Acute Limb Compartment Syndrome: A Review

Stavros Gourgiotis, MD, Constantinos Villias, MD,* Stylianos Germanos, MD,† Athanasios Foukas, MD,‡ and Marco Pericoli Ridolfini, MD§*

*Second Surgical Department, 401 General Army Hospital of Athens, Athens, Greece; †Upper GI and General Surgery, Leighton Hospital, Crewe, United Kingdom; ‡Wrightington Hospital, Wigan, United Kingdom; and §Department of Surgical Sciences, 'A. Gemelli' University Hospital, Rome, Italy

Acute limb compartment syndrome (LCS) is a limb-threatening and occasionally life-threatening condition caused by bleeding or edema in a closed muscle compartment surrounded by fascia and bone, which leads to muscle and nerve ischemia. Well-known causative factors are acute trauma and reperfusion after treatment for acute arterial obstruction. Untreated compartment syndrome usually leads to muscle necrosis, limb amputation, and, if severe, in large compartments, renal failure and death. Alertness, clinical suspicion of the possibility of LCS, and occasionally intracompartmental pressure (ICP) measurement are required to avoid a delay in diagnosis or missed diagnosis. Open fasciotomy, by incising both skin and fascia, is the most reliable method for adequate compartment decompression. The techniques of measuring ICP have advantages and disadvantages, whereas the pressure level that mandates fasciotomy is controversial. Increased awareness of the syndrome and the advent of measurements of ICP pressure have raised the possibility of early diagnosis and treatment. This review reports LCS, including etiology, pathophysiology, diagnosis, ICP measurement, management, and outcome. (J Surg 64:178-186. © 2007 by the Association of Program Directors in Surgery.)

KEY WORDS: compartment syndrome, intracompartmental pressure, limb, fasciotomy

COMPETENCY: Medical Knowledge

INTRODUCTION

Acute limb compartment syndrome (LCS) is a surgical emergency and is caused by raised pressure within a closed fascial space. Increase of the intracompartmental pressure (ICP) reduces the capillary perfusion below a level necessary for tissue viability.

Hippocrates[1] first described the dangers of raised ICP and its sequelae in 400 BC. Volkmann[2] recognized LCS in 1881, describing the contracture that is a common sequela. The first reported treatment was by Peterson in 1888,[3] followed by Jepson[4] who reported that venous obstruction may lead to muscle changes and demonstrated LCS experimentally in 1926. Sir Reginald Watson-Jones[5] attributed the pathology solely to arterial injury in 1952, whereas Whitesides et al[6] and Rorabeck[3] demonstrated the current concept that the increased ICP reduces the microcirculatory perfusion and leads to macrocirculatory arterial occlusion.

The limbs are enclosed in a deep fascial covering that divides skeletal muscle groups and the neurovascular bundles accompanying them into different compartments. Because of the unyielding nature of this fascial envelope, an increase in the ICP may reduce the capillary blood inflow, which eventually leads to arteriolar compression, followed by muscle and nerve ischemia with muscle infarction and nerve damage, if decompression is not performed promptly.

Limb compartment syndrome typically follows fractures, arterial injuries, extensive venous thrombosis, ischemic reperfusion injuries, crush injuries, burns, and prolonged limb compression after intra-arterial drug injection or patient malpositioning on the operating table. The most important determinant of a poor outcome from LCS after injury is delay in diagnosis.[3,7] The complications are usually disabling and include infection, contracture, deformity, and amputation. Continuous monitoring of ICP may allow the diagnosis to be made earlier and the complications to be minimized.[7,8]

In this review the etiology, pathophysiology, diagnosis, ICP measurement, management, and outcome of LCS are discussed. The literature about acute compartment syndrome of upper and lower limbs was identified with Medline, and additional cited works not detected in the initial search were obtained. Articles reporting on prospective and retrospective comparisons and case reports were included.

ETIOLOGY

Limb compartment syndrome may be caused by either an increase in volume within a compartment raising the pressure or

Correspondence: Inquiries to Stavros Gourgiotis, MD, PhD, 41 Zakinthinou Street, Papagou, Athens, 15669 Greece; fax: 30 210 6525802; e-mail: drsgourgiotis@tiscali.co.uk

1931-7204/07/$30.00
doi:10.1016/j.jsurg.2007.03.006

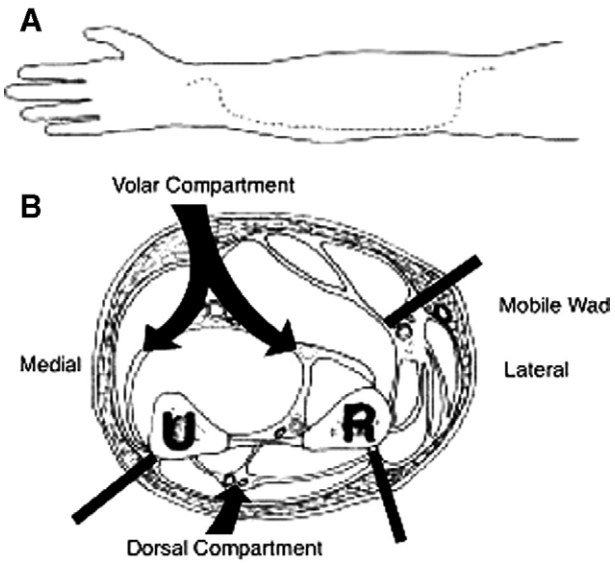

FIGURE 1. (A) Fasciotomy incision in the forearm. (B) Forearm compartments (R: radius; U: ulna).

