COMPARTMENT SYNDROMES

Diagnosis, Treatment, and Complications

Jorma Styf

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Preface

This book is for physicians, nurses, and physiotherapists who treat patients with injuries that are associated with acute compartment syndrome. It is also for physicians and staff personnel who see patients with chronic musculoskeletal pain induced by exercise. Despite the differences in the clinical appearance and etiology of acute and chronic syndromes, a common pathophysiological basis ties them together. Patients with acute compartment syndrome are seen within many medical fields, e.g., surgery, orthopedics, internal medicine, sports medicine, and psychiatry. Patients with chronic compartment syndrome are seen by physicians practicing sports medicine and by general practitioners. I hope that this book will facilitate the diagnosis and treatment of compartment syndromes. Most of the information in the book can also be used during basic training for medical students and nurses.

The work to combine the knowledge of others with my own on acute and chronic compartment syndromes has been stimulating. *Compartment Syndromes* is based on 1200 scientific publications. I am grateful to the many colleagues who shared their difficult patient cases of trauma and chronic pain with me. I hope my book will be helpful for both old and new colleagues. I gladly accept pedagogic and other comments on the book to improve the future mediation of knowledge on compartment syndromes.

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Acknowledgment

I would like to acknowledge the many colleagues who over the years shared their questions on patients with difficult musculoskeletal pain or complex trauma cases.

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Contents

1 Definitions and Terminology

INTRAMUSCULAR PRESSURE

Intramuscular pressure is defined as the fluid hydrostatic pressure in the interstitial space of skeletal muscle tissue. In a normally hydrated muscle at rest, this pressure ranges between 2 and 8 mmHg. Subcutaneously it is normally below atmospheric pressure. Intramuscular pressure depends on the positions of the subject and the catheter depth in the muscle.[1–3] Edema, external compression, passive stretch of a muscle and muscle contraction, or any combination of the four factors increases intramuscular pressure. Intramuscular pressure can be defined by the Starling equation (Equation 1.1),[4] which describes the magnitude and direction of fluid shift (Jc) over the capillary wall by the two hydrostatic and two osmotic pressures (Figure 1.1):

$$Jc = Kf\,[(Pc - Pt) - \beta(\pi_c - \pi_t)] \tag{1.1}$$

where Jc is the net fluid flow across the capillary wall, Kf the capillary filtration coefficient, Pc the capillary blood hydrostatic pressure, Pt the hydrostatic pressure in the interstitial fluid, β the capillary membrane reflection coefficient, πc the capillary blood colloid-osmotic pressure, and π_t the osmotic pressure in the interstitial fluid.

At steady state, the capillary net fluid flow is zero. In this situation, hydrostatic pressure in the interstitial space (P_t) can be expressed as:

$$P_t = P_c - (\pi c - \pi t)] \tag{1.2}$$

Pressure (P) may also be a vector expressed as force (F) per unit area (A). In this case, pressure has a magnitude and a direction:

$$P = F/A \tag{1.3}$$

Contact pressure (P) acts on a surface (A), e.g., pressure between the bone and an implant. Contact pressure and frictional forces require physical contact, a surface, to be transmitted. Forces (F) may also be transmitted through solid components of the tissues. Body forces such as gravity and electrostatic forces act without physical contact. Stress (Pascal) is the force (F) per unit area (m^2). It is a vector, because it has a magnitude and a direction. Solid tissues may become strained and deformed by forces. These forces are sometimes labeled "solid pressure."[5] In this book, "pres-

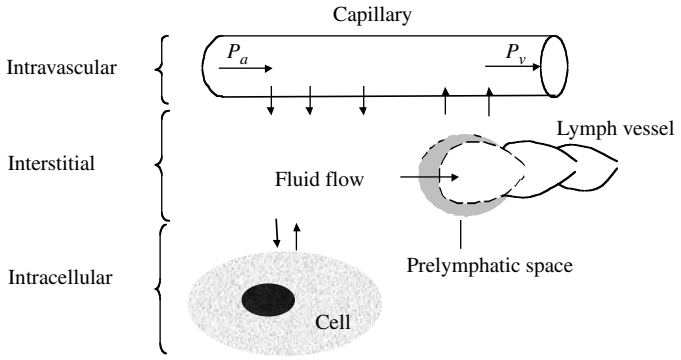

FIGURE 1.1 Schematic illustration of how the two hydrostatic (P_c and P_i) and two osmotic ($\prod_c$ and $\prod_i$) pressures act over the capillary wall. The area within the dotted line indicates the prelymphatic space. The drawing illustrates the central position of the interstitial fluid hydrostatic pressure (P_i) and its importance to direct fluid flow among the intravascular, intracellular, and interstitial spaces.

sure" is regarded to be a scalar-like fluid hydrostatic pressure. It has a magnitude but no direction.

Generally, pressure is defined as the force exerted per unit area. In clinical practice as well as in physiological experiments, pressure is expressed as millimeter of mercury (mmHg) or in kiloPascal (kPa). One kPa is ca. 7 mmHg. If it is assumed that the density of mercury is 13.6 g/cm^3, the density of blood is 1.06 g/cm^3, and that of physiological saline 1.04 g/cm^3, then 1 mmHg is equal to 12.9 mm blood or 13.1 mm saline, which compare at 0.14 kPa. The hydrostatic pressure of 1 mmHg at 0°C is 1330 dyn/cm^2 if the gravitational force is 0.981 m/sec^2.

In clinical practice, pressure is measured relative to atmospheric pressure. Some pressure transducers are sealed. Therefore, they must be recalibrated repeatedly against atmospheric pressure to avoid errors.

EDEMA

Edema is a sign of overhydration of the interstitial space and is caused by a disturbed microcirculation or dysfunctional lymph flow, or both.[6] Figure 1.2 illustrates the relation between the degree of interstitial hydration and interstitial fluid hydrostatic pressure. Abnormally increased intramuscular pressure for any reason during an extended period of time may jeopardize local tissue nutrition and viability. Patients might even experience ischemic pain from posttraumatic edema. As opposed to edema is the condition of dehydration. During dehydration, subcutaneous pressure decreases rapidly below atmospheric pressure and becomes negative.

