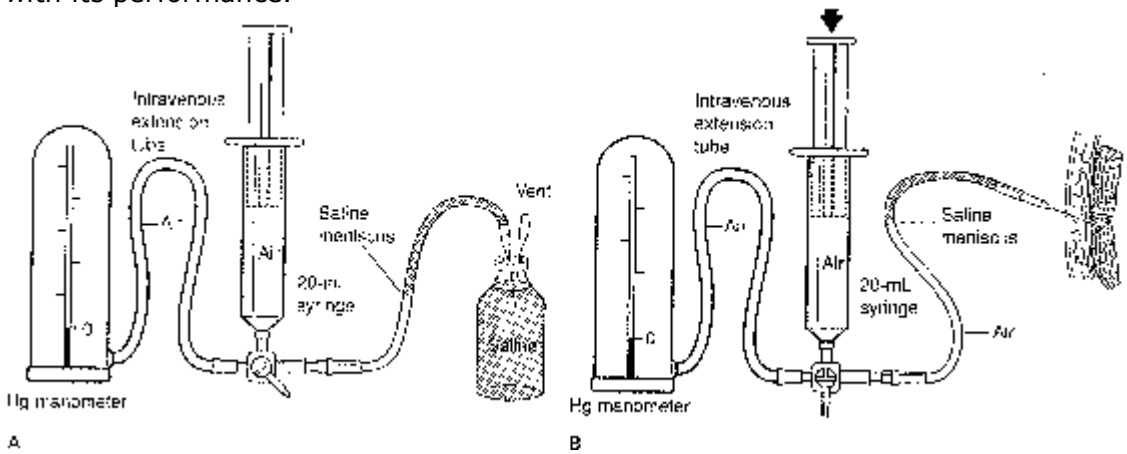

Fig. 2. Updated Compartment Syndrome (Heppinstall, RB).

Measurement Location

Heckman and Whitesides [10] published a prospective study of tibial fractures which documented the relationship between site of injury, the compartment involved, and the distance from the fracture site to the site of measurement in the leg. The highest tissue pressure was usually at the level of the fracture or within 5 cm of the fracture. Tissue pressures invariably decreased when sampled at increasing distances proximal and distal to the site of the fracture. This decrease in pressure was statistically significant. They recommend that measurements be taken at the level of the fracture as well as proximal and distal to the zone of the fracture. The highest pressure obtained is then referenced to the blood pressure in determining the need for fasciotomy.

Surgical Techniques

Surgical decompression for the most common areas of involvement, will be discussed in this section.

Forearm

The antebrachial fascia covers the superficial volar compartment containing the superficial digital flexors, flexor carpi radialis, flexor carpi ulnaris, pronator teres, and palmaris longus (if it is present). A deeper fascial layer is also present enveloping the flexor digitorum profundus, flexor pollicis longus, and pronator quadratus. To achieve complete compartment decompression, release of both the superficial and deep volar fascia is required. There are three main compartments in the forearm: the volar, dorsal, and mobile wad. It is important to realize that these compartments are more closely interconnected than the fascial compartments of the leg, because a release of the volar compartment alone may decompress the other two compartments. However, it is important to include measurements of the dorsal compartment and, if this is elevated, fasciotomy is indicated in this compartment as well. The volar exposure may be accomplished by one of three separate techniques [28,30]. These include a volar-ulnar approach, a volar approach, and a zig-zag approach. The volar-ulnar approach is preferred by many authors because it is very useful in maintaining soft tissue coverage over the flexor tendons on the ulnar side as well as the ulnar and median nerves. If the patient has signs and symptoms of median nerve involvement, exploration of the median nerve in the proximal forearm and decompressing the carpal tunnel are indicated. After the fascial release of the volar compartment, the pressure in the dorsal compartment is then remeasured. If the pressure is still elevated, a dorsal fasciotomy is required. However, cadaver studies have demonstrated that volar fasciotomy frequently decreases the pressure in the dorsal and mobile wad compartments. If a dorsal incision is required, it is usually a straight longitudinal incision originating 2 cm distal to the lateral epicondyle and a straight line distally to the mid-line of the wrist. The dorsal fascia is released directly. If a fasciotomy of the median nerve has been performed, it is important to cover this area by tacking the ulnar-based forearm flap over the nerve.