externally applied pressure compressing a compartment. Each limb contains several compartments that are at risk for LCS. The upper arm contains the anterior (the biceps-brachialis muscle, and the radial, ulnar, and median nerves) and posterior (the triceps muscle) compartments. The forearm has both a volar and a dorsal compartment. The volar compartment contains the wrist and finger flexors, whereas the dorsal compartment holds the wrist and finger extensors. In the thigh, there are 3 muscle compartments (anterior, posterior, and medial), whereas there are 4 in the calf (anterior, lateral/peroneal, deep posterior, and superficial posterior). The anterior compartment of the lower leg is most frequently involved in this syndrome and contains the tibialis muscles and the extensors of the toes. Diagrammatic presentation of forearm, thigh, and calf compartments are shown in Figs. 1, 2, and 3, respectively.

Causes of LCS are summarized in Table 1.[9-11]

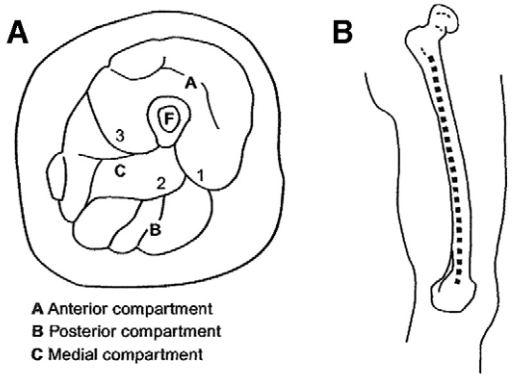

FIGURE 2. (A) Thigh compartments. (B) Fasciotomy incision in the thigh (F: femur; 1: lateral intramuscular septum; 2: posterior intramuscular septum; 3: medial intramuscular septum).

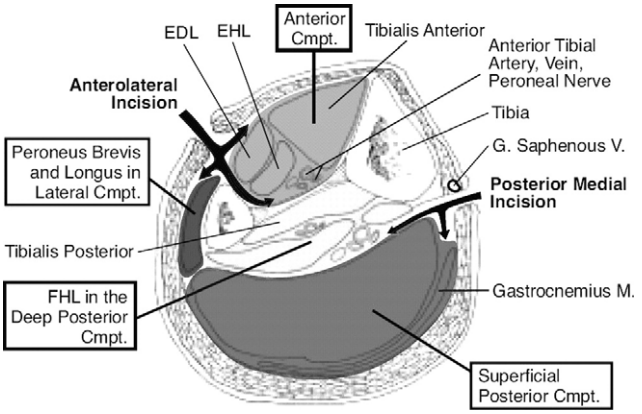

FIGURE 3. Calf compartments.

Orthopedic Causes (Fracture-Related)

Fractures of the tibial shaft, the distal radius, and ulna are the most common orthopedic causes of LCS.[3,7,12-14] The incidence of LCS after tibial fractures ranges widely from 1% to 29%.[7,8,12,14,15] The common belief that open fractures adequately decompress the compartments and hence prevent an increase in pressure is often incorrect. In fact, there is no difference in ICP between open and closed fractures; both types of fractures are likely to develop LCS.[8] In the leg the anterior compartment is most commonly affected, whereas in the forearm it is the flexor compartment.[7]

Vascular Causes

Both arterial and venous injuries can give rise to LCS.[16] Popliteal artery and vein injuries, particularly when a concomitant fracture occurs, often result in LCS.[17,18] If both artery and vein

TABLE 1. Common Causes of Acute Limb Compartment Syndrome[10-12]

Orthopedic (fracture-related)	Tibial fracture
	Distal radial and ulna fractures
	Supracondylar fracture of the humerus
	Femoral and calcaneal fractures
Vascular	Arterial and/or venous injuries
	Revascularization procedures
	Phlegmasia caerulea dolens
	Intra-aortic balloon pumping
	Isolated limb perfusion
Iatrogenic	Intravenous/intra-arterial drug injection
	Tourniquet
	Pneumatic anti-shock garment
	Hemophiliac
	Anticoagulation
	Extravasation of fluid after an arthroscopic procedure
	Prolonged surgery
Soft tissue	Crush injury without fractures
	Burn
	Oedema or intramuscular hematoma
	Drug or alcohol overuse induced stupor
	Snake bite

are injured, then more than 50% of patients require a fasciotomy presumably for increased compartment pressures.[19] Embolectomy, thrombolysis, and bypass surgery can cause LCS with an incidence that ranges up to 21%.[20-22] This condition is known as a post-ischemic compartment syndrome and is caused by revascularization of the leg after a prolonged period of ischemia.[20] The post-ischemic compartment syndrome is from reperfusion of the ischemic limb leading to tissue swelling and compartmental hypertension. An iliofemoral thrombosis causing phlegmasia caerulea dolens can produce a compartment syndrome.[23,24]

Iatrogenic Causes

Intravenous or intra-arterial administration of drugs has led to both forearm and leg LCS.[25] The use of a pneumatic anti-shock garment for severe hypotension can lead to LCS.[26] Prolonged surgery, especially in the Lloyd-Davies position, occasionally causes LCS,[27,28] which is caused by compression of the arterial inflow causing soft tissue necrosis, as a result of direct pressure.