COMPARTMENT AND COMPLIANCE

A compartment is a well-defined anatomical space, e.g., the anterior tibial compartment of the leg[7] or paravertebral muscles of the dorsolumbar compartment of the

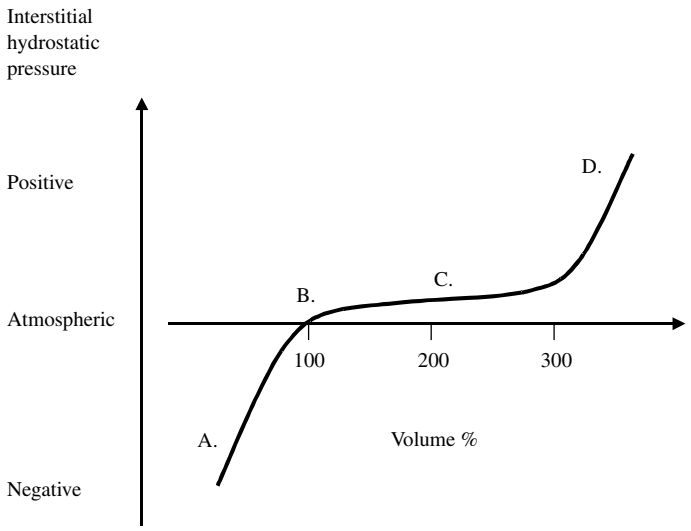

FIGURE 1.2 Relation between hydrostatic tissue pressure and volume in the interstitial space. The curve has three different slopes depending on the degree of tissue hydration. The compliances of the dehydrated (A) and normally hydrated (B) tissues are very low. On the contrary, at C the compliance is very high in the overhydrated tissue. Finally, when tissue hydration is excessive (D), compliance decreases again.

back.[8,9] Most often, the compartment tissues are contained in relatively unyielding osteofascial spaces (Figure 1.3).

Compliance of muscle tissue within a compartment describes the relation between volume and pressure in that space (Figure 1.4). Compliance (*C*) is defined as the relative change between pressure (*P)* and volume (*V)*:

$$C = (P_1 - P_2)/(V_1 - V_2) \tag{1.4}$$

The contents of a compartment are sometimes surrounded by relatively unyielding osteofascial structures. Intramuscular pressure might increase rapidly when the volume of the contents in an osteofascial space increases (Figure 1.4). In thin, flat muscles, e.g., the trapezius muscle with a soft epimysium, the compliance value is high. The compliance of the subcutaneous tissue is even higher.

COMPARTMENT SYNDROMES

Compartment syndrome is a condition in which elevated intramuscular pressure reduces local blood flow and impairs function of the tissues within that compartment (Figure 1.5).[10,11] The local tissue ischemia produces pain and tissue dysfunction that persists until the pressure within the compartment is normalized. Symptoms reverse once the local blood flow corresponds to the demands for nutrition and viability of the tissues. Two forms of compartment syndrome exist: acute compartment syndrome and chronic compartment syndrome.

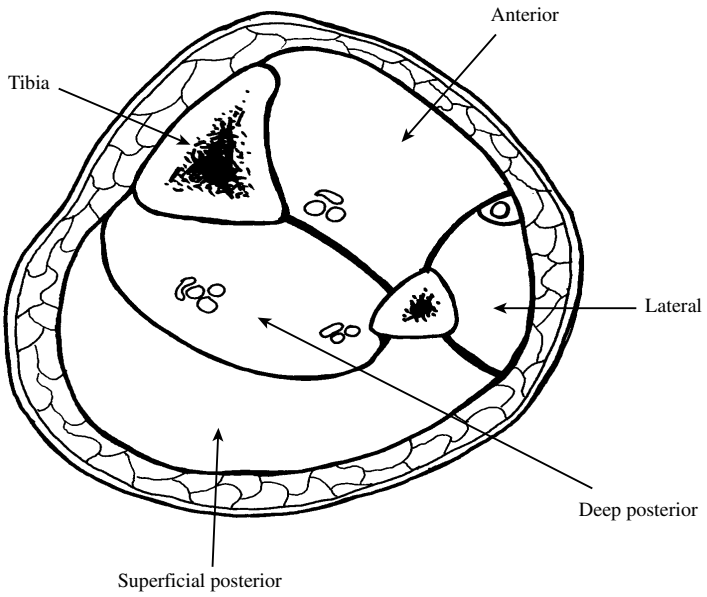

FIGURE 1.3 Cross section through the leg illustrating the anterior, lateral, deep posterior, and superficial posterior compartments with their relatively unyielding osteofascial boundaries. Nerves and vessels running through the compartments are exposed to the same magnitude of hydrostatic pressure as are muscles.

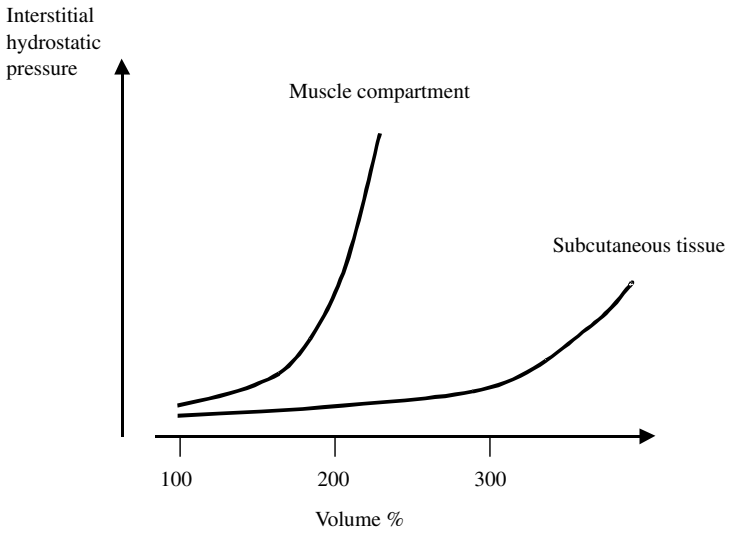

FIGURE 1.4 Schematic illustration of compliance in a muscle compartment and in subcutaneous tissue. Compartment muscles are usually more susceptible to volume increase than subcutaneous tissue.

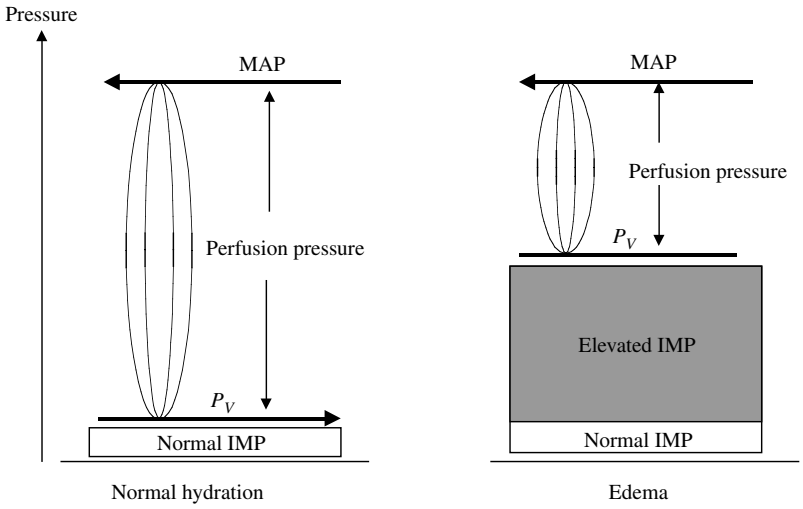

FIGURE 1.5 Schematic illustration of perfusion pressure at the capillary level in a normally hydrated tissue (left) and in an edematous tissue (right). Perfusion pressure is the difference between mean arterial pressure (MAP) and venous pressure (P_v). Venous pressure within a compartment cannot be lower than the local intramuscular pressure (IMP). Therefore, increased intramuscular pressure decreases perfusion pressure within the compartment.