Lower leg

The lower leg has four compartments including the anterior, lateral, superficial posterior, and deep posterior. Occasionally, the tibialis posterior muscle will reside in a separate fascial compartment. Three separate techniques are advocated for decompression of these compartments. The two most commonly used techniques include a two incisional method and a single incision involving a perifibular approach. The final technique, that of performing a fibulectomy as outlined by Whitesides several years ago, has been discarded in favor of the perifibular approach.

Techniques

Perifibular approach

This technique is carried out through a straight lateral incision originating just posterior and parallel to the fibula at the level of the fibular head to a point above the tip of the lateral malleolus [42]. It is important to expose and protect the common peroneal nerve at the proximal end of the incision. Once this has been isolated, the dissection progresses through the deep tissues incising the fascia between the soleus and the flexor hallucis longus distally and is extended proximally to the soleus origin from the fibula. In this manner, access to the entire length of the superficial, posterior, and deep posterior compartments is accomplished. Exposure is important with this technique because it is more difficult to decompress the posterior compartment with this technique than from the medial side. It is important to ensure that the gastrocnemius, the flexor digitorum longus, flexor hallucis longus, and the tibialis posterior have been completely decompressed. Attention is then directed to the anterior portion of the incision which is mobilized and retracted to expose the anterior and lateral compartments. It is important to avoid the superficial peroneal nerve during its exit from the fascia of the lateral compartment traversing anterior in the distal third of the leg. It is also important to ensure that the tibialis anterior, extensor digitorum longus, and peroneus brevis have been thoroughly decompressed.

The shoe-lace type technique using vascular rubber loops along with skin staples has been a useful technique to gradually close large areas of gaping skin from the fascial release. This technique allows gradual closure of the skin over several days. In this manner, the size of the skin graft may be decreased or the necessity for a skin graft may be avoided and a secondary closure accomplished. We have recently [30] reviewed the histories of 27 patients with compartment syndrome treated with fasciotomies. Seventeen were closed by delayed primary closure and 10 by the technique of split thickness skin grafting. There was no difference in age, sex, mechanism of injury, associated injury, or fracture type observed. In particular, no difference was found between time of hospitalization to time of fasciotomy wound closure. However, analysis of post-closure in-patient hospital days demonstrated a significant ($p < 0.02$) difference between the treated cohorts. Patients undergoing delayed primary closure spent an average of 6--7 days in the hospital after wound closure. Patients who had split thickness skin-grafting techniques averaged an 11--12 day hospital stay. Therefore, in addition to avoiding a skin graft, the technique of delayed primary closure resulted in a decreased hospital stay which is important in these days of diagnosis-related group containment. However, this technique is only performed if there is no residual tension in the skin margins at the time of attempted secondary closure.

Double incision fasciotomy

Two vertical incisions with a bridge of skin greater than 8 cm are created. The first is between the anterior and lateral compartments. The second is 1--2 cm behind the posterior medial border of the tibia. A fasciotomy is then performed 1 cm in front of the intramuscular septum. A fasciotomy is created 1 cm behind the septum for the lateral compartment.

In the posterior medial incision, it is important to protect the saphenous nerve and vein, incising the fascia over the gastrocnemius muscle, exposing the deep

compartment fascia overlying the flexor digitorum longus and deep posterior compartment. This is exposed and incised. In this manner, both the superficial and deep posterior compartments are released.

The Foot

In the past, there have been five specific compartments requiring release in the foot. These include the medial, central, lateral, interossei and calcaneal compartments. This surgical technique requires three separate incisions to appropriately decompress the compartments.

The two parallel dorsal incisions are made over the second and fourth metatarsal shafts. The dorsal veins and the subcutaneous tissue are elevated laterally and medially to expose the respective interosseous musculature. The thin fascia over each of the interosseous areas is incised longitudinally. The first dorsal and plantar interossei are stripped from the shaft of the second metatarsal which is then retracted medially and the fascia of the adductor compartment is released longitudinally deep within the inner space. These two incisions have also been noted to help decompress the remainder of the foot but are not always sufficient for adequate decompression of the remaining compartments.

The hindfoot incision is located over the medial portion of the heel and begins 4 cm anterior to the posterior aspect of the heel and 3 cm superior to the plantar surface. The incision is made parallel to the bottom of the heel pad and is approximately 6--7 cm long. The incision is carried down to the fascia of the medial compartment. The subcutaneous tissue is elevated both in a superior and inferior direction which exposes the plantar aponeurosis. The medial compartment may be decompressed using the medial approach of Henry.

