

Compartment Syndrome of the Foot

Compartment syndrome is a relatively common posttraumatic diagnosis in the forearm and lower leg. Although the existence of this syndrome in the foot has been documented, it is not a well-known entity. Because delays in recognition and treatment can lead to catastrophic complications, practitioners should be aware of both its existence and its presentation. The authors present a case report of pedal compartment syndrome and a discussion of the literature pertaining to its diagnosis and treatment.

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Compartment syndrome results when an elevation of tissue pressure within a nonyielding fascial compartment impedes vascular inflow. The consequences of this posttraumatic syndrome are related to the effects of prolonged ischemia on neuromuscular structures. Compartment syndrome sequelae range from transient neurologic compromise following short periods of ischemia to complete myoneural necrosis, fibrosis, ischemic contractures (Volkmans ischemic contracture or claw foot), or all three following long-term ischemia. Compartment syndrome in the foot is a podiatric emergency because delays in diagnosis and treatment can result in severe structural and functional deficits. Compartment syndrome of the foot has been discussed infrequently in comparison to the large volume of literature devoted to compartment syndromes of the lower leg and forearm (1-3). As in the case of leg and arm compartment syndromes, compartment syndrome of the foot occurs most commonly after severe trauma. Causes of compartment syndrome include fracture, contusion, bleeding disorder, burns, trauma, postischemic swelling after arterial injury or thrombosis, exercise, drug or alcohol overdose, and venous obstruction (4). There are often multiple fractures of the midfoot, rearfoot, or both, or a crushing component is present. The authors present a case report and discussion of compartment syndrome within this context.

Case Report

A 31-year-old healthy man arrived at the emergency department at approximately 6:00 p.m. after he had fallen from a height of about 10 feet and landed on concrete. He complained of pain and swelling in both feet and ankles, greater in the left ankle than in the right. Palpation of the left calcaneus revealed tenderness. The left ankle was tender and swollen and was found to have decreased ankle-joint range of motion and diminished pedal pulses compared with the right side. An x-ray film revealed a comminuted fracture of the left calcaneus with good alignment maintained (Fig. 1). No other injuries were noted on x-ray. After application of a compressive posterior splint in the emergency department, the patient was discharged to home with crutches (nonweightbearing on the left foot) and pain medications.

During the next several hours, the patient experienced increasing pain in the left foot. He returned to the emergency department at approximately 6:00 a.m. the next day with excruciating pain and considerable swelling of the left foot. This pain was refractory to 10 mg. of intravenous morphine sulfate plus 10 mg. of diazepam administered in the emergency department. Following podiatry consult, the patient was admitted to the hospital for observation to rule out a probable compartment syndrome of the left foot.

Physical examination of the left foot revealed severe swelling with tenseness of the skin (Fig. 2). The patient was alert and oriented but in moderate to severe stress secondary to pain in the foot. Paresthesia was noted in all digits and the distal portion of the left foot, with hypesthesia and anesthesia of the plantar portion. Passive extension or flexion of the digits elicited excruciating pain. Pedal pulses were noted to be intact on both sides and capillary refill was normal. No open lesions or fracture blisters were noted.

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Figure 1. Lateral radiograph showing fracture of the left calcaneus.

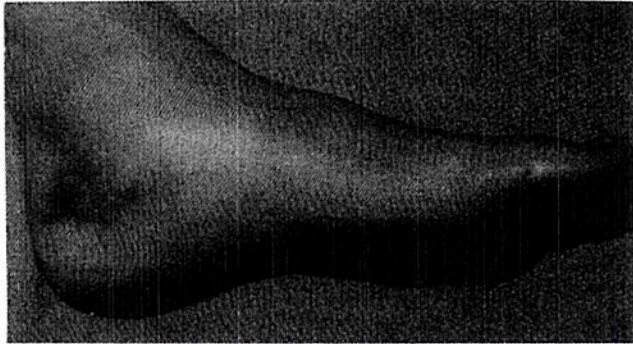


Figure 2. Severely swollen left foot before surgical decompression by fasciotomy.

An arterial-line monitor with an 18-gauge needle was used to evaluate compartmental pressures of the left foot. Pressure recordings taken from the central compartment of both feet revealed pressures of 75 mm. Hg in the left foot compared with 23 mm. Hg in the injured, but asymptomatic, right foot.

Laboratory studies on admission revealed a white blood count of 12.6×10^9 /liter with a normal differential, hemoglobin of 146 g/liter, hematocrit of 42.6%, and platelet count of 278×10^9 /liter. Partial thromboplastin time was 29.5 S (normal range, 29.9 to 35.5 S) and prothrombin time was 11.4 S (normal range, 10.9 to 12.9 S). Bleeding time on admission was greater than 15 min. (normal range, 1 min. 30 S to 9 min. 30 S). The patient denied having ingested any aspirin, nonsteroidal anti-inflammatory medications, or nonprescription medications that might contain aspirin. Before admission, the patient had taken no medications other than the acetaminophen with codeine prescribed in the emergency department for foot pain. Vital signs on admission included a blood pressure of 150/80 mm. Hg, pulse of 76 beats/min., and temperature of 36.1°C.

History and physical examination were consistent with a compartment syndrome of the left foot. The patient was taken to the operating room at approximately noon and decompression was undertaken using a long plantar medial utilitarian incision (1) (Fig. 3A).



Figure 3. A, a longitudinal plantar medial approach was used to decompress the plantar compartments of the foot. B, primary wound closure was not attempted intraoperatively.

An immediate reduction in the tenseness of the skin was noted. The wound was not closed primarily at the time of surgery (Fig. 3B).

The next 4 to 6 hr. following decompression revealed that the patient had a notable decrease in pain in the left foot. During the next 2 days there was progressive decrease in pain and swelling and a partial resolution of the digital paresthesia. Neurovascular status remained intact to the left foot. The patient was followed on an inpatient basis for the next 6 days, during which time he received intravenous antibiotics.

A hematology consult was obtained during hospitalization. Although the initial bleeding time was found to be 15 min., a repeat bleeding time was noted to be 5 min., which was within normal limits. The patient reported a previous incidence of oral bleeding following intubation for herniorrhaphy several years earlier; no bleeding was noted at the operative site. The patient noted occasional gum bleeding; however, previous dental extractions have not resulted in excessive bleeding. The patient works as a carpenter and has never noted unusual bleeding following trauma. He reported that his mother and sister have experienced heavy bleeding at time of delivery of their children. No male relatives were known to have bleeding disorders. Von Wille-

brand's antigen was 184% (normal), ristocetin cofactor was 104% (normal), and factor VIII was 81% (normal). Differential diagnosis per hematology included mild hemophilia (factor VIII deficiency), Type IIB Von Willebrand's antigen, primary platelet disorder, aspirin ingestion, and technical difficulty with bleeding time (the patient was moving, according to the technician).

Ten days following initial surgery, the patient returned to the operating room, where a split-thickness skin graft from the thigh was harvested to cover the residual defect on the medial aspect of the left foot following decompression of the compartment syndrome. The patient remained in the hospital for another week for observation and elevation; dependency of the foot was contraindicated because the patient was not allowed to be weightbearing. Neurovascular status was noted to be intact to the entire left foot and swelling was minimal at this time. The following day the patient was discharged home in a nonweightbearing, posterior splint with oral pain medication (hydrocodone).

Subsequently, the patient was followed on an outpatient basis in both the plastic surgery and podiatry clinics. There was an approximate 85% to 90% initial take of the split-thickness skin graft, and the wound was completely healed by 6 weeks (Fig. 4). The calcaneal fracture healed uneventfully. Painful neuropraxia noted several weeks postoperatively responded well to amitriptyline (25 mg. taken at bedtime). Neurologic status returned to normal during the next several months.

Discussion

Compartment syndrome of the plantar foot compartment has been infrequently described in the literature. Several authors (1-3) who have described this



Figure 4. Clinical appearance of the left foot following coverage of the wound with a split-thickness skin graft harvested from the thigh.

condition note findings consistent with compartment syndrome of the arm and leg. Most commonly, there is pain out of proportion for the acute injury that is not relieved by reduction, immobilization, or pain medication. Pain with passive dorsiflexion of the toes is an early indicator. After several hours of increased pressure and ischemia, signs of neurologic compromise such as numbness, burning, or paresthesias are apparent.

Other clinical signs of compartment syndrome can include a tense, indurated appearance of the skin over the compartment and a waxy or shiny character to the skin. Pedal pulses and capillary refill to the toes generally remain intact. The presence of a pulse distal to the closed compartment does not indicate that tissue perfusion is occurring in that compartment (5). In evaluating a possible compartment syndrome, it is important to avoid elevation of the extremity by maintaining it at heart level, which ensures that any circulatory compromise is not caused by elevation (1, 6). Pain caused by compartment syndrome may be exacerbated by elevation and sometimes diminished by dependency. This is because elevation decreases the intravascular pressure and thereby increases the discrepancy between tissue pressure and intravascular pressure, promoting closure of small vessels and increasing ischemia. Compartment syndromes may occur at lower pressures in hypotensive patients (7). The guidelines for tissue-pressure evaluation may need to be adjusted for variations in blood pressure. The position of the extremity during monitoring will also affect the tissue pressure.

The duration of pressure elevation is just as important as the magnitude of pressure elevation in producing neuromuscular deficits (6). Only 3 hr. of ischemia was sufficient to affect the capillary endothelium and give rise to postischemic swelling of 30% to 60% (8). Although skeletal muscle has been shown to remain functional for 3 hr. under total ischemia, nerve tissue shows reversible loss of function after 70 to 75 min. of total ischemia (5). According to Matsen (8), nerve tissue demonstrates functional abnormalities (paresthesia and hypesthesia) within 30 min. of the onset of ischemia and irreversible neurologic deficits are noted after 12 to 24 hr. of total ischemia. Motor function deficits occur later in compartment syndrome. Functional changes were noted after 2 to 4 hr. of ischemia and irreversible muscle deficits were noted after 4 to 12 hr. More than 12 hr. of ischemia were generally required to produce contractures (8). Hence, both the frequency and severity of complications are inversely related to the promptness with which decompression is achieved.

Microscopic changes in skeletal muscle caused by ischemic injury are also time related. Wiggins (9) noted damage to no more than 5% of muscle cells in dogs' legs after 4 hr. of total ischemia. After 8 hr., however,

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nearly 100% of muscle cells were damaged, and extensive damage was present after only 6 hr. Late microscopic changes in ischemically damaged cells ranged from no change to acute inflammatory changes and edema immediately following tissue reperfusion. Cell destruction and fibrosis were found several days after reperfusion in those limbs with ischemic damage. Regeneration was evident when ischemia was relieved before all cells were killed (10).

Clinically, in an acute compartment syndrome, the muscle fibers appear dull, gray, edematous, and friable, and lack normal bleeding (10, 11). Later, the muscle becomes frankly avascular, necrotic, and fibrotic. Response to electrical stimulation remains intact for approximately 3 hr. under total ischemia but may remain longer under relative ischemia (5).

In the case presented here, the patient arrived in the emergency department several hours after falling from a ladder and injuring both feet. An Essex-Lopresti tongue-type calcaneal fracture was noted on the left foot. There were soft tissue injuries to the right foot and ankle, but no fractures were noted. The patient was experiencing extreme pain in his left foot that was not relieved by intravenous morphine (10 mg.) and diazepam (10 mg.). The plantar portion of the foot was extremely tense and the skin had a waxy or shiny appearance. The patient related a history of relative pain relief with dependency of his left leg. Pulses and capillary refill were normal and there was excruciating pain with passive dorsiflexion of the digits. On the basis of clinical examination, a tissue-pressure measurement of the plantar foot compartments was obtained. Pressure in the left foot was 75 mm. Hg compared with 23 mm. Hg in the right foot.

Although both feet had sustained trauma, only the left side, which had a calcaneal fracture, developed a compartment syndrome, most likely caused by bony bleeding and inflammatory edema into a closed plantar space. This phenomenon may have been increased by abnormal bleeding tendencies in this patient. Although the patient had no diagnosed bleeding disorders, he had a family history of excess bleeding after trauma and had an elevated bleeding time (15 min.) when he arrived.

Several methods of measuring compartment pressure have been described in the literature (7, 12, 13) along with determination of normal compartment pressures and guidelines for fasciotomy. In the forearm and leg, the average compartment pressure is 4 mm. Hg (± 4) (12). Although no similar studies have been performed to determine normal compartment pressures in the foot, it is reasonable to assume these pressures would be similar. A study is currently in progress in our

podiatric surgery department to evaluate foot compartment pressures.

Whitesides *et al.* (5, 7) have described a method using an 18-gauge needle connected by saline-filled intravenous tubing to a manometer. The pressure required to inject a small amount of saline into the tissue is a measurement of compartment pressure. They found this method to be reliable and relatively easy to perform with practice. In spite of its relative simplicity, this method requires practice to gain competence. Also, injection of saline into a small compartment such as the central compartment of the foot could exacerbate symptoms if large amounts of saline are introduced (Fig. 5).

Mubarak *et al.* (12) described a modification of Whitesides' *et al.* (5, 7) technique that uses a wick catheter inserted into the tissue. They claimed better reproducibility and improved results when used for continuous monitoring. The wick was found to keep the catheter orifice open better and increased the surface area of contact.

A slit-catheter technique was described by Rorabeck *et al.* (13) and compared to both the wick and needle methods. They found the slit-catheter technique to be the most accurate, with a standard deviation of ± 2 mm. Hg with the slit technique compared to ± 5 mm. Hg with the needle technique.

Although "compartment syndrome" is largely a clinical diagnosis, compartment pressure measurements have proved to be good indicators of the necessity for decompression. Some advocate that all compartments with pressures >30 mm. Hg for 8 hr. (1) be decompressed. Because symptoms may not appear until damage already has occurred, clinical signs are relied on heavily to guide treatment. There is little dispute that pressures >40 mm. Hg pose great threat to neuromuscular structures. Whitesides *et al.* (7) advocates that compartment pressures of 20 to 30 mm. Hg be closely followed, with repeat measurements at 1- to 2-hr intervals, and that fasciotomy be performed when compartment pressures are within 10 to 30 mm. Hg of diastolic pressure. In a normotensive patient, the critical pressure would be 40 to 60 mm. Hg. Because diastolic pressure can be increased by pain from trauma, it would be dangerous to follow this recommendation strictly unless blood pressure readings could be obtained under baseline conditions.

In the face of clinically suspicious circumstances, pressure monitoring is definitely indicated. Fasciotomy should be performed when pressures approach that of diastolic pressure. Marginal pressures that are present for extended periods should also be considered for fasciotomy.

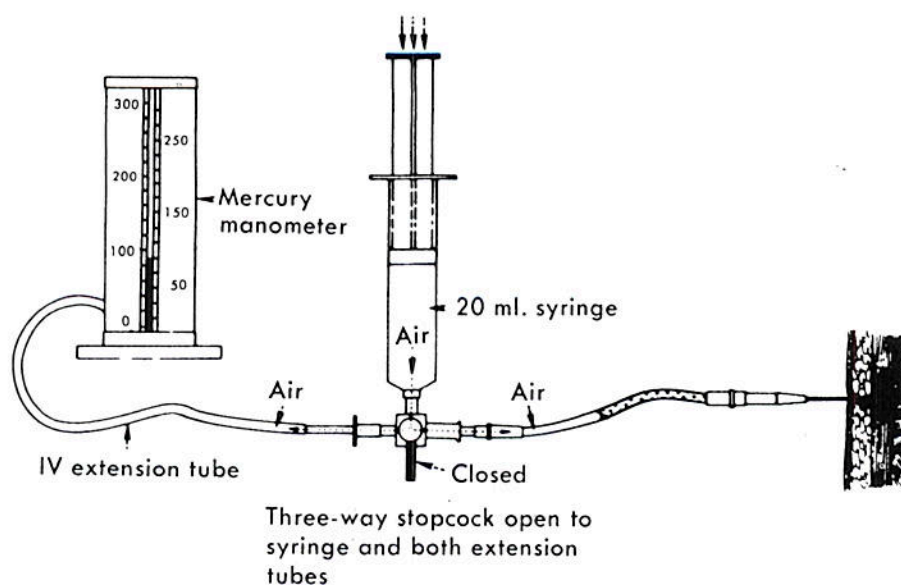


Figure 5. Needle manometer technique for measuring compartment pressures requires the injection of a small amount of saline into the closed compartment. The tissue pressure measured reflects the amount of pressure within the measuring system required to overcome pressure within the compartment. (From Whitesides, T. E., et al. *Instr. Course Lect.* 26:179-96, 1977 (5); reprinted with permission from C. V. Mosby.)

The authors' measurement technique involved an 18-gauge needle as a probe with an arterial line monitor as a pressure gauge. An intra-arterial line standard transducer (American Edward #53-DPS-260³) and tubing set were used. The needle was inserted into the center of the central plantar compartment. Central and interosseous compartment measurements were taken. Comparison values were obtained from the contralateral foot in similar fashion. This method required little practice and proved to be technically simple to perform, requiring only minimal amounts of saline (<1 ml./hr.) to be injected into the compartment. A standard deviation of ± 5 mm. Hg was expected from this technique, based on Whitesides' work. Previous authors (13) have shown the plain-needle technique to give elevated readings at pressures <50 mm. Hg and falsely lower pressures at 50 to 100 mm. Hg. Loss of accuracy during long-term monitoring has also been noted with the plain-needle technique (13). Neither of these drawbacks had an effect in this particular case, although they might in another instance. In these cases, a slit or wick catheter may be more appropriate. The method described, however, uses readily available supplies and equipment available in virtually all institutional inpatient and outpatient settings. Although the standard error for this method is ± 5 mm. Hg, the results from compartment pressure monitoring remain significant if taken in the

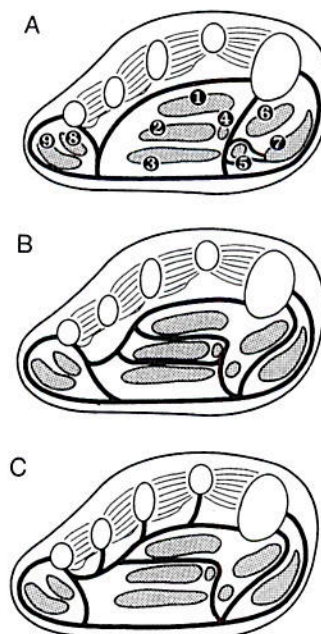


Figure 6. A-C, Compartments of the foot according to various authors. (From Goldman, F. D. *J.A.P.M.A.* 77:441, 1987; reprinted with permission from The American Podiatric Medical Association.)

context of a patient with clinically diagnosed compartment syndrome. In those patients whose pressures measure $30 \text{ mm. Hg} \pm 10 \text{ mm. Hg}$, close monitoring of clinical signs as well as pressures should be performed

³ American Edwards Laboratories, American Hospital Supply Company, Irvine, California 92714-5686.

and fasciotomy considered if improvement is not seen after several hours.

Treatment of compartment syndrome revolves around decompression of the closed fascial space (Fig. 6). Early fasciotomy is the best treatment. Prompt decompression can: 1) lead to less postischemic swelling, and 2) make infection less likely because there will be less devitalized tissue present (8). Sheridan and Matsen (14) showed that 68% of extremities decompressed within 12 hr. of the onset of symptoms regained normal function, while only 8% of those decompressed after ≥ 12 hr. had normal function. They also reported complication rates for the early and late fasciotomized groups of 4.5% and 54%, respectively.

Two methods to decompress the plantar foot compartments have been used and described: 1) Grodinsky (15) and Loeffler and Ballard (16) described a medial longitudinal incisional approach that involves an incision along the first metatarsal on the medial aspect of the foot. The central compartment is entered by passing between the first metatarsal shaft and the abductor hallucis muscle belly. The interosseous and lateral compartments are also easily accessible with this approach. A skin graft is commonly needed to close the wound. Decompression usually is achieved in approximately 1 min. using this method (Fig. 7); and 2) the two dorsal incision approach involves incisions over the second and fourth metatarsal shafts (1, 18). This approach provides exposure to all compartments but requires more time (11 min.) for complete decompression. It is advantageous in patients with midfoot fractures because reduction, and internal fixation can be achieved through these same incisions (Fig. 8).

In the authors' case, the medial approach was chosen. Decompression was achieved as noted by decreased tenseness of the skin intraoperatively and by a dramatic decrease in pain postoperatively. Paresthesias decreased over the postoperative course, and the patient had full recovery of function and sensation in 3 months. The medial approach provided good exposure to decompress the central, medial, and lateral compartments. A skin graft was later required to close the wound.

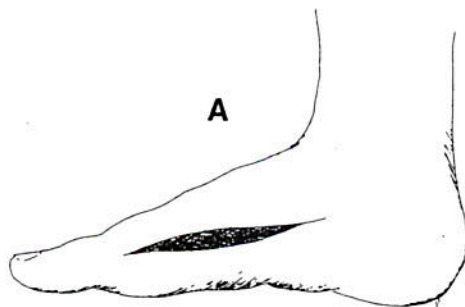


Figure 7. A and B, Fasciotomy of the foot using a medial longitudinal incision. (From Myerson, M. Bull. Hosp. J. Orthop. Inst., 47:260, 1987 (1); reprinted with permission from The Bulletin of the Hospital for Joint Diseases Orthopaedic Institute.)

Occasionally, skin becomes the limiting boundary in extremity swelling; this usually occurs when fasciotomy is attempted through limited skin incisions (17). In this case, a decompression dermatomy needs to be performed.

Debridement of muscle should be minimized at the time of fasciotomy. Any obviously necrotic muscle should be excised to help prevent scar formation (18). However, Matsen and Krugmire (19) noted that muscle which is noncontractile at the time of fasciotomy may still have good potential for recovery or regrowth. Eaton and Green (20) also contend that muscle necrosis is avoidable provided that circulation can be restored before fibrous replacement has occurred.

In the postoperative period, it is recommended that the limb be splinted and passive range-of-motion exercises be used to prevent development of contractures (6). By the third to fifth day following decompression, consideration can be given to skin closure. This may be accomplished by a delayed primary closure, or with reapproximation using tape skin closure. A skin graft ultimately may be required to cover the defect. Delayed

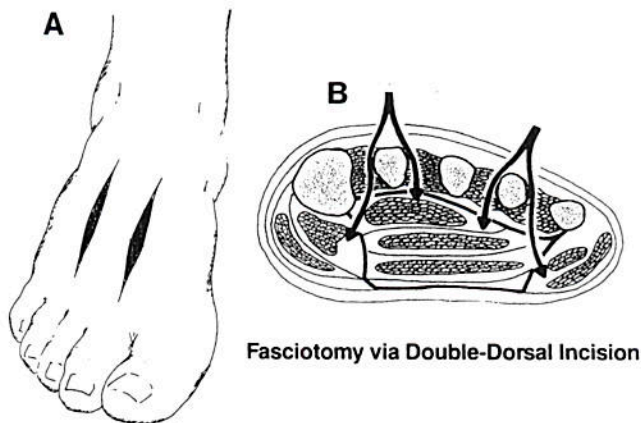
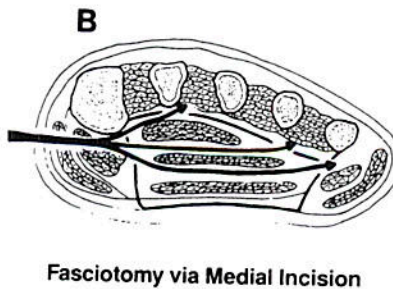


Figure 8. A and B, Fasciotomy of the foot using the two dorsal incision approach. (From Myerson, M. Bull. Hosp. J. Dis. Orthop. Inst., 47:259, 1987 (1); reprinted with permission from The Bulletin of the Hospital for Joint Diseases Orthopaedic Institute.)



Fasciotomy via Medial Incision

grafting has several important functions: 1) it permits inspection of the exposed muscle and debridement as necessary; 2) infection, if present, will be superficial; 3) it permits application of a split-thickness skin graft to a clean, granulating surface; and 4) it shortens the initial surgical procedure (17).

Included in the differential diagnosis for compartment syndrome are both nerve and artery damage. Precise diagnosis is critical because the treatments for each of these differ dramatically. Neuropraxia is treated by observation alone; arterial injury requires immediate operative repair; and compartment syndrome requires surgical decompression.

Summary

Compartment syndrome of the foot is an unusual, but certainly not rare, posttraumatic syndrome. Diagnosis depends on a good history and thorough clinical examination, as well as measurement of compartment pressures. Various methods of measuring compartment pressures of the foot have been described. Prompt recognition and treatment can help avoid the development of severe structural and functional problems.

Treatment of compartment syndrome revolves primarily around decompression of the closed fascial spaces. Early fasciotomy provides the best long-term prognosis for the patient because there is a decreased likelihood of residual neuromuscular deficits and a lower incidence of postoperative complications. Two different approaches for achieving decompression of the plantar compartment of the foot have been described in the text.

In the case presented, compartment syndrome of the foot was diagnosed following a calcaneal fracture. A medial longitudinal approach was used to decompress the plantar foot compartments. Because primary closure was not attempted, a split-thickness skin graft was later used to cover the wound. Both the skin graft and the calcaneal fracture healed uneventfully, and the patient has no residual neuromuscular deficits at this time.

References

1. Myerson, M. Acute compartment syndromes of the foot. *Bull. Hosp. Jt. Dis. Orthop. Inst.* 47:251-61, 1987.
2. Myerson, M. S. Experimental decompression of the fascial compartments of the foot—the basis for fasciotomy in acute compartment syndromes. *Foot Ankle* 8:308-14, 1988.
3. Bonutti, P. M., Bell, G. R. Compartment syndrome of the foot: a case report. *J. Bone Joint Surg.* 68A:1449-50, 1986.
4. Mubarak, S. J., Hargens, A. R. Acute compartment syndromes. *Surg. Clin. North Am.* 63:539-65, 1983.
5. Whitesides, T. E., Harada, H., Morimoto, K. Compartment syndromes and the role of fasciotomy: its parameters and techniques. *Instr. Course Lect.* 26:179-96, 1977.
6. Matsen, F. A., III, Winquist, R. A., Krugmire, Jr., R. B. Diagnosis and management of compartmental syndromes. *J. Bone Joint Surg.* 62A:286-91, 1980.
7. Whitesides, Jr., T. E., Haney, T. C., Morimoto, K., Harada, H. Tissue pressure measurements as a determinant for the need of

- fasciotomy. *Clin. Orthop.* 113:43-51, 1975.
8. Matsen, F. A. Compartmental syndrome: an unified concept. *Clin. Orthop.* 113:8-14, 1975.
9. Wiggins, H. E. The anterior tibial compartmental syndrome: a complication of the Hauser procedure. *Clin. Orthop.* 113:90-4, 1975.
10. Sanderson, R. A., Foley, R. K., McIvor, G. W. D., Kirkaldy-Willis, W. H. Histological response on skeletal muscle to ischemia. *Clin. Orthop.* 113:27-35, 1975.
11. Sarokhan, A. J., Eaton, R. G. Volkmann's ischemia. *J. Hand Surg.* 8:806-9, 1983.
12. Mubarak, S. J., Hargens, A. R., Owen, C. A., Gareto, L. P., Akeson, W. H. The wick catheter technique for measurement of intramuscular pressure. *J. Bone Joint Surg.* 58A:1016-20, 1976.
13. Rorabeck, C. H., Castle, G. S. P., Hardie, R., Logan, J. Compartmental pressure measurements: an experimental investigation using the slit catheter. *J. Trauma* 21:446-9, 1981.
14. Sheridan, G. W., Matsen, F. A., III. Fasciotomy in the treatment of the acute compartment syndrome. *J. Bone Joint Surg.* 58A:112-4, 1976.
15. Grodinsky, M. A study of the fascial spaces of the foot and their bearing on infections. *Surg. Gynecol. Obstet.* 49:737-51, 1929.
16. Loeffler, Jr., R. D., Ballard, A. Plantar fascial spaces of the foot and a proposed surgical approach. *Foot Ankle* 1:11-4, 1980.
17. Gaspard, D. J., Kohl, Jr., R. D. Compartmental syndromes in which the skin is the limiting boundary. *Clin. Orthop.* 113:65-8, 1975.
18. Mubarak, S., Owen, C. A. Compartmental syndrome and its relation to the crush syndrome: a spectrum of disease: a review of 11 cases of prolonged limb compression. *Clin. Orthop.* 113:81-9, 1975.
19. Matsen, F. A., III, Krugmire, R. B. Compartmental syndromes. *Surg. Gynecol. Obstet.* 147:943-9, 1978.
20. Eaton, R. G., Green, W. T. Volkmann's ischemia: a volar compartment syndrome of the forearm. *Clin. Orthop.* 113:58-64, 1975.

Additional References

- Andrews, J. R. Overuse syndromes of the lower extremity. *Clin. Sports. Med.* 2:137-48, 1983.
- Ashton, H. The effect of increased tissue pressure on blood flow. *Clin. Orthop.* 113:15-26, 1975.
- Goldman, F. D. Deep space infections in the diabetic patient. *J. A. P. M. A.* 77:431-43, 1987.
- Holden, C. E. A. Compartmental syndromes following trauma. *Clin. Orthop.* 113:95-102, 1975.
- Jepson, P. N. The classic: ischemic contracture experimental study. *Clin. Orthop.* 113:3-7, 1975.
- Kirby, R. L., McDermott, A. G. P. Anterior tibial compartment pressures during running with rearfoot and forefoot landing styles. *Arch. Phys. Med. Rehabil.* 64:296-9, 1983.
- Lapuk, S., Woodbury, D. F. Volkmann's ischemic contracture: a case report. *Orthop. Rev.* 17:618-24, 1988.
- Nogi, J. Common pediatric musculoskeletal emergencies. *Emerg. Med. Clin. North. Am.* 2:409-23, 1984.
- Patman, R. D., Compartmental syndromes in peripheral vascular surgery. *Clin. Orthop.* 113:103-10, 1975.
- Reneman, R. S. The anterior and the lateral compartmental syndrome of the leg due to intensive use of muscles. *Clin. Orthop.* 113:69-79, 1975.
- Rorabeck, C. H., Macnab, I. The pathophysiology of the anterior tibial compartmental syndrome. *Clin. Orthop.* 113:52-7, 1975.
- Rorabeck, C. H., Macnab, I. Anterior tibial-compartment syndrome complicating fractures of the shaft of the tibia. *J. Bone Joint Surg.* 58A:549-50, 1976.
- Sheridan, G. W., Matsen, III, F. A. An animal model of the compartmental syndrome. *Clin. Orthop.* 113:36-42, 1975.