Acute Compartment Syndrome

Acute compartment syndrome is a severe irreversible form of abnormally elevated intramuscular pressure that leads to tissue necrosis and permanent loss of function if left untreated. The condition may even be life threatening if several compartments, i.e., large muscle masses, are affected simultaneously. Common reasons for acute compartment syndrome include trauma with bleeding into the compartment, postischemic swelling following arterial injury, and long-term external compression of the tissues. Treatment includes decompression of the compartment tissues. Synonyms for acute leg compartment syndrome in previous literature are anterior tibial pain,[12] calf hypertension,[13] rhabdomyolysis,[14] and march gangrene.[15]

Chronic Compartment Syndromes

Chronic compartment syndromes are defined as painful conditions in which increased intramuscular pressure during exercise impedes local muscle blood flow and impairs the neuromuscular function of the tissues within a compartment.[16,17] The chronic syndrome is a mild reversible form of abnormally increased intramuscular pressure during exercise. If the patient stops exercising, the symptoms will reverse. However, if the exercise continues beyond the pain limit and the muscle continues to swell, an acute irreversible condition might develop.[18] Chronic compartment syndrome is most often diagnosed in athletes, who have a high level of physical activity.[17] Synonyms for chronic compartment syndrome found in literature are

anterior tibial pain,[19] chronic anterior tibial compartment syndrome,[20] anterior compartment syndrome,[21] exertional compartment syndrome,[22] recurrent compartmental syndrome,[23] and idiopathic compartment syndrome.[24] In this book, the condition is labeled chronic compartment syndrome.

OTHER CONDITIONS OF ABNORMALLY ELEVATED TISSUE HYDROSTATIC PRESSURE

Conditions similar to acute compartment syndrome may also develop within other nonyielding spaces such as the orbital globe (glaucoma), intracranial cavity, intrathoracic cavity (tension pneumothorax), and in the kidney.[25] Such conditions have been described in intraabdominal organs following elevated intraperitoneal pressure.[26] High intraperitoneal pressure impairs blood supply of abdominal organs by reducing local perfusion pressure. Acute abdominal compartment syndrome is defined as increased intraperitoneal hydrostatic pressure to levels that impair blood perfusion pressure of intraperitoneal and retroperitoneal organs, and induce respiratory and cardiovascular dysfunction.

ISCHEMIC CONTRACTURE

Ischemic contracture is a limb deformity that represents one of the final stages of muscle and nerve fibrosis. It is a local ischemic damage of selective tissues due to an untreated acute compartment syndrome. In 1981 Von Volkmann first described the clinical entity in the forearm.[27] He proposed that the syndrome was caused by tight dressings in traumatized limbs. Ischemic contracture may also follow local arterial injury. Treatment is individualized and may include complex soft tissue releases of muscles, tendons, and nerves. In late stages, it may require surgery on bones and joints. In 1980, *compartment syndromes* in the *Index Medicus* replaced the index word *anterior compartment syndrome*, which was used since 1972. To date, *Volkmann's contracture* has been used as an index entry.

CRUSH SYNDROME

Crush syndrome is the system manifestations of an untreated acute compartment syndrome or by other trauma to the muscle tissue. System manifestations include renal dysfunction, hypovolemic shock, and cardiac arrhythmia. Crush syndrome is different from crush injury, which describes a variety of localized traumas to soft tissues and bones, and which may also threaten tissue nutrition and viability. Patients with crush injury may have peripheral ischemia, causing crush syndrome without having acute compartment syndrome.

THE INTERSTITIAL SPACE

The volume of the interstitial space is 15% of the total body volume and is three times higher than the intravascular space. The interstitial space is the tissue located

between the cells and the vessels. The volume of the space varies between tissues. The space contains 95% of the volume of cartilage tissue and 5% of muscle tissue. In muscle tissue, the interstitial network between muscle fibers participates in the transmission of tensile force to the muscle tendon.

The interstitial space consists of a fluid phase, which includes microscopic channels that end in the prelymphatic space, and a phase of solid network. The exchange of fluid between the capillary bed and the cells is transported through the interstitial space (Figure 1.1). Diffusion is transportation of small molecules along the concentration gradient. Convection is transportation of solutes along the hydrostatic pressure gradient.

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2 Historical Perspectives of Acute Compartment Syndrome

INTRODUCTION

In 1667 Stenson first described ischemic paralysis in the lower limb.[1] He observed paralysis of the posterior extremities of an animal following occlusion of the abdominal aorta. A patient with ischemic contracture, presumably from a compartment syndrome, was reported in 1850 by Hamilton according to Hildebrand.[2] Research on the pathologic process of ischemic contractures of the extremities started more than 150 years ago. Theories on external compression, inflammation, arterial injury and spasm, venous obstruction, and tissue pressure have been investigated thoroughly for the past 100 years. The effects of strenuous exercise as a cause of acute compartment syndrome were described in the 1940s.[3,4]

Historical perspectives of the evolution of different theories on etiology, pathogenesis, pathophysiology, and treatment of acute compartment syndrome are discussed in this chapter (Figure 2.1). Reviews of the historical perspectives on the exciting search for clarity regarding ischemic contractures have been presented previously.[5,6]

EXTRINSIC PRESSURE

In a classic paper from 1881, Von Volkmann presented the hypothesis that fibrosis and contracture of the muscle in an injured extremity were caused by interrupted arterial blood supply.[7] He observed cases where the contracture followed arterial ligature, contusion to extremities, and prolonged external compression (or extrinsic pressure). Since then, prolonged external compression has been accepted as a cause of muscle ischemia.

In 1884, Leser reported seven patients in whom muscle contracture followed the application of constricting bandages.[8] He investigated the effects of ischemia on muscles in animal experiments and concluded that contractures and paralysis were caused by decreased oxygen tension in muscle tissue. It is fair to say that Volkmann described the clinical presentation and Leser introduced a laboratory model of ischemic contracture of limb muscles. More recently, external compression has been used as a model of compartment syndrome in humans.[9,10]

INFLAMMATION AND HYPEREMIA

Based on Leser's reports, in 1888 Petersen suggested that an inflammatory process caused muscle contracture.[11] In 1900, Bernays showed that lymphocytes and