Recently, an alternative approach has been advocated by Manoli and Weber [15]. This approach involves a hindfoot incision over the medial portion of the heel. The incision begins 4 cm anterior to the posterior portion of the heel and 3 cm from the plantar surface. The incision is parallel to the bottom of the heel pad and is approximately 6--7 cm in length. The incision is carried down to the fascia wall of the medial compartment. The subcutaneous tissue is elevated both superiorly and inferiorly, which exposes the plantar aponeurosis. The medial compartment is then opened longitudinally with an incision 1 cm from its inferior border. The 1-cm fascial strip that remains can be followed laterally and serves as a landmark for the deeper incisions. The abductor hallucis muscle is then reflected superiorly and its attachment to the lateral fascial wall of the medial compartment is stripped. The fascia lying superior to the fascial strip is lifted and an incision is made in this fascia. Careful attention is paid not to damage the lateral plantar nerve and artery which lie just deep to this fascia. The incision is extended distally and decompresses the calcaneal compartment. The second deep incision is placed inferiorly to the fascial strip which projects along the entire length of the skin incision opening the superficial compartment. At its most proximal portion, the fascial incision provides access to the lateral compartment where the abductor digiti quinti muscle takes origin from the undersurface of the calcaneus. The lateral compartment is then released with an incision on the inferior-medial aspect of its overlying fascia, extending to the lateral side of the foot. Most surgeons have continued to describe the usual five compartments of the foot. However, Manoli and Weber have described a total of nine compartments. All of the compartments are released by the type of incisions discussed above [15].

Delayed and Late Treatment

Patients will occasionally present for treatment greater than 24--48 hours after the onset of an acute compartment syndrome. This creates a dilemma for the orthopaedic surgeon. There is no question that decompression of a missed compartment syndrome with necrotic muscle in the entire compartment is an excellent culture media for infection. This has been demonstrated in the past and it has generally been the policy not to rush in and decompress a compartment syndrome that has been missed by a time frame of greater than 24--48 hours. Under these conditions, particularly in the leg, it is best to refrain from an extensive decompression and allow the dead muscle to resorb with some scarring. In this manner, significant infection and possible amputation are avoided. It is rare to encounter a patient with severe total necrosis of the extremity and dry gangrene. The usual presentation is a patient with varying stages of muscle death with contracture and secondary deformity including paralysis of the distal musculature and loss of sensation in the nerve distribution that traverse the involved compartment. In the anterolateral compartment of the leg, it is beneficial to have fibrotic scarring occur within the muscle because this frequently acts as a check reign to prevent foot drop. It is unusual to benefit from any neurolysis of nerves as they traverse the involved compartment. Disastrous results may occur if decompression is attempted during the liquefaction stage of the dead muscle because this is an excellent culture medium and severe infections including clostridia with loss of limb and threat to life may ensue. It is more appropriate to allow fibrotic scarring of the muscles within the compartment and then attempt tendon transfers, depending on the muscles and compartment involved, including reconstructive procedures on the toes which may demonstrate clawing from missed posterior compartment syndrome. This type of reconstruction along with appropriate bracing will allow the patient to be ambulatory and functional with his own leg, which is obviously a better situation than a prosthetic replacement.

Recent Observations at Penn

We have recently encountered two specific clinical conditions that warrant discussion. The first of these is the development of a compartment syndrome in the well leg which is placed in hemilithotomy position during treatment of femoral fractures and nonunions in the injured leg [1]. In a review of the world literature, we were able to document 17 cases of compartment syndrome associated with hemilithotomy position [1,31]. In addition to these reports, we were able to obtain five patients in who we will be reporting this complication. Although very useful for the surgical treatment of hip and femoral fractures, the hemilithotomy well-leg position may cause a compartment syndrome through compartment compression and decreased arterial perfusion. This is particularly true in patients with heavy lower limbs. To avoid this complication, the hemilithotomy position should be avoided, if at all possible. If this position is required because of the size of the limb and inability to either lower the good limb or spread-eagle the patient, then a maximum time interval of 4--5 hours in this position should be allowed; the leg should then be lowered to a level position in an attempt to avoid this complication.

The second condition is the observation that rapid intravenous fluid resuscitation particularly with blood, can extravasate into tissue and produce a compartment

syndrome. This must be kept in mind during rapid resuscitations if this complication is to be avoided. We have identified two such cases in our institution.

I would like to end with a reminder of the possibility of this syndrome developing in all trauma patients that are evaluated; if in doubt, measure the pressures and use a DeltaP of 40 mmHg or less as your guide to fasciotomy; in nontraumatized muscle, a DeltaP of 30 mmHg or less.

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