Excessive anticoagulation with bleeding after major joint replacement has led to LCS.[29] Spontaneous bleeding in a hemophiliac is reported to result in LCS.[30] Inadvertent injection of pressurized fluid is associated with perioperative LCS.[31]

Soft-Tissue Causes

Swelling of the soft tissue without a fracture may occur after a direct blow to a muscle compartment or after a crushing injury, especially in patients with a coagulopathy or a known bleeding disorder.[13,32] In contusion injury, the continued use of the involved muscles may further increase ICP and increase bleeding leading to LCS; this may be the result of even minor injuries. Burns can also cause LCS by external compression of the compartment from burn eschar, which increases the ICP.[33]

The relationship between LCS and drug or alcohol overuse is well documented.[25] Intra-arterial injections of barbiturates and intravenous diazepam have been known to cause vascular damage, whereas drug addicts may suffer non-traumatic rhabdomyolysis leading to LCS.[34]

PATHOPHYSIOLOGY

In 1978, Matsen and Krugmire[35] first described the arteriovenous pressure gradient theory, which is the most popular hypothesis among the number of theories regarding the impairment of the microcirculation that occurs in an LCS. The theory suggests that ischemia begins when local blood flow cannot meet the metabolic demands of the tissue. Rising ICP increases the intraluminal venous pressures leading to a reduction in the arteriovenous pressure gradient with subsequent diminished or absent local perfusion. Reduced venous drainage increases interstitial tissue pressure, which allows tissue edema to form. Lymphatic drainage initially increases, but the growing tissue edema impedes lymphatic drainage. Eventually, arteriolar compression occurs, which leads to muscle and nerve ischemia. Sensory changes in the nerve, such as paresthesia and hypesthesia, develop after 30 minutes of ischemia. Irreversible nerve damage begins after 12 to 24 hours of total ischemia.[36] Irreversible functional changes start in the muscle after 4 to 8 hours.[37]

Post-ischemic compartment syndrome[38] is a consequence of a biphasic ischemia-reperfusion injury, in which tissue damage initiated in the ischemic phase is continued by the reintroduction of oxygenated blood.[39] The ischemia-reperfusion injury is mediated by the interaction of oxygen-derived free radicals, endothelial factors, and neutrophils.[40] Oxygen-derived free radicals can peroxidate the lipid component of cell membranes, which leads to enhanced capillary permeability.[41]

DIAGNOSIS

An initial high index of suspicious is required to make the diagnosis. A traditional hallmark element in the history of a patient presenting with LCS is pain disproportionate to the size of injury, which assumes a conscious and aware patient. Clinical suspicion should be heightened by the following clinical signs of LCS: pain, paresthesia, paralysis, pallor, and pulselessness.

Pain is different to the pain experienced by the initial trauma, which is produced by muscle ischemia and is only partially relieved with the usual analgesics used by surgeons for fractures. Therefore, pain out of proportion to that expected with the injury should provoke a strong suspicion of LCS.[42] An important early finding suggesting need for referral is pain on passive stretching or a compression of ischemic muscles in the compartment.[34]

Paresthesia results from a disordered cell membrane function in nerves running through the affected compartment, which may be an early sign of impending nerve ischemia. Anesthesia will result if the pathology is allowed to proceed. Both pain and anesthesia can only be assessed if peripheral nerve injury can be excluded. The end point of paresthesia is paralysis.

Pallor confirms lack of blood supply but is an unreliable sign. Pulselessness is common in arterial occlusion but otherwise only noted at a late stage.[43] Absence of pulses is indicative of direct arterial trauma, or of prolonged delay in making the diagnosis of raised ICP, with ICP rising to reach levels approximating mean arterial pressure, and occluding the main axial arteries. An LCS with an absent arterial pulse and no arterial trauma has a poor prognosis.

Seriously increased levels of creatinine phosphokinase (CPK) may indicate severe muscle damage, or ischemia. In the absence of clinical signs, it could indicate an unsuspected LCS. However, for early diagnosis, it is clearly not helpful.

INTRACOMPARTMENTAL PRESSURE MEASUREMENT

The measurement of ICP is only necessary when the clinical signs of compartment syndrome are unclear, in an unconsciousness or uncooperative patient, in a young child, or when the

clinical symptoms and signs are equivocal. The normal pressure in the muscle compartments is below 10-12 mm Hg.[44]

An 18-G needle is attached to an intravenous extension tube and then to a stopcock. Approximately half the tubing is filled with sterile saline, being certain that air is not allowed into the tubing. A second intravenous extension tube is attached to the 3-way tap and attached to the blood pressure manometer. The skin should be prepped with an antiseptic solution and infiltrated with local anesthesia at the prospective site. Only the subcutaneous tissue should be infiltrated. Intramuscular injection may artificially increase ICP. The needle is placed in the compartment, and the apparatus is kept at the level of the needle. The stopcock is then turned so that it is open in the direction of the intravenous tubing on either side of a syringe. The syringe filled with air is slowly compressed, which causes air to move into extension tubes. As soon as movement occurs in the fluid column, the ICP is read from the manometer. A particular problem of measuring ICP is to ensure that the correct size is chosen for the measurement. The highest pressure is thought to occur in the deep posterior and the anterior compartment in the leg, and when the catheter is close to a fracture, within 5 cm of the fracture line.[45]

Several techniques are available for ICP determinations, and those commonly used by surgeons are described below.

- The simple needle manometry technique.[46]
- The central venous pressure manometer technique.
- The wick catheter technique.[47]
- The slit catheter technique.[48,49]
- The side-ported needle technique.[50]
- The fibreoptic transducer.[51]
- The near-infrared spectroscopy (NIRS) is a noninvasive method that can measure the levels of muscle hemoglobin and myoglobin.[52]
- The laser doppler flowmetry[53] and the 99Tcm-methoxy-isobutyl isonitril (MIBI) scintigraphy.[54]

The Absolute ICP

Some debate exists in the literature regarding what pressure level mandates fasciotomy. Some investigators base recommendations based on absolute ICP,[1,55] whereas others feel the ICP is meaningful only as it relates to the mean blood pressure or diastolic blood pressure.[8,56] However, most recommendations are based on absolute ICP.

Pressure levels ranging exceeding 30 mm Hg,[3,12,57,58] 45 mm Hg,[59] and 50 mm Hg[60] have been proposed as the critical levels of pressures above which the viability of the compartment is compromised. Experimental studies have shown a big difference between individuals, when correlating absolute pressure levels, clinical signs, nerve function, and oxygen levels in the muscle tissue.[44] The lower level of 30 mm Hg is the most commonly used by surgeons. It is based on the theory that the capillary pressure is insufficient to maintain muscle capillary blood flow when the tissue fluid pressure is greater than 30 mm Hg, and in this pressure level, fascial compliance decreases sharply.[58]

The Critical Pressure

Whitesides et al[56] reported that the level of ICP at which ischemic compromise of muscle tissue occurs is related to the perfusion pressure. They used a difference between the diastolic pressure and the ICP to suggest that a differential pressure within 10-30 mm Hg of the diastolic pressure is the threshold for performing a fasciotomy.

Mubarak et al[47] used an ICP of more than 30 mm Hg as a basis of performing fasciotomy. They identified that the critical ICP of 30 mm Hg can be present only for a period of 6-8 hours before irreversible changes occur.[61]

Others used the "delta pressure." It is the diastolic blood pressure minus the ICP, and the most commonly cited delta pressure, which is currently used by surgeons to diagnose LCS, is less than or equal to 30 mm Hg.[7,8,36] In a study that applied this "delta pressure" for surgical intervention, it was demonstrated that many unnecessary fasciotomies were avoided, whereas patients who had developed an increase in ICP sufficient to cause obvious tissue compromise as observed at the time of fasciotomy were identified correctly.[8]

Finally, Mars and Hadley[62] reported that, by using the mean arterial pressure minus the ICP, greater accuracy can be achieved. They found that a differential pressure of 30 mm Hg or less indicated the need for fasciotomy in children.

The Time Factor

A dynamic relationship exists among the blood pressure, the level of ICP, and the duration of time for which a raised pressure is maintained. It is known that the higher the pressure, the faster and greater is the damage, but a lower pressure maintained for a longer period of time may also cause similar tissue damage. Many authors[7,14] concluded that catastrophic clinical results were inevitable if fasciotomies were delayed for over 12 hours, whereas a full recovery was achieved if decompression was performed within 6 hours of making the diagnosis.

MANAGEMENT

The goal of treatment of LCS is to decrease tissue pressure, restore blood flow, and minimize tissue damage and related functional loss. External pressure should be released by removing any cast, splint, or occlusive dressing that may lower the ICP. If a cast is bivalved, the ICP may decrease as much as 55%, and if a cast is completely removed, the pressure may decrease as much as 85%.[62-65] Elevation of the limb is sometimes used by surgeons as a temporary measure in an attempt to reduce ICP, but this has no effect and may even increase the pressure in both upper and lower limb syndromes.[59,66] However, in

phlegmasia caerulea dolens, early aggressive limb elevation is very important.[23]

Surgery

The treatment of LCS requires expedient fasciotomy. If left untreated, increased tissue pressure leads to muscle mass damage resulting in myoglobinuria, renal failure, metabolic acidosis, hyperkalemia, and ultimately contracture formation or loss of the limb.[64,65] Unfortunately, no definitive way exists to know when muscle, subject to an established LCS, is still viable, and when subsequent fasciotomy will prevent additional muscle necrosis. For this reason, irrespective of the length of time that susceptible muscle has been subjected to sustained compartmental hypertension, fasciotomy is advised once an LCS has been identified. According to Mabee,[67] absolute indications for fasciotomy are (1) clinical signs of LCS, (2) raised tissue pressure greater than 30 mm Hg in a patient with the clinical picture of LCS, and (3) interrupted arterial circulation to an extremity for greater than 4 hours. Adequate treatment is decompression of all 4 compartments.

In the forearm, volar fasciotomy is suggested,[3,50] but concurrent dorsal fasciotomy is also performed by some authors.[25,50] Fig. 1 shows the fasciotomy incision in the forearm. A complete forearm fasciotomy requires decompression of each nerve and muscle. If the clinical status of the hand remains unclear, the volar incision should be extended into the palm and the carpal tunnel should be released. For hand compartment syndromes, the carpal tunnel is always decompressed.[26]

In the thigh, all 3 compartments can be decompressed via a lateral incision (Fig. 2). To prevent muscle hernia, some authors[68] favor 2 parallel incisions in the fascia lata, with an intervening bridge of at least 4-5 cm. In the lower leg, the fasciotomy can be performed by a single lateral incision, with or without fibulectomy, or by a double incision technique, with medial and lateral incisions (Fig. 4).[69] The skin incision must be long enough to ensure that all underlying muscle is decompressed. The single incision technique allows adequate exposure of all 4 compartments.[70] Fibulectomy should be avoided, especially in patients with complex tibial fractures. The skin incision is made directly over the fibula. A transverse incision is made in the fascia to identify the intermuscular septum between the anterior and the lateral compartments and to identify the superficial peroneal nerve. Longitudinal fasciotomies of the anterior, the lateral, and the superficial dorsal compartments are then performed. The final step is to divide the origins of the soleus from the fibula and then to release the deep posterior compartment by incising directly behind the fibula.

When the double incision technique is used by a surgeon, the posterior compartments are opened from medially, and the anterior and lateral compartments from the lateral side. Advantages include the possible use of local anesthesia; there is no need for removal of bone, it is quicker, and it carries less risk of damage to neurovascular structures, with adequate decompression of the posterior compartment. The disadvantage is that it requires 2 separate incisions.

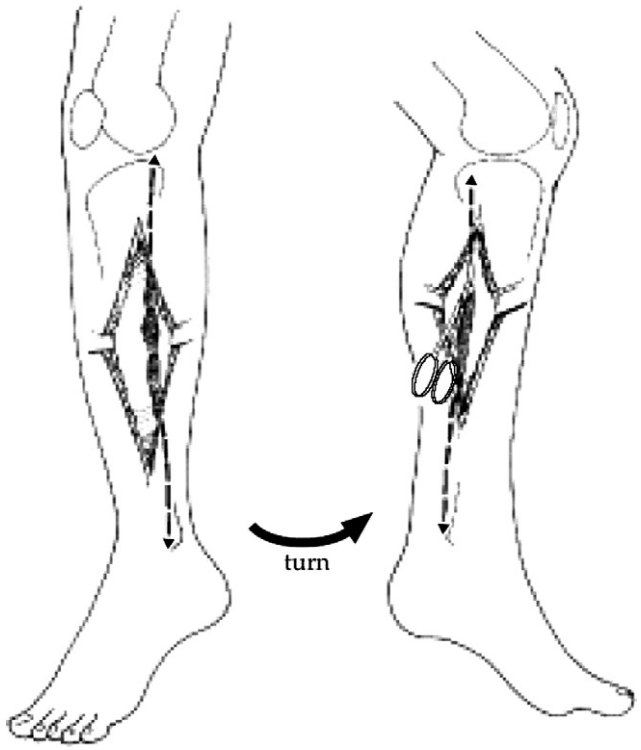

FIGURE 4. Fasciotomy incisions of the calf.

Pearse and Nanchahal[71] recommended 2 incisions for fasciotomies: the superficial and deep posterior compartments are decompressed through a medial longitudinal incision placed 1 to 2 cm posterior to the medial border of the tibia, and the anterior and peroneal compartments are decompressed by a second longitudinal incision placed 2 cm lateral to the anterior tibial border. Schepsis et al[72] recommended that, when doing a fasciotomy for exertional anterior LCS, a lateral compartment release is not necessary, because it increases morbidity and lengthens recovery. Patman[73] used multiple small skin incisions for decompression in fasciotomy for vascular causes. Leversedge et al[74] described endoscopically assisted fasciotomy for treating chronic exertional compartment syndrome in the lower leg.

The length of the fasciotomy wound, using a double-incision technique, was evaluated by Cohen et al.[58] A skin incision of 8 cm in length decreased the mean ICP. Mubarak et al[47] used an incision 15 cm long and found that the skin incision alone reduced the ICP by 5-9 mm Hg. The skin incision in the leg should be approximately 16 cm because small, subcutaneous, and closed incisions may not decompress the compartments fully.

The optimal method of wound closure after fasciotomy is not resolved. Early secondary wound closure after fasciotomy has been advocated for same patients on the third or fourth day after operation, but only with concurrent ICP monitoring because closing the wound even on the fourth day can still increases ICP.[75] Various techniques and devices have been pro-

posed to increase the rate of skin closure and reduce the need for skin grafting. These techniques and devices include intracutaneous skin sutures, skin stretching and mechanical closing devices, dynamic skin sutures, vacuum-assisted closure, and shoe lace suturing techniques.[76-78]

After fasciotomy, mortality rates of 11% to 15%, amputation rates of 11% to 21%, and serious morbidity have been reported.[42,64] Bermudez et al[79] found that patients who have undergone fasciotomy have poor calf muscle function. In a retrospective study by Heemskerk and Kitslaar,[80] poor correlation existed between prophylactic versus therapeutic treatments and clinical outcome, or between interval delay in fasciotomy and clinical outcome. Alternatively, Reis and Michaelson[81] suggest that when fasciotomy is delayed for more than 24 hours in patients with lower extremity crush injury suffered increased morbidity and mortality compared with those treated nonoperatively. Other reports note poor results when fasciotomy for LCS is delayed.[3,82] In a study by Sheridan and Matsen, the complication rates for early and late fasciotomized extremities were 4.5% and 54%, respectively. Almost one half of the late procedures reported by Sheridan and Matsen went on to amputation.[82] Finklestein et al[83] reported that fasciotomies performed after 36 hours from injury were invariably associated with severe infection, dismal rates of limb salvage, and even death.

The role of fasciotomy in cases of LCS that have been diagnosed at a late stage is questionable. Established myoneural deficits seldom recover after fasciotomy. Finklestein et al[83] recommended not performing fasciotomies in patients with established LCS more than 8 to 10 hours after injury. If performed within 12 hours of symptoms onset, fasciotomy can prevent most ischemic myoneural deficits and most patients can regain excellent function.[84,85] On the other hand, many patients have minimal limb dysfunction when fasciotomy has been performed promptly and to an adequate depth. Lagerstrom et al[86] reported that decompressive fasciotomy before the development of ischemic myoneural deficits prevents the ischemic sequelae of acute clinically evident LCS.

CONCLUSIONS

Limb compartment syndrome occurs when raised ICP causes tissue ischemia. It is a potentially limb-threatening and life-threatening problem after trauma and vascular injury to the limbs. A high index of clinical suspicion is required to diagnose this pathologic process. For early detection, it is necessary to educate those taking care of patients at risk, especially in the early symptoms and signs. Monitoring of ICP should be routine, particularly in patients in whom subjective clinical assessment is not available, ie, in unconscious, sedated, and uncooperative patients, and in those with injuries to the lower leg. Delay in diagnosis or treatment of LCS can result in significant morbidity for the patient. Full and extensive fasciotomies should be performed within 6 hours if the delta pressure remains less than 30 mm Hg and/or clinical symptoms and signs persist despite conservative measures. Although the morbidity of fasciotomies is significant, it is preferable to the outcome of a missed LCS.

REFERENCES

1. Mars M, Hadley GP. Raised intracompartmental pressure and compartment syndromes. *Injury*. 1998;29:403-411.
2. Volkmann R. Die ischaemischen Muskellahmungen und Kontrakturen. *Centralbl Chir*. 1881;8:801-803.
3. Rorabeck CH. The treatment of ccompartment syndromes of the leg. *J Bone Joint Surg Br*. 1984;66:93-97.
4. Jepson PN. Ischemic contracture: experimental study. *Ann Surg*. 1926;84:785-795.
5. Watson-Jones R. *Fractures and Joint Injuries*. 4th ed. Edinburgh: Livingstone ES; 1952.
6. Whitesides TE, Harada H, Morimoto K. Compartment syndromes and the role of fasciotomy, its parameters and techniques. In: American Academy of Orthopaedic Surgeons, eds. *Instructional Course Lectures*. St. Louis, MO: Mosby CV; 1977:179-196.
7. McQueen MM, Christie J, Court-Brown CM. Acute compartment syndrome in tibial diaphyseal fractures. *J Bone Joint Surg Br*. 1996;78:95-98.
8. McQueen MM, Court-Brown CM. Compartment monitoring in tibial fractures. The pressure threshold for decompression. *J Bone Joint Surg Br*. 1996;78:99-104.
9. Tiwari A, Haq AI, Myint F, Hamilton G. Acute compartment syndromes. *Br J Surg*. 2002;89:397-412.
10. Köstler W, Strohm PC, Südkamp NP. Acute compartment syndrome of the limb. *Injury*. 2004;35:1221-1227.
11. Choudhary RK, Waseem M, Thalva R, Dunlop DG. Acute compartment syndrome: diagnosis and immediate care. *Hosp Med*. 2003;64:296-298.
12. Blick SS, Brumback RJ, Poka A, Burgess AR, et al. Compartment syndrome in open tibial fractures. *J Bone Joint Surg Am*. 1996;68:1348-1353.
13. McQueen MM, Gaston P, Court-Brown CM. Acute compartment syndrome. Who is at risk? *J Bone Joint Surg Br*. 2000;82:200-203.
14. Mullet H, Al Abed K, Prasad CV, O'Sullivan M. Outcome of compartment syndrome following intramedullary nailing of tibial diaphyseal fractures. *Injury*. 2001;32:411-413.
15. Tornetta P 3rd, French BG. Compartment pressures during nonreamed tibial nailing without traction. *J Orthop Trauma*. 1997;11:24-27.

16. Shah PM, Wapnir I, Babu S, et al. Compartment syndrome is combined arterial and venous injuries of the lower extremity. *Am J Surg*. 1989;158:136-140.

17. Nair R, Abdool-Carrim AT, Robbs JV. Gunshot injuries of the popliteal artery. *Br J Surg*. 2000;87:602-607.

18. Abouezzi Z, Nassoura Z, Ivatury RR, et al. A critical reappraisal of indications for fasciotomy after extremity vascular trauma. *Arch Surg*. 1998;133:547-551.

19. Thomas DD, Wilson RF, Wiencek RG. Vascular injury about the knee: improved outcome. *Am Surg*. 1989;55: 370-377.

20. Shah DM, Bock DE, Darling RC, et al. Beneficial effects of hypertonic mannitol in acute ischemia-reperfusion injuries in humans. *Cardiovasc Surg*. 1996;4:97-100.

21. Clagett GP, Valentine RJ, Hagino RT. Autogenous aortoiliac/femoral reconstruction from superficial femoral-popliteal veins: feasibility and durability. *J Vasc Surg*. 1997;25:255-266.

22. Field CK, Senkowsky J, Hollier LH, et al. Fasciotomy in vascular trauma; is it too much, too often? *Am Surg*. 1994; 60:409-411.

23. Hamilton G, Platts A. Deep venous thrombosis. In: Beard JD, Gaines PA, eds. *Vascular and Endovascular Surgery*. London: Saunders WB; 1998:351-396.

24. Wood KE, Reedy JS, Pozniak MA, Coursin DB. Phlegmasia cerulean dolens with compartment syndrome: a complication of femoral vein catheterization. *Crit Care Med*. 2000;28:1626-1630.

25. Ouellette EA, Kelly R. Compartment syndromes of the hand. *J Bone Joint Surg Am*. 1996;78:1515-1522.

26. Aprahamian C, Gessert G, Bandyk DF, et al. MAST-associated compartment syndrome (MACS): a review. *J Trauma*. 1989;29:549–545.

27. Chase J, Harford F, Pinzur M, Zussman M. Intraoperative lower extremity compartment pressures. *Dis Colon Rectum*. 2000;43:678-680.

28. Perez RO, Pecora RA, Giannini CG, et al. Lower limb compartment syndrome associated with Lloyd-Davies/ lithotomy position in colorectal surgery. *Hepatogastroenterology*. 2004;51:100-102.

29. Nadeem RD, Clift BA, Martindale JP, et al. Acute compartment syndrome of the thigh after joint replacement with anticoagulation. *J Bone Joint Surg Br*. 1998;80:866-868.

30. Dumontier C, Sautet A, Man M, et al. Entrapment and compartment syndromes of the upper limb in haemophilia. *J Hand Surg Br*. 1994;19:427-429.

31. Seiler JG, Valadie AL, Drvaric DM, et al. Perioperative compartment syndrome. A report of four cases. *J Bone Joint Surg Am*. 1996;78:600-602.

32. Rooser B, Bengtson S, Hagglund G. Acute compartment syndrome from anterior thigh muscle contusion: a report of eight cases. *J Orthop Trauma*. 1991;5:57-59.

33. Perron AD, Brady WJ, Keats TE. Orthopaedic pitfalls in the ED: acute compartment syndrome. *Am J Emerg Med*. 2001;19:413-416.

34. Franc-Law JM, Rossignol M, Vernec A, et al. Poisoning-induced acute atraumatic compartment syndrome. *Am J Emerg Med*. 2000;18:616-621.

35. Matsen FA 3rd, Krugmire RB. Compartmental syndromes. *Surg Gynecol Obstet*. 1978;147:943-949.

36. Matsen FA 3rd. Compartment syndrome: a unified concept. *Clin Orthop*. 1975;113:8-14.

37. Whitesides TE, Harada H, Morimoto K. The response of skeletal muscle to temporary ischemia: an experimental study. *J Bone Joint Surg Am*. 1971;53:1027-1028.

38. Perry MO. Compartment syndromes and reperfusion injury. *Surg Clin North Am*. 1988;68:853-864.

39. Beyersdorf F. Protection of the ischemic skeletal muscle. *Thorac Cardiovasc Surg*. 1991;39:19-28.

40. Grace PA. Ischaemia-reperfusion injury. *Br J Surg*. 1994; 81:637-647.

41. Duran W, Takenaka H, Hobson R. Microvascular pathophysiology of skeletal muscle ischaemia-reperfusion. *Semin Vasc Surg*. 1998;11:203-214.

42. Jensen SL, Sandermann J. Compartment syndrome and fasciotomy in vascular surgery. A review of 57 cases. *Eur J Vasc Endovasc Surg*. 1997;13:48-53.

43. Leach R, Hammond G, Stryker W. Anterior tibial compartment syndrome. Acute and chronic. *J Bone Joint Surg*. 1967;49:451-462.

44. Willy C, Sterk J, Volker HU, et al. Acute compartment syndrome. Results of a clinico-experimental study of pressure and time limits for emergency fasciotomy. *Unfallchirurg*. 2001;104:381-391.

45. Heckman MM, Whitesides TE, Grewe SR, Rooks MD. Compartment pressure in association with closed tibial fractures. *J Bone Joint Surg Am*. 1994;76:1285-1292.

46. Whitesides TE Jr, Haney TC, Harada H, et al. A simple method for tissue pressure determination. *Arch Surg*. 1975;110:1311-1313.

47. Mubarak SJ, Hargens AR, Owen CA, et al. The wick catheter technique for measurement of intramuscular pressure. *J Bone Joint Surg Am*. 1976;58:1016-1020.

48. Rorabeck C, Castle G, Hardie R, Logan J. Compartmental pressure measurements: an experimental investigation using the slit catheter. *J Trauma*. 1981;21:446-449.

49. Barnes MR, Gibson MJ, Scott J, et al. A technique for the long term measurement of intracompartmental pressure in the lower leg. *J Biomed Eng*. 1985;7:35-39.

50. Awbrey BJ, Stenkiewicz PS, Mankin HJ. Chronic exercise induced compartment pressure elevation measured with a miniaturized fluid pressure monitor: a laboratory and clinical study. *Am J Sports Med*. 1988;16:610-615.

51. Ovre S, Hvaal K, Holm I, et al. Compartment pressure in nailed tibial fractures. A threshold of 30mmHg for decompression gives 29% fasciotomies. *Arch Orthop Trauma Surg*. 1998;118:29-31.

52. Arbabi S, Brundage SI, Gentilello LM. Near-infrared spectroscopy: a potential method for continuous, transcutaneous monitoring for compartment syndrome in critically injured patients. *J Trauma*. 1999;47:829-833.

53. Abraham P, Leftheriotis G, Saumet JL. Laser doppler flowmetry in the diagnosis of chronic compartment syndrome. *J Bone Joint Surg Br*. 1998;80:365-369.

54. Edwards PD, Miles KA, Qwens SJ, et al. A new noninvasive test for the detection of compartment syndromes. *Nucl Med Commun*. 1999;20:215-218.

55. Heckman MM, Whitesides TE Jr, Grewe SR, et al. Histologic determination of the ischemic threshold of muscle in the canine compartment syndrome model. *J Orthop Trauma*. 1993;7:199-210.

56. Whitesides TE Jr, Haney TC, Morimoto K, Hirada H. Tissue pressure measurements as a determinant for the need of fasciotomy. *Clin Orthop*. 1975;113:43-51.

57. Vollmar B, Westermann S, Menger MD. Microvascular response to compartment syndrome-like external pressure elevation: an in vivo fluorescence microscopic study in the hamster striated muscle. *J Trauma*. 1999;46:91-96.

58. Cohen MS, Garfin SR, Hargens AR, Mubarak SJ. Acute compartment syndrome. Effect of dermotomy on fascial decompression in the leg. *J Bone Joint Surg Br*. 1991;73:287-290.

59. Chidgey LK, Szabo RM, Kolack B. Effects of elevation on nerve function in an acute upper extremity nerve compression model. *J Orthop Res*. 1989;7:783-791.

60. Allen MJ, Stirling AJ, Crawshaw CV, Barnes MR. Intracompartmental pressure monitoring of leg injuries: an aid to management. *J Bone Joint Surg Br*. 1985;67:53-57.

61. Hargens AR, Romine JS, Sipe JC, et al. Peripheral nerve-conduction block by high muscle-compartment pressure. *J Bone Joint Surg Am*. 1979;61:192-200.

62. Mars M, Hadley GP. Raised compartmental pressure in children: a basis for management. *Injury*. 1998;29:183-185.

63. Heppenstall RB, Sapega AA, Izant T, et al. Compartment syndrome: a quantitative study of high-energy phosphorus compounds using 31P-magnetic resonance spectroscopy. *J Trauma*. 1989;29:1113-1119.

64. Hoover TJ, Siefert JA. Soft tissue complications of orthopaedic emergencies. *Emerg Med Clin N Am*. 2000;18:115-139.

65. van Essen GJ, McQueen MM. Compartment syndrome of the lower limb. *Hosp Med*. 1998;59:294-297.

66. Styf J, Wiger P. Abnormally increased intramuscular pressure in human legs: comparison of two experimental models. *J Trauma*. 1998;45:133-139.

67. Mabee JR. Compartment syndrome: a complication of acute extremity trauma. *J Emerg Med*. 1994;12:651-656.

68. Köstler W, Strohm PC, Südkamp NP. Acute compartment syndrome of the limb. *Injury*. 2004;35:1221-1227.

69. Mubarak S, Owen C. Double-incision fasciotomy of the leg for decompression in compartment syndromes. *J Bone Joint Surg Am*. 1977;59:184-187.

70. Cooper G. A method of single-incision, four compartment fasciotomy of the leg. *Eur J Vasc Surg*. 1992;6:659-661.

71. Pearse MF, Nanchahal J. Acute compartment syndrome of leg. *Br Med J*. 2002;325:557-558.

72. Schepsis AA, Gill SS, Foster TA. Fasciotomy for exertional anterior compartment syndrome: is lateral compartment release necessary? *Am J Sports Med*. 1999;27:430-435.

73. Patman RD. Compartmental syndromes in peripheral vascular surgery. *Clin Orthop*. 1975;113:103-110.

74. Leversedge FJ, Casey PJ, Seiler JG, Xerogeanes JW. Endoscopically assisted fasciotomy: description of technique and in vivo assessment of lower leg compartment decompression. *Am J Sports Med*. 2002;30:272-278.

75. Wiger P, Tkaczuk P, Styf J. Secondary wound closure following fasciotomy for acute compartment syndrome increases intramuscular pressure. *J Orthop Trauma*. 1998;12:117-121.

76. Caruso DM, King TJ, Tsujimura RB, Weiland DE, Schiller WR. Primary closure of fasciotomy incisions with a skin-stretching device in patients with burn and trauma. *J Burn Care Rehabil*. 1997;18:125-132.

77. McKenney MG, Nir I, Fee T, et al. A simple device for closure of fasciotomy wounds. *Am J Surg*. 1996;172:275-277.

78. Simpson NS, Jupiter JB. Delayed onset of forearm compartment syndrome: a complication of distal radius fracture in young adults. *J Orthop Trauma*. 1995;9:411-418.

79. Bermudez KM, Knudson MM, Nelken NA, et al. Long-term results of lower-extremity venous injuries. *Arch Surg*. 1997;132:963-967.

80. Heemskerk J, Kitslaar P. Acute compartment syndrome of the lower leg: retrospective study on prevalence, technique and outcome of fasciotomies. *World J Surg*. 2003;27:744-747.

81. Reis ND, Michaelson M. Crush injury to the lower limbs: treatment of the local injury. *J Bone Joint Surg Am*. 1986; 68:414-418.

82. Sheridan GW, Matsen FA. Fasciotomy in the treatment of the acute compartment syndrome. *J Bone Joint Surg Am*. 1976;58:112-115.

83. Finklestein JA, Hunter GA, Hu RW. Lower limb compartment syndrome: course after delayed fasciotomy. *J Trauma*. 1996;40:342-344.

84. Bae DS, Kadiyala RK, Waters PM. Acute compartment syndrome in children: contemporary diagnosis, treatment, and outcome. *J Pediatr Orthop*. 2001;21:680-688.

85. Howard JL, Mohtadi NGH, Wiley JP. Evaluation of outcomes in patients following surgical treatment of chronic exertional compartment syndrome in the leg. *Clin J Sports Med*. 2000;10:176-184.

86. Lagerstrom CF, Reed RL, Rowlands BJ, Fischer RP. Early fasciotomy for acute clinically evident posttraumatic compartment syndrome. *Am J Surg*. 1989;158:36-39.