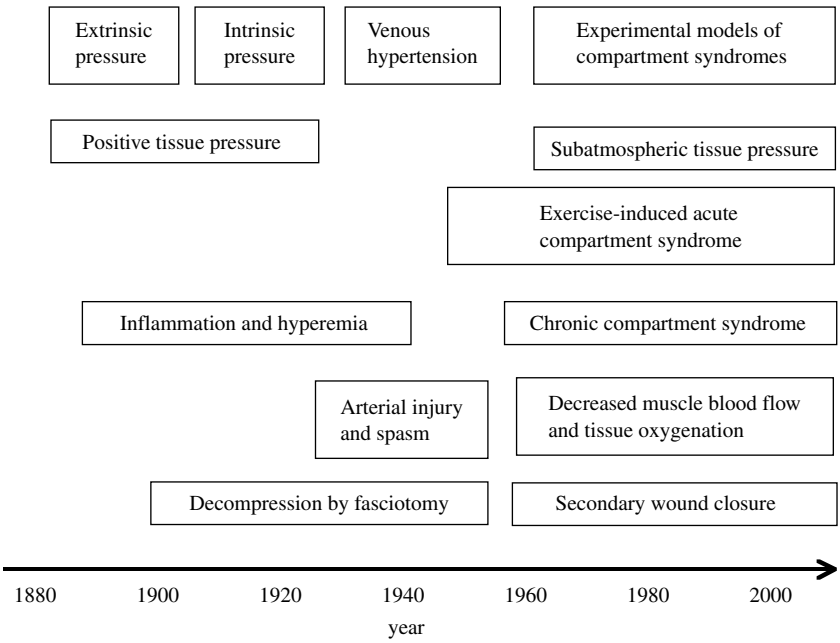

FIGURE 2.1 Summary of time aspects on the beginning of different views and theories on pathophysiology, pathogenesis, and treatment of acute and chronic compartment syndromes. Extrinsic pressure is currently regarded as increased tissue pressure due to prolonged external compression.

monocytes surrounded the ischemic contracture of the muscle.[12] In 1901, Wallis demonstrated that neurologic damage and muscle scarring was induced by hyperemia.[13] He suggested that hyperemia was the critical feature in creating Volkmann's ischemic contracture.

INTRINSIC PRESSURE

Increase of intrinsic pressure following hemorrhage and effusion into the muscle was suggested by Littlewood[14] in 1900 and by Murphy[15] in 1906. Murphy thought that venous obstruction rather than arterial injury caused the muscular contracture. Later in the 1970s, Rorabeck and McNab as well as Scheridan and Matsen created elevated intracompartmental pressure by infusing a hypertonic mannitol solution into the anterior compartment of dogs.[16,17] The clearance of Tc 99 from the anterior tibial muscle decreased with increasing intramuscular pressure.

VENOUS HYPERTENSION

Hildebrand in 1906 and Murphy in 1914 suggested that effusion by elevated venous pressure increased pressure in the muscle.[2] Brooks produced ischemic contracture by obstructing venous return in a laboratory model.[18] He experimentally created muscle

necrosis by venous ligature. Arterial ligature did not create any necrosis. Jepson's results in the 1920s indicated that contracture deformity was due to a combination of factors, of which impaired venous flow was the most important.[19] Furthermore, he demonstrated that decompression of the limb within a few hours of the start of vein obstruction prevented contracture formation. Recently, venous hypertension by vein obstruction has been used to study the effects of abnormally increased intramuscular pressure on neuromuscular function and muscle blood flow in human legs.[20–22]

ARTERIAL INJURY AND SPASM

Bardenheuer described the relation between ischemic state and arterial wall injury in 1906.[23] Leriche suggested that the mechanism of arterial spasm, caused by a nervous reflex mediated through the sympathetic nervous system, was the pathogenetical mechanism of ischemic contracture.[24] Theories regarding venous obstruction were challenged also by Griffiths in 1940, who stated that Volkmann's ischemic contracture "was due to arterial injury with reflex spasm of the collateral circulations."[25] This concept was supported by Sirbu et al. in 1944, who presented a case of bilateral gangrene in the anterior compartment of the leg, which was developing merely from marching. They explained the gangrene by vulnerability of the anterior tibial artery in the anterior compartment.[26] Although Kinmonth et al. in 1949 showed that it was not possible to precipitate arterial spasm experimentally,[27] the concept of arterial spasm was popular for many years and was later supported by Seddon[28] and by Gardner.[29] The theory of postischemic swelling in a limb following restoration of arterial flow had already been suggested by Rowlands[30] in 1910. Currently, the condition is regarded as a reperfusion syndrome.

EXERCISE

Severin in 1943 and Vogt in 1943 first described the anterior tibial syndrome.[3,4] The anterior tibial syndrome was defined as the triad of (1) necrosis of the anterior tibial muscle, (2) paralysis of the extensor digitorum brevis muscle, and (3) anesthesia of the first interdigital cleft of the foot.[31,32] Today, anterior tibial syndrome should rather be termed as an untreated acute compartment syndrome of the leg. The term *march gangrene* is used synonymously with anterior tibial syndrome.[26] In a review of the literature, Bradley reported in 1973 that 33% of all anterior tibial syndromes were induced by exercise.[6] Sixteen percent of these patients had a long history of recurrent pain induced by exercise in the anterior compartment predating the episode. These patients had participated in physical activities such as football, long marches, and other strenuous exercises. The findings suggested that a less harmful recurrent form could induce the anterior tibial syndrome.

Carter and co-workers first described a reversible form of anterior tibial syndrome in 1949.[31] Mavor first described the chronic, recurrent, reversible form of anterior tibial syndrome in 1956.[33] The existence of this syndrome was questioned by Griffiths in 1956 and by Grunwald and Silberman in 1959.[34,35] However, French and Price in 1962 and Reneman in 1968 confirmed the existence of the syndrome by measuring intramuscular pressure and local muscle blood flow.[36,37]

INTRAMUSCULAR PRESSURE MEASUREMENTS

Landerer performed the first measurement of interstitial tissue fluid hydrostatic pressure in 1884 with a needle manometer technique.[38] Some years later, Starling (1896) measured the tissue fluid pressure by injecting several milliliters of fluid through a needle into subcutaneous tissue.[39] He observed how the pressure fell to a plateau of 5 mmHg. Later the needle injection technique was improved by Henderson et al., who injected one or two cubic millimeters during measurements to make sure the needle was not occluded.[40] Burch and Sodeman improved the technique by introducing side holes in the needle.[41] They injected only 0.1 to 0.5 mm^3 at pressure recordings. In 1938, Wells and co-workers showed that intramuscular pressure increased to about 40 mmHg during venous stasis induced by a tourniquet inflated to about 70 mmHg. They also showed that intramuscular pressure during a muscle contraction reached 90 mmHg. The needle injection technique was later popularized for clinical use.[37,42,43] However, this method of pressure recording has been questioned.[44–46]

In 1941, McMaster found that fluid moved into the cutaneous tissue at atmospheric pressure.[47] He measured pressure by a 0.3-mm (30-gauge) hypodermic needle that was connected to a 0.2-mL micropipette. The fluid meniscus in the pipette moved intermittently toward the tissue. This was probably the first report indicating that interstitial fluid pressure is subathmospheric. However, the concept of slightly positive interstitial fluid hydrostatic pressures in the range of 1 to 5 mmHg in the human body prevailed until the 1960s.

In 1963, Guyton introduced the concept of a negative interstitial fluid pressure recorded by the chronically implanted capsule.[48] Negative tissue pressures were also recorded by Scholander et al. in 1968 by a wick catheter method.[49] These results were also confirmed by Snashall et al. and Wiederhielm,[50,51] but they suggested that the negative hydrostatic pressure was largely due to osmotic pressure of the interstitium. Currently, it is generally agreed that certain tissues, e.g., subcutaneous tissue, have negative tissue fluid pressures whereas others have positive pressures. Muscle tissue pressure is considered to be positive by most authors,[45,52,53] but a slightly negative pressure of about –1 mmHg has been reported.[54]

TREATMENT OF ACUTE COMPARTMENT SYNDROME AND ISCHEMIC CONTRACTURE

Surgical decompression by fasciotomy as a treatment for acute compartment syndrome was first suggested by Bardenheuer in 1906.[23] The treatment was based on his theory that the ischemic state was due to a combination of arterial injury, venous stasis, and edema. In 1914, Murphy suggested treatment by fasciotomy to prevent the contracture state from developing.[15] Later, in the 1920s, Jepson performed fasciotomy in dog experiments.[19] He demonstrated that decompression of the limb within a few hours prevented contracture formation.

In 1888, Petersen treated ischemic contracture by surgical release of contracted muscles and scar tissue.[11] He demonstrated some return of muscle function following this treatment. Seddon in 1964 reported that the process of skeletal muscle

regeneration after muscle ischemia in animals was observed by Kirby in 1892.[55] Bunnell in 1948 and Harris in 1954 described the clinical entity and surgical treatment of ischemic contracture of the hand.[56,57]

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3 Etiology and Pathogenesis of Acute Compartment Syndrome

INTRODUCTION

Acute compartment syndrome most often affects muscles contained in relatively unyielding osteofascial spaces. The syndrome is more likely to evolve in round, fusiform muscles than in flat, thin muscles with a thin epimysium. However, the syndrome may evolve in any muscle as long as the tissue pressure is high enough during a prolonged period of time.

The incidence is significantly higher in patients under 35 years of age. The young patient with tibial diaphyseal fracture and the young male with a forearm fracture sustained in high-energy trauma have increased risk for developing an acute compartment syndrome.[1,2]

Pathogenesis of the syndrome is the abnormally increased intramuscular pressure within one or more well-defined osteofascial spaces. Abnormally increased intramuscular pressure may be induced in several ways (Figure 3.1). The first etiology is increased volume of the compartment contents. This is seen in patients with hematoma, edema, or a combination of the two. The second etiology is prolonged ischemia of a limb with or without initial swelling. It may be due to external compression, arterial occlusion, limb elevation, or to any combination thereof. The third etiology is decreased size of the compartment, which may be induced by scar formation following circumferential burn injuries. Finally, pathogenesis of an acute compartment syndrome may include one or more of these etiologies simultaneously. Knowledge about the etiology and pathogenesis of the syndrome is helpful to diagnose and take measures to prevent the development of an imminent acute compartment syndrome.

INCREASED VOLUME OF THE COMPARTMENT CONTENTS

Hematoma, edema, or a combination of the two increases intramuscular pressure because of increased volume of compartment contents (Table 3.1).

Fractures

Fractures are the most common reason for abnormally increased tissue pressure. Thomas showed this in 1909.[3] Fracture of the tibia may induce an acute compartment

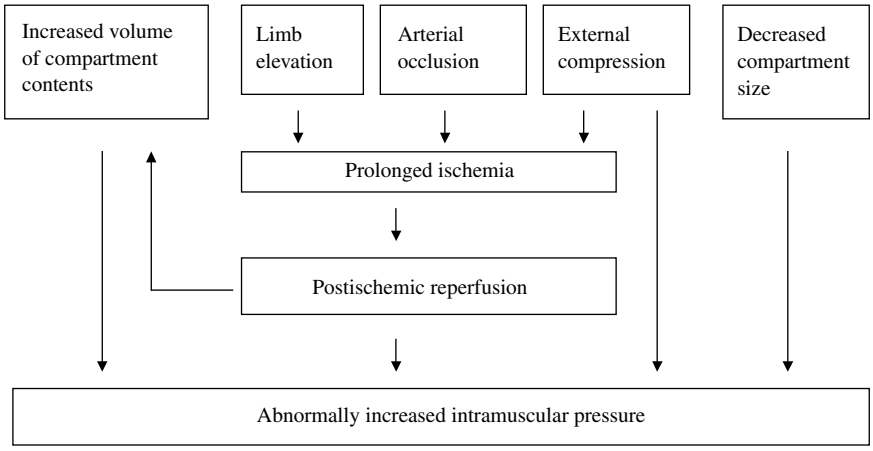

FIGURE 3.1 The possible mechanisms for pathogenesis of elevated intramuscular pressure and acute compartment syndrome include increased volume of the compartment, prolonged ischemia, and decreased size of the compartment, or any combination thereof.

syndrome in about 3 to 10% of the patients.[4–7] The risk may be even higher in open fractures.[8] Compartment syndrome occurred in 20% of the children with fractures of the midshaft of the radius and ulna.[9] The syndrome may occur after reduction of a displaced fracture.[10] Intramuscular pressure increased by 7 mmHg when fractures were reduced by traction. In another study, pressure increased an average of 12 mmHg when calcaneal traction was used.[11]

Transient increase in compartment pressure occurs during reaming of tibial fractures.[12–15] Patients with associated head injuries have increased risks for sequel from missed compartment syndrome.[14] Therefore, intracompartmental pressure should be measured in any patient with signs of elevated intramuscular pressure in whom a reliable clinical examination cannot be performed. Both the duration and magnitude of the elevated pressure must be considered.

Excessive traction of the lower limb is likely to increase the risk of compartment ischemia. Application of 6 kg of traction increased the mean resting pressure by more than 30%.[11] This may be explained by the sudden decrease in the volume of the compartment when the relaxed soft tissues and bones suddenly are distracted.

Soft Tissue Trauma

Ischemic contractures have been described following soft tissue injury alone.[16] Permanent neuromuscular damage may also occur in patients with no or few clinical symptoms.[7] Closed muscle rupture is an uncommon cause for the syndrome.[17–19] It has been described following peroneal muscle rupture in a rugby player and in the forearm of a forestry worker.[19] It has also been described in the thigh following rupture of the vastus lateralis muscle.[20]

TABLE 3.1
Literature on Etiology and Pathogenesis of Acute Compartment Syndromes: Increased Volume of Compartment Contents[a]

Etiology and Pathogenesis	Ref.
Bleeding	
Fractures	6, 8, 10, 13, 14, 87–91
Muscle rupture	17–19, 92
Surgery	41, 93 12, 15, 75, 94, 95
Arterial injury	37, 96–98
Edema	
Venous stasis	51, 68, 99–103
Capillary leak syndrome	104–107
Exercise	24, 108, 109
Inflammation	110, 111
Bleeding and Edema	
Fractures	4, 6, 7, 9, 10, 14, 87, 112–122
Other Reasons for Volume Load	
Intraosseus infusion	40, 123
Combined arterial and venous injuries	97
Dissecting popliteal cyst	124
Treatment by thrombolysis and coronary bypass	125, 126
Knee arthroscopy	127
Hip arthroplasty	41–43

[a] Increased volume of the compartment contents due to bleeding, edema, or a combination of the two may increase intramuscular pressure to abnormal levels.

Surgical Treatment

The syndrome may develop in patients following surgical treatment by the Hauser operation, proximal tibia osteotomy, and intramedullary nailing of the tibia.[15,21,22] Postoperative hematoma may play a significant role in the development of acute compartment syndrome. Patients treated by wound drainage following tibial osteotomy have significantly lower pressure in the anterior compartment compared to that of patients without drainage.[23]

Exercise

Exercise-induced edema by marching and mountain climbing may be another reason for acute compartment syndrome. In a review of literature, Bradley reported that in one third of all patients with acute compartment syndromes, the conditions were induced by exercise.[24] Recent publications report that exercise is an uncommon reason for acute compartment syndrome in the upper extremities,[25,26] the thigh,[27,28] leg,[29–32] and foot.[33,34]

Other Reasons for Increased Volume

Bleeding because of vascular injury and coagulopathy are other reasons for hematoma.[35] Also, intramuscular injections in patients who receive anticoagulation treatment increase the risk for the syndrome to develop.[36,37] Intravascular injections may also induce acute compartment syndrome.[36,38] Ischemic contracture was described following arterial aneurysm of a fractured tibia.[39] Intraosseos infusion may induce the syndrome.[40] The syndrome may follow in a swollen thigh after total hip arthroplasty.[41–43]

PROLONGED ISCHEMIA

The second etiology for increased intramuscular pressure is prolonged external compression, arterial occlusion, limb elevation, or any combination thereof (Table 3.2). They may all induce a postischemic reperfusion injury to the capillary bed. Tissue hypoxia induces dysfunction of the endothelial cells of the capillary bed, which may leak plasma and water into the interstitial space. The reperfusion syndrome increases intramuscular pressure directly and by increased volume of the interstitial space of the compartment contents.

External Compression

After prolonged ischemia due to external compression, a compartment syndrome may occur due to postischemic swelling. Ischemia induces dysfunction of the endothelial cells, which leak plasma into the interstitial space following ischemic damage.[44–47] Six hours of external compression induced a reperfusion injury. About 50% of the muscle fibers were damaged.[48] However, distal muscles that were exposed to

TABLE 3.2
Literature on Etiology and Pathogenesis of Acute Compartment Syndromes: Prolonged Ischemia[a]

Etiology and Pathogenesis	Ref.
Eternal Compression	
Pneumatic antishock garments	52, 55–58, 71, 128, 129
Lumbar spine surgery	130
Tourniquet	131
External compression and limb elevation	61, 63, 69, 132
Arterial Occlusion	
Arterial thrombosis	96, 133
External Compression and Limb Elevation	
Prolonged surgery in the lithotomy position	51, 61–65, 67, 68, 132, 134

[a] Prolonged ischemia due to external compression, arterial occlusion, and limb elevation may induce ischemia and a postischemic reperfusion syndrome.

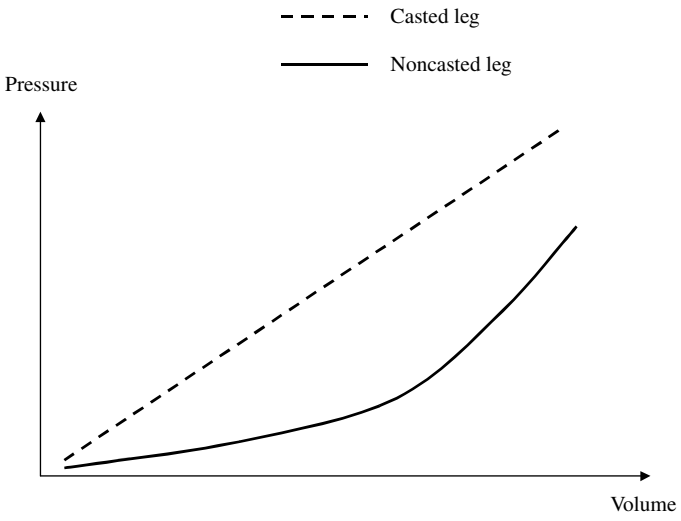

FIGURE 3.2 Schematic drawing of the relationship between volume and pressure in a compartment of a casted leg (dotted line) and noncasted leg (solid line).

ischemia but not to external compression did not demonstrate any increased damage with reperfusion.

External compression by tight bandages such as orthoses, plaster cast, or elastic stockings increases intramuscular pressure. A tight plaster cast decreases the compliance of compartment tissues.[49–51] A normal swelling of a limb may therefore create pathologically increased intramuscular pressure (Figure 3.2).

Another reason for increased intramuscular pressure by external compression is long-term unconsciousness in drug-addicted patients[52–54] and in patients treated by military antishock trousers.[55–60] Figure 3.3 illustrates how intramuscular pressure in the arm increases to high levels if it is compressed between the chest and a hard undersurface.

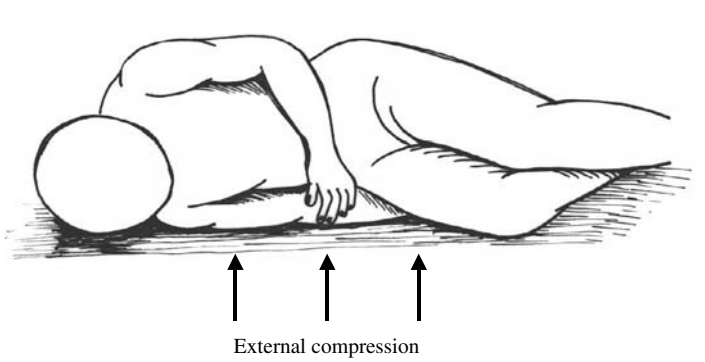

FIGURE 3.3 The right arm of the subject exposed to external compression of a forearm.

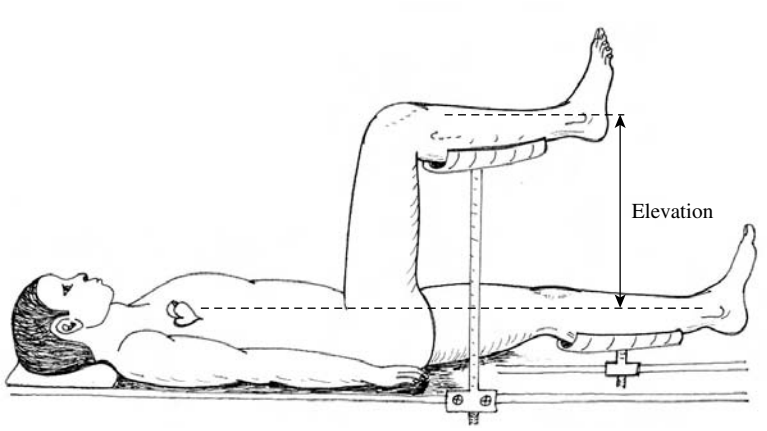

FIGURE 3.4 Elevated limbs in the lithotomy position decrease the leg perfusion pressure significantly. The risk for acute ischemia is increased if the limb is elevated during an extended period of time.

Arterial Occlusion

Intramuscular pressure is initially normal in patients with total arterial occlusion. Successful surgical treatment of arterial occlusion may induce a reperfusion injury of the capillary bed. The time period for warm ischemia of extremity is important to estimate and document. If it exceeds 6 hours of complete ischemia, the endothelial cells most likely will be damaged and leak plasma into the interstitial space.[44,48]

Limb Elevation

Patients with arteriosclerosis and diabetes of the lower limb have increased risks for insufficient blood perfusion and development of ischemia. Acute compartment syndrome of the lower extremities has been described following colorectal surgery,[61] urology (lithotomy position),[62–64] and pelvic surgery.[65] Adverse effects in subjects with an elevated limb and abnormally increased intramuscular pressure have been described in experimental studies on humans (Figure 3.4).[51,61,66–70]

DECREASED SIZE OF THE COMPARTMENT

The third etiology for increased tissue pressure is decreased volume of the compartment. This may be due to closure of fascial defects and scar formation due to burn and frost injuries (Table 3.3). Restricted swelling of a normally sized compartment may occur in patients treated by plaster casts and other bandages after surgery and trauma.[71,72]

Fascial Defect

A fascial defect may be painful if the muscle tissue herniates through its opening. This may occur during exercise when muscle tissue may swell up to 20% of its initial volume. Acute compartment syndrome may occur following surgical repair

TABLE 3.3
Literature on Etiology and Pathogenesis of Acute Compartment Syndromes: Decreased Size of the Compartment[a]

Etiology and Pathogenesis	Ref.
Closure of fascial defect	20, 73–75, 135–139
Burn injury	52, 76–79

[a] Decreased size of the compartment increases intramuscular pressure of the compartment tissues.

of symptomatic fascial defects.[73–75] Therefore, fascial defects must not be sutured. They should be treated by fasciotomy through the defect. Patients with circumferent injuries on the extremities may develop an inelastic scarring, which will prevent normal volume increase of the underlying tissues.

Burn Injury

In deep circumferential burns of an extremity, the skin loses its elasticity and becomes relatively rigid. The constrictive effects of circumferential burns may cause acute compartment syndromes,[52,76,77] which may be relieved by long, linear incisions through the skin along the axis of the extremity. About 10% of patients with burn injuries require escharotomy.[78] Most of these patients may need a fasciotomy as well. The anterior tibial muscle may be necrotic in burn injuries of the leg.[76] Acute compartment syndrome is difficult to recognize because these patients usually have multiple concurrent problems.[79]

OTHER ETIOLOGIES

Additional etiologies for acute compartment syndrome include prolonged anesthesia,[80] different infections,[81,82] and viper bite (Table 3.4).[83] Excessive traction of the lower limb is likely to increase the risk of compartment ischemia. Application of 6 kg of traction increased the mean resting pressure by more than 30%.[11] This may be explained by the sudden decrease in the volume of the compartment when the relaxed soft tissues and bones are suddenly distracted. Viral infections may cause rhabdomyolysis by direct cell injury or by toxin release.[84,85] Rhabdomyolysis induced by influenza infection leading to acute compartment syndrome has been described in patients with severe myalgia.[84,85] Patients with sickle cell trait have been reported to suffer from acute compartment syndrome of the leg.[86]

SUMMARY

The etiology of acute compartment syndrome includes abnormally increased volume of a compartment, prolonged ischemia, external compression, or any combination

TABLE 3.4
Literature on Etiology and Pathogenesis of Acute Compartment Syndromes: Combined Etiologies and Other Mechanisms

Etilogy and Pathogenesis	Ref.
Anesthesia	
Local nerve block	114, 140
Epidural anesthesia	80, 141
Infection	81, 82, 85
Poisoning	
Snake bite	83, 142
Limb elevation	51, 61–69, 99, 132, 134, 143–145
Drugs	52–54

[a] Combined etiologies and other mechanisms may increase intramuscular pressure to abnormal levels.

thereof. The pathogenesis is abnormally increased intramuscular pressure, which impedes the local muscle blood flow. Fractures, soft tissue trauma, surgical treatment, and exercise may induce increased volume of the compartment. Prolonged external compression, arterial occlusion, burn injuries, and limb elevation may create ischemia. They may all induce a postischemic reperfusion injury to the capillary bed. Tissue hypoxia induces dysfunction of the endothelial cells of the capillary bed, which may leak plasma and water into the interstitial space. The reperfusion syndrome increases intramuscular pressure directly and by increased volume of the interstitial space of the compartment contents. When released, these conditions may induce a reperfusion injury to the endothelial cells of the capillary bed. The third etiology is decreased compartment size following suture of fascial defects and circumferent burn injuries. Other causes include prolonged anesthesia, infections, viper bite, and any condition that causes rhabdomyolysis.

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4 Pathophysiology of Acute Compartment Syndrome

INTRODUCTION

One function of the local circulation is to deliver oxygen and to return waste products, i.e., to maintain tissue nutrition and viability. In acute compartment syndrome, the local circulation does not meet this demand. The pathophysiology of acute compartment syndrome describes the effects of increased compartment pressure on muscle blood flow and muscle and nerve function. It describes how arterial pressure, venous pressure, and perfusion pressure are affected by increased tissue pressure.

Tissue damage due to ischemia is directly related to microvascular perfusion. Several mechanisms possibly act together in different clinical situations. The different mechanisms for regulation of local muscle blood flow may act together.

THEORIES ON LOCAL REGULATION OF MUSCLE BLOOD FLOW

Understanding the pathophysiology of acute compartment syndrome is understanding the determinants of regional blood flow and how they are affected by increased intramuscular pressure. Some authors have related tissue pressure to diastolic blood pressure,[1,2] whereas others have found the relationship between mean arterial blood pressure and intramuscular pressure to better correlate with local perfusion pressure and muscle viability.[3–5] Theories on local regulation of muscle blood flow include perfusion pressure, microvascular occlusion, critical closing pressure, tidal wave, and arterial spasm.

Perfusion Pressure

The arteriovenous gradient theory is based on the hydrodynamic principles described in the law of Poiseuille:

$$Q = k \times r^4 (P_1 - P_2)/L \times N \tag{4.1}$$

where Q is the flow volume, K a constant (P/8), $P_1 - P_2$ the pressure difference in the direction of flow, r the radius of the vessel, L the length of the vessel, and N the

viscosity of the fluid. According to this law, the resistance (R) for blood flow is given by:

$$R = 8L \times N \tag{4.2}$$

Therefore, muscle blood flow (MBF) in the arteriovenous gradient theory can be expressed as:

$$\text{MBF} = (P_a - P_v)/R \tag{4.3}$$

where P_a is the pressure at the arterial end and P_v the pressure at the venous end of the capillary bed.

Mean arterial pressure (MAP) is a function of the systolic (S) and diastolic (D) blood pressures. It is calculated by adding one third of the pulse pressure ($S - D$) to the diastolic blood pressure:

$$\text{MAP} = D + 1/3(S - D) \tag{4.4}$$

Perfusion pressure (PP) is the difference between MAP and venous pressure (P_v) at the end of the capillary (Figure 4.1):

$$\text{PP} = \text{MAP} - P_v \tag{4.5}$$

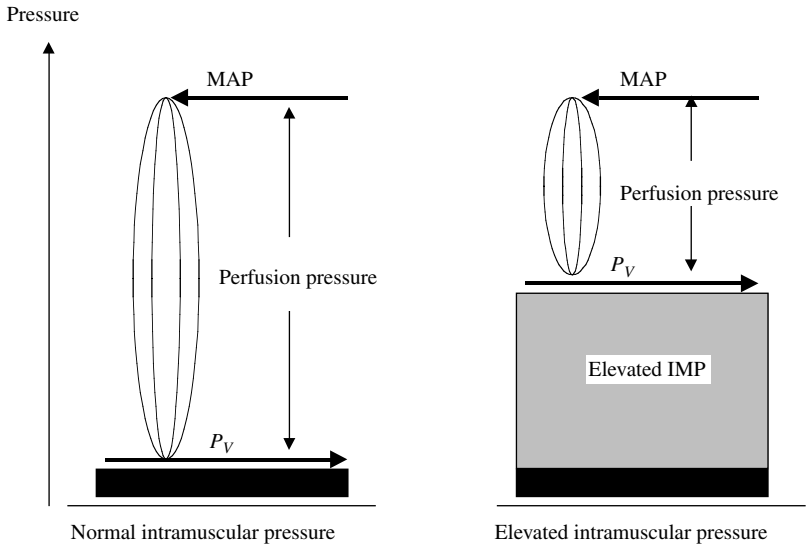

FIGURE 4.1 Schematic illustration of the pathophysiology of acute compartment syndrome. Intramuscular pressure (IMP) at rest is normally ca. 5 mmHg at heart level in the supine subject. Perfusion pressure (PP), which is the difference between mean arterial pressure (MAP) and intramuscular pressure, normally exceeds 70 mmHg (left). The elevated IMP decreases the local PP as illustrated to the right. Redrawn after Matsen et al. (1980).[33]

The pressure inside patent veins (P_v) can never be less than the extramural pressure.[6,7] Increased intramuscular pressure decreases the arteriovenous pressure difference by increasing the venous pressure (P_v). This leads to decreased PP. Therefore, intramuscular pressure (P_t) can be equal to venous pressure in Equation 4.3 and Equation 4.5. It has been shown that increased intramuscular pressure reduces the local arteriovenous gradient and thereby reduces the local MBF[8–10]:

$$PP = MAP - P_t \tag{4.6}$$

Local PP is a function of both MAP and local muscle pressure. By measuring intramuscular pressure, one can estimate PP because blood pressures are known. The overall goal is to obtain a measure on the local PP. PP must exceed 40 mmHg in a traumatized muscle and 30 mmHg in a normal muscle. Which level of intramuscular pressure a patient can manage depends on the level of MAP and the position of the limb.

Microvascular Occlusion

According to the microvascular occlusion theory, an absolute compartment pressure exists, which induces the syndrome.[11–13] Normal capillary pressure at rest is between 20 and 30 mmHg. The limiting factor in MBF disturbances has been shown to be at the capillary level.[5,11,14] Increased intramuscular pressure causes capillary vessels to collapse at their distal end.[15] This finding may be explained by the theory of microvascular occlusion.

Critical Closing Pressure

Burton first suggested the critical closing pressure theory in 1951.[6] Evidence for this concept was reported by Ashton in 1962,[16] who showed that the positive pressure intercept at zero blood flow was 33.4 (range 10 to 68) mmHg in the forearm of normal subjects at rest (Figure 4.2). This means that MAP is 33 mmHg higher than intramuscular pressure at zero blood flow.[6,17] This was explained in terms of active closure of the small arteries in muscle tissue.[18] With this theory, it is possible to explain the decrease of MBF when intramuscular pressure exceeds ca. 40 mmHg in a normotensive subject. Active closure of small arterioles under vasomotor tone may thus occur when the transmural pressure is lowered by decreased intravascular pressure or increased tissue pressure. On the other hand, others have found no evidence of a critical closing pressure by using nonpulsatile flow.[19]

The work by Heppenstall and co-workers also supports the critical closing pressure theory. They showed that blood flow was a function of the difference between MAP and intramuscular pressure in the compartment. When PP (Heppenstall's delta-*p* pressure) declined to below 30 mmHg, the requirements for normal tissue viability were not fulfilled.[20]

Tidal Wave

With this theory, Dahn et al. explained the importance of diastolic blood pressure for perfusion of the capillary bed in patients with hypotension.[21] They found that

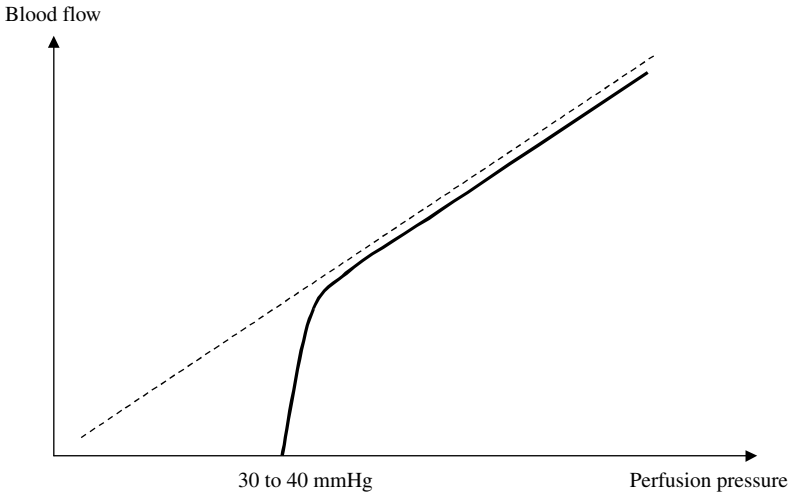

FIGURE 4.2 The critical closing pressure theory illustrates how muscle blood flow (MBF) ceases to zero when the local PP is still between 30 and 40 mmHg.

MBF did not cease until external compression reached the systolic blood pressure when venous stasis was used. They also found that MBF stopped when the locally applied compression was equal to the diastolic blood pressure. The explanation for this is given by the tidal wave theory or Starling valve theory,[21] which postulates that the microcirculation does not remain open long enough to allow perfusion (Figure 4.3). The vascular bed is thought to collapse when the transmural pressure falls to zero.

Arterial Spasm

Increased intramuscular pressure and arterial injuries have been suggested to induce arterial spasm.[22–24] The finding of normal pulses of the dorsal pedis artery of patients