Compartment Syndrome Of The Foot And Leg

A. Louis Jimenez, D.P.M. Mickey D. Stapp, D.P.M.

INTRODUCTION

Compartment syndrome is a condition in which increased tissue pressure in a closed fascial compartment reduces capillary blood perfusion below the level necessary for tissue viability. If the pressure remains sufficiently elevated for several hours, loss of function of muscles and nerves may occur. Myoneural necrosis will eventually result if treatment is not rendered. A variety of complications can result, depending on the length of time the compartment syndrome has been present. Ischemic contractures of the lower extremity and foot are a later finding, and can result in severe sequelae, ranging from a partial loss of function to a full-blown Volkmann's contracture. For this reason, early appropriate conservative and/or surgical treatment should be initiated.

Synonyms of an acute compartment syndrome include anterior tibial syndrome and Volkmann's ischemia. A Volkmann's ischemic contracture is the residual limb deformity which represents the last stage of muscle and nerve necrosis following an acute compartment syndrome.

A better appreciation and understanding of the histologic, biomechanic, and physiologic changes have been well espoused by Whitesides, et al., Hargens, et al., Szaba, et al., Heppenstall, et al., Matsen, et al., and Mubarak, et al. The excellent work done by these groups has led to a logical and sequential method of evaluating patients with a suspected compartment syndrome. They have also set parameters regarding the timing of, and necessity for fasciotomy.

HISTORY

The condition was first described by Von Volkmann in 1872 as an affliction of the upper extremity. A better understanding of the condition evolved with time, and involvement of the lower extremity and foot was recognized.

A variety of conditions can lead to a compartment syndrome, including arterial injury (iatrogenic and traumatic), thermal burn, crush injury, arterial injection, gunshot wound, osteotomy, embolectomy, snakebite, fracture, and acute and chronic exertional states. Other causes include circumferential dressings and casts.

PATHOGENESIS

A compartment syndrome usually results from muscle injury which causes increased pressure within the limited space of a myofascial compartment. Muscular trauma causes bleeding, resulting in edema. As the volume within a closed compartment increases, arterial circulation to tissues distal to this site becomes compromised, resulting in a compartment syndrome. This is a common occurrence with fractures. As hematoma formation ensues, volume increases within the closed space, thereby increasing the intracompartmental pressure. Tissue damage will depend on the amount of ischemia and duration of the event. Fracture of the tibia represents the most common cause of an acute compartment syndrome. A severe soft tissue injury, or contusion without fracture is the second leading cause of an acute compartment syndrome.

Intracompartmental tissue pressure is usually lower than the pressure of arterial blood flow. For this reason, peripheral pulses remain intact. Digital capillary beds drain into extracompartmental veins, therefore digital arteriovenous gradients and blood flow remain intact. Thus, peripheral pulses and digital circulation are poor indicators of the blood flow within a compartment.

Nerves will continue to conduct impulses for one hour after onset of total ischemia. Nerves may survive after four hours of ischemia, with resultant neuropraxic damage only. After eight hours of ischemia, axonotomesis ensues, resulting in irreversible neural damage. When trauma has been the inciting incident for a suspected compartment syndrome, trauma to a nerve may cause the neurologic symptoms. The cause of these neurologic symptoms must be differentiated between direct nerve trauma and that caused by the suspected compartment syndrome.

TISSUE HEMODYNAMICS

Ischemia is dependant upon the perfusion gradient of the tissue, which is directly related to the patient's blood pressure. Traumatized tissue, in order to meet its metabolic demands, requires more blood flow than normal. Therefore, this tissue has decreased resistance to ischemia. A pressure of 30-40 mm Hg below mean arterial pressure, or 10-30 mm Hg below diastolic pressure, has been shown to cause a significant decrease in tissue perfusion, resulting in ischemia. Since ischemia is based on the perfusion gradient of tissues, patients with a high diastolic pressure will be able to withstand a higher tissue pressure before ischemia ensues. Based on these findings, fasciotomy is indicated when: 1) The tissue pressure is 20-30 mm below diastolic blood pressure, 2) Pressures greater than 40 mm Hg for 20-30 minutes have been present, 3) Pressures greater than 30 mm Hg are combined with other positive clinical findings, and 4) Pressures between 30-40 mm Hg are not decreasing with monitoring every two to three hours.

SIGNS AND SYMPTOMS

Historically, pain, pallor, pulselessness, paresthesias, and paralysis have been the classical findings in a compartment syndrome. However, a patient with a compartment syndrome may not demonstrate all of these findings. The most impressive symptom with an acute compartment syndrome is pain out of proportion to the primary problem (fracture, soft tissue injury, etc.). Pain initiated by ischemia is not relieved by immobilization, and may be aggravated by a circumferential dressing or cast. Pain, which is aggravated by passive stretch of the affected tissues, is the most reliable physical finding. As tissue pressures approach 30-40 mm Hg (normal: 0-4 mm Hg) below a resting diastolic pressure of 70 mm Hg, passive stretch will result in increased pain. Other symptoms include edema, a feeling of tenseness in the involved compartment, and paresthesias of involved nerves. The earliest finding is a swollen, tense compartment which is a direct result of increased intracompartmental pressure. The skin may be shiny and warm. Even though the experienced examiner cannot determine intracompartmental pressure by palpation, the presence of a tense compartment throughout its boundaries suggests a compartment syndrome. When superficial subcutaneous edema is present, compartment tenseness may be difficult to ascertain.

As the compartment syndrome continues and ischemia is prolonged, further paresthesias, paralysis, and sensory changes ensue. As tissue pressure approaches diastolic pressure, capillary filling time increases. Pulselessness and pallor are rare unless significant arterial damage has occurred, or the artery passes through a compartment subjected to tissue pressures that approach the patient's systolic blood pressure. The prime indication for fasciotomy is the presence of the characteristic clinical symptoms and signs of a compartment syndrome, including deficits in neuromuscular function.

DIAGNOSIS

The differential diagnosis for a compartment syndrome includes fractures and contusions, cellulitis, and deep vein thrombosis. Fractures and contusions are less of a diagnostic dilemma. Cellulitis and deep vein thrombosis may show painful edema in the extremity. The former can be ruled out by signs and symptoms of infection. Deep vein thrombosis will exhibit calf pain and positive stretch pain, but neurovascular deficits are absent. Intracompartmental pressure measurements, doppler studies, and venography may be needed to make the appropriate diagnosis.

When the patient is conscious and able to respond to the physician's questions, the diagnosis is easier to make and/or monitor. However, in patients who are incoherent or unconscious, monitoring of the site is extremely important. Tissue pressure measurement may be the only mechanism by which the diagnosis can be made. In shock and hypotensive states, a slight increase in tissue pressure may be the only objective means of making the diagnosis.

TISSUE PRESSURE MEASUREMENTS

In a suspected compartment syndrome, tissue pressure measurements should not be limited to the immediate area of greatest symptoms. Tissue pressures need to be acquired for the entire extremity. Pressure measurements have been shown to vary significantly, both statistically and clinically, in areas as close as 5 cm apart. Even in open fractures, trauma to tissue results in segmental pressure gradients. Even though the immediate fracture site appears normal, irreversible tissue pressure increase may ensue. This can create segmental nerve and muscle injury, resulting in abnormal function distal to the fracture site.

When a fasciotomy is under consideration, tissue pressures should be acquired every one to two hours while observing the patient. A compartment syndrome may develop insidiously up to 72 hours after injury, and monitoring should be maintained for at least this period of time. Fasciotomy is performed after evaluation of a patient's signs and symptoms. If pain is progressively getting worse, in addition to fulfillment of the previously discussed criteria for fasiotomy, fasciotomy should be performed. The main objective of the fasciotomy is to create and maintain normal neuromuscular and musculoskeletal function. If a stabilizing or decreasing pressure is noted, conservative therapy may be indicated. Even after fasciotomy, multiple compartments must be measured to assure proper stabilization and lowering of intracompartmental pressures.

EQUIPMENT AND TECHNIQUES FOR MEASUREMENT

A variety of methods to measure compartment pressures have been described. These include Whiteside's Infusion Technique (Fig. 1), the Wick Catheter (Fig.2), the Slit Catheter (Fig. 3), The Stryker Stic Device (Fig. 4), Magnetic Resonance Imaging, and Intravenous Alarm Control (IVAC) Pump. Each has its own method of interpretation with which the examiner must be familiar. These pressure measurements must be clinically related to subjective and objective findings.



Figure 1. Whiteside's Infusion Technique.



Figure 2. Wick Catheter.



Figure 3. Slit Catheter.



Figure 4. Stryker S.T.I. C. Device.

TREATMENT

Approaches for treatment will depend on whether the patient has an incipient compartment syndrome or an acute compartment syndrome. An incipient compartment syndrome is defined as one which would become a full blown compartment syndrome if steps were not taken to prevent it. Examples include soft tissue injuries, overuse syndromes, closed LisFranc injuries, closed tibial fractures, and patients who complain of inordinate pain under a cast in which signs of a compartment syndrome are absent.

Non-operative steps utilized in a full blown compartment syndrome include removal of tight dressings and casts, maintaining systolic arterial pressure, and elevating or lowering the extremity to the level of the heart. This position maximizes tissue perfusion without compromising venous drainage. When conservative non-operative steps fail to diminish signs and symptoms or reduce tissue pressures, surgical fasciotomy/decompression is performed. Rorabeck emphasizes that there is no non-operative treatment that alone will alleviate a compartment syndrome. In approaching a fasciotomy, proximal involvement should always be ruled out. Appropriate vascular or orthopedic consultations are recommended.

DECOMPRESSION OF THE LOWER LEG

Anatomically, the lower extremity is divided into four compartments. These include the anterior, lateral, superficial posterior, and deep posterior compartments (Fig. 5). A compartment syndrome involving



Figure 5. Compartments of the lower extremity.

one of the four leg compartments is unusual, and in fact, two or more compartments are usually involved. Therefore, the technique used for decompression should allow access to all four compartments. There are a number of surgical approaches for decompression of the lower leg, including: 1) Fibulectomy, 2) Perifibular fasciotomy as described by Davey, Rorabeck, and Fowler, and 3) Double incision fasciotomy as described by Mubarak and Hargens.

Fibulectomy

Fibulectomy will undoubtedly decompress all four compartments. However, a fibulectomy is a radical procedure and generally not recommended. Advocates of fibulectomy as a decompressive procedure for all four compartments of the lower extremity suggest that it is indicated in patients with a severe compartment syndrome, particularly with involvement of the deep posterior compartment. Fibulectomy is contraindicated in children and in compartment syndromes secondary to fracture of the tibia.

Perifibular Fasciotomy

This approach has the advantage of allowing access to all four compartments through a single lateral incision. From a cosmetic standpoint, it is the method of choice. The incision is made over the lateral aspect of the fibula (Fig. 6). Following retraction and undermining of subcutaneous tissues anteriorly, access is gained to the anterior and lateral compartments. They are decompressed with individual fasciotomies. The posterior skin flap is retracted, and the fascia over the superficial



Figure 6. Perifibular Fasciotomy.

posterior compartment is incised. The plane between the peroneus longus and soleus muscles is followed down to the fibula, with care taken to avoid the peroneal nerve. The fibular origin of the soleus muscle is released through the proximal portion of the incision. The peroneal compartment is then retracted anteriorly. By retracting the superficial posterior compartment posteriorly, the deep posterior compartment is visualized. The fascia overlying the deep compartment is then incised along its entire length.

This is a viable technique as long as the anatomy of the extremity has not been distorted. In severe traumatic injuries where the normal anatomy has been distorted, the double incision technique is recommended.

Double Incision Approach

The double incision approach uses two vertical incisions. The anterior incision allows access to both the anterior and peroneal compartments. The second incision is placed on the posterior and medial aspect of the tibia, giving access to both the superficial and deep posterior compartments. The anterior incision is placed midway between the tibia and the fibula, and courses the length of the two compartments. Following subcutaneous dissection, the interval between the anterior and lateral compartments (anterior intramuscular septum) is identified, and two vertical fasciotomy incisions are made. The first incision is along the tibialis anterior, in front of the anterior intramuscular septum, and the second incision is posterior to the peroneus longus, toward the anterior intramuscular septum (Fig. 7A-C). Identification of the peroneal nerve is necessary to prevent trauma to



Figure 7A. Double incision Fasciotomy, posterior-medial incision.



Figure 7B. Double incision fasciotomy, anterolateral incision.



Figure 7C. Cross-sectional view of double incision fasciotomy.

this structure.

The posterior medial incision is placed 2.5 cm behind the posterior border of the tibia, and progresses the entire length of the compartment. The saphenous vein and nerve are identified and protected. The fascia overlying the gastrocnemiussoleus complex is initially incised and the muscle is retracted. Deep to this layer lies the flexor digitorum longus muscle. The fascia overlying it is incised, thus completing the fasciotomy of the deep posterior compartment of the leg.

The advantage of this technique is that it is simple to perform and there are very few neurovascular, muscular, or tendinous structures that can be injured, except for the saphenous vein medially and the peroneal nerves laterally. It allows selective decompression of one or two compartments as needed, with minimal soft tissue dissection.

DECOMPRESSION OF THE FOOT

The foot is anatomically divided into four compartments. The medial compartment contains the abductor hallucis and the flexor hallucis brevis muscles. The central compartment contains the adductor hallucis, quadratus plantae, flexor digitorum brevis muscles, as well as the tendons of the flexor digitorum longus and flexor hallucis longus muscles. The lateral compartment contains the flexor digiti minimi and abductor digiti minimi muscles. The fourth compartment, which is the interosseous compartment, contains both the dorsal and plantar interossei. A fifth compartment has been reported, which includes the quadratus plantae which is in communication with the deep posterior compartment of the lower leg (Fig. 8).



Figure 8. Compartments of the foot.

Tissue pressure measurements should be recorded for each plantar compartment, as well as each dorsal interosseous compartment. Decompression is accomplished with the use of three incisions (Fig. 9). Two dorsal incisions are used to release two interosseous compartments each. Access to the plantar compartments can be accomplished using either Henry's Approach, a curvilinear medial incision, or a standard medial incision.

Henry's approach starts at the medial aspect of the first metatarsophalangeal joint, courses toward the first metatarsocuneiform joint, and then curves plantarly over the talonavicular joint. This incision allows the surgeon to divide the foot into four layers. Henry's approach may also be extended proximally for release of the tarsal tunnel. The alternate approach to the medial compartment is through the use of a straight linear incision. Following subcutaneous dissection, the abductor hallucis muscle belly is mobilized from the plantar aspect of the first metatarsal, navicular, and underlying fibers of the flexor hallucis brevis. The Master Knot of Henry is exposed and incised. Care is taken to not injure the medial and lateral neurovascular bundles. After release of the Master Knot of Henry, the entire deep anatomy of the foot may be visualized. The first, second, and third layers of the foot may be retracted plantarly so as to follow the route of the medial and lateral plantar arteries and nerves (on the dorsal side of the third layer, Fig. 10). After plantar retraction of the third layer, the inferior fascia overlying the interosseous compartment may be visualized and decompressed. Interosseous release from the dorsal side, however, is preferred, as it allows easier access to and visualization of each of the individual



Figure 9. Incision placement for decompression of the foot. A. Standard medial incision, B. Henry's or curvilinear medial incision.



Figure 11. Decompression of the foot via the dorsal approach.

interosseous compartments (Fig. 11).

In open traumatic wounds, appropriate lavage and debridement is first performed. Skin and soft tissue margins which are not viable are removed. Individual lacerations of the wound may then be used as a site of access for decompression. This will eliminate the need for additionally placed incisions.

POSTOPERATIVE CARE

Postoperatively, the fasciotomy sites are packed open with sterile gauze, followed by a soft bulky dressing, placing the foot 90 degrees to the leg and at the level of the heart. Delayed primary closure with or without split thickness skin grafting is performed 48-72 hours later. When the compartment has necrotic muscle or muscle of questionable viability, the patient is returned to the operating room for debridement. The process is repeated until all



Figure 10. Decompression of the foot via the medial approach.

necrotic muscle is removed. The late result of secondary wound closure and skin grafting is quite satisfactory from a cosmetic standpoint of view.

LATE COMPLICATIONS

The degree of post-compartment syndrome complication is related to the time before diagnosis and treatment, and the compartment involved. The greater the nerve and muscle damage, the worse the prognosis. Without decompression or with incomplete decompression in a compartment syndrome, fibrosis of muscle occurs. Residual muscle function and deformity are directly related to the degree of muscle fibrosis.

A variety of residual deformities from a compartment syndrome may be seen, depending on the compartment(s) affected. In deep posterior compartment involvement, deformities can range from mild digital and metatarsophalangeal contractures to talipes equinovarus. Sensory loss to the plantar aspect of the foot and loss of innervation to the intrinsic musculature may result from tibial nerve damage.

Anterior compartment syndrome may result in a dropfoot. An equinus deformity may be seen with superficial posterior compartment involvement. Lateral compartment involvement may result in contracture of the peroneus longus, resulting in a plantarflexed first ray and heel varus. Loss of the peroneus brevis will allow overpowering of the antagonistic tibialis posterior muscle, and contribute to a cavus foot deformity.

A true Volkmann's or Dupuytren's contracture may result from tissue fibrosis caused by neuro-

muscular damage associated with a compartment syndrome. Treatment of such contractures centers around daily splinting and stretching exercises, bracing, orthotics, and shoe modifications. Surgical intervention may be necessary to correct severe contractural deformities. Each involved compartment is explored, and necrotic and fibrotic tissue is excised in its entirety. Neurolysis, as well as tendinous and osseous stabilizing procedures, are performed as necessary. In cases of a severe uncontrollable deformity or an insensate foot, amputation may be the treatment of choice.

SUMMARY

A compartment syndrome is a condition which the podiatric physician infrequently encounters. However, as more trauma and sports injuries are treated by the podiatric physician, this condition will be encountered more often.

Pain out of proportion to the involved surgery or trauma, and stretch pain, are the most reliable clinical signs and symptoms of a compartment syndrome. Paresthesias, paralysis, and sensory changes are relatively late findings. Pulses are generally palpable, and pallor is not generally present, in an ensuing or short-lived compartment syndrome.

It is extremely important that the podiatric physician be familiar with the different methods of measuring intracompartmental pressures. The decision of whether or not to perform foot and/or leg decompression will be based upon a sound understanding and evaluation of clinical signs and symptoms. Objective tissue pressure measurements, although not necessary to render emergency decompression, are a valuable study in helping to determine the timing and necessity of fasciotomy.

BIBLIOGRAPHY

- Amendola A, Rorabeck CH, Vellett D, Vezina W, Rutt B, Nott L: The use of magnetic resonance imaging in exertional compartment syndromes. Am J Sports Med 18:29-34, 1990.
- Bonutti PM, Bell GR: Compartment syndromes of the foot: a case report. J Bone Joint Surg 68A:1449, 1986.
- Bourne RB, Rorabeck CH: Compartment Syndromes of the Lower Leg. Clin Orthop 240:97-104, 1989.
- Clement JF, Pietrukso RG: Pit viper snakebite in the United States. J Fam Pract 6:269, 1978.
- DeLee JCU, Stiehl JB: Open tibia fracture with compartment syndrome. Clin Orthop 160:175, 1981.
- Goldman FD, Dayton PD, Hanson CJ: Compartment Syndrome of the Foot. J Foot Surg 29:37-43, 1990.

- Halpern AA, Nagel DA: Anterior compartment pressures in patients with tibia fractures. *J Trauma* 20:786, 1980.
- Hargens AR, Schmidt DA, Evans KL, et al: Quantitation of skeletal muscle necrosis in a model compartment syndrome. J Bone Joint Surg 63A:631, 1981.
- Hasman L: Volkmann's Ischemic Contracture. ACTA Chirug Plast, 27:26-41, 1985.
- Hayden JW: Compartment syndromes: Early recognition and treatment. Postgraduate Medicine, 74:191-202, 1983.
- Heckman MM, Grewe SR, Whitesides TE: Compartment syndromes of the foot. In Gould JS (ed): Operative Foot Surgery. Philadelphia, WB Saunders, pp. 568-578, 1994.
- Heckman MM, Whitesides, TE, Grewe SR: Spatial relationships of compartment syndromes in lower extremity trauma. Orthop Transactions, J Bone Joint Surg 11:537, 1987.
- Henry AK: *Extensile Exposure*, 2nd Ed. New York, Churchill Livingstone, 1982.
- Heppenstall RB, Sapega AA, Scott R, et al: The compartment syndrome: An experimental and clinical study of muscular energy metabolism using phosphorus nuclear magnetic resonance spectroscopy. *Clin Orthop* 226:155, 1988.
- Heppenstall RB, Scott R, Sapega AA, et al: A comparative study of the tolerance of skeletal muscle to ischemia: Tourniquet application compared with acute compartment syndrome. J Bone Joint Surg 68A:820, 1986.
- Lee BY, Brancato RF, Park IH, Shaw WW: Management of Compartment Syndrome: Diagnostic and Surgical Considerations. *Am J Surg* 148:383-388, 1984.
- Manoli, A II: Compartment Syndromes of the Foot: Current Concepts. Foot Ankle 10:340-344, 1990.
- Matsen FA III: Compartmental Syndromes. New York, Grune and Stratton, pp. 86-93, 1980.
- Matsen FA III: A practical approach to compartmental syndromes. Part I Definition, theory, and pathogenesis. AAOS Instr Course Lect, 32:88-92, 1983.
- Matsen FA III, Clawson DK: The deep posterior compartmental syndromes of the leg. J Bone Joint Surg 57A:34, 1975.
- Matsen FA III, Winquist RA, Krugmire RB: Diagnosis and management of compartment syndromes. J Bone Joint Surg 62A:28, 1980.
- McDermott AGP, Marble AE, Yabsley RH: Monitoring acute compartment pressures with the s.t.i.c. catheter. *Clin Orthop* 190:192, 1984.
- Meyerson MD: Experimental decompression of the fascia of the foot the basis of fasciotomy in acute compartment syndrome. Foot Ankle 6:308-314, 1988.
- Mubarak SJ, Carroll NC: Volkmann's contracture in children-etiology and prevention. J Bone Joint Surg 61A:285, 1979.
- Mubarak SJ, Hargens AR: Acute Compartment Syndromes. Surg Clin of N Am 63-3:539-565, 1983.
- Mubarak SJ, Hargens AR: Compartment Syndromes and Volkmann's Contracture. Philadelphia, WB Saunders, pp. 100-101, 1981.
- Mubarak SJ, Hargens AR, Owen CA, et al: The wick technique for measurement of intramuscular pressure. J Bone Joint Surg 58A:1016, 1976.
- Mubarak SJ, Owen CA: Double incision fasciotomy of the leg for decompression in compartment syndromes. J Bone Joint Surg 59A:184, 1977.
- Mubarak SJ, Owen CA, Hargens AR, et al: Acute compartment syndromes. Foot Ankle 6:308-314, 1988.
- Mubarak SJ, Owen CA, Hargens AR, Garetto LP, Akeson WH: Acute compartment syndromes: diagnosis and treatment with the aid of the wick catheter. J Bone Joint Surg 60A:1091, 1978.
- Naidu SI, Berquist VF: Compartment Syndromes in the Leg: A Surgical Emergency. Amer Surg May 1977, pp. 303-309.
- Nghiem DD, Boland JP: Four-Compartment Fasciotomy of the Lower Extremity without Fibulectomy: A New Approach. Amer Surg July 1980, pp. 414-417.
- Nkele C, Aindow J, Grant L: Study of Pressure of the Normal Anterior Tibial Compartment in Different Age Groups Using the Slit-Catheter Method. J Bone Joint Surg 70A:98-101, 1988.
- Rorabeck CH: The treatment of compartment syndromes of the leg. J Bone Joint Surg 66B:93-97, 1984.
- Rorabeck CH: A practical approach to compartmental syndromes. Part

III. Definition, theory, and pathogenesis. AAOS: Instr Course Lect, 32:102-113, 1983.

- Rorabeck CH, Castle GSP, Hardie R, Logan J: Compartmental pressure measurements: An experimental investigation using the slit catheter. J Trauma 21:446, 1981.
- Rorabeck CH, Macnab I: The Pathophysiology of the Anterior Tibial Compartment Syndrome. *Clin Orthop* 113:52-57, 1975.
- Shereff MJ: Compartment Syndromes of the Foot. AAOS: Instr Course Lect, 39:127-132, 1989.
- Smith GH: Measurement of the Intracompartmental Pressures of the Foot. J Foot Surg 29-6:589-591, 1990.
- Subotnick SI: Compartment Syndromes in the Lower Extremities. J Am Podiatry Assoc 65-4:343-348, 1975.
- Szabo RM, Gelberman RH, Williamson RV, Hargens AR: Effects of increased systemic blood pressure on the tissue fluid pressure threshold of peripheral nerve. J Orthop Res 1:172-178, 1983.
- Uppal GS, Smith RC, Sherk HH, Mooar P: Accurate compartment pressure measurement using the intravenous alarm control (ivac) pump. J Orthop Trauma 6:87-89, 1992.
- VonVolkmann R: Von Verletzungen und Krankheiten der Bewegungsorgane. Handbuch der Allgemeinen und Speziellen Chirurgie, 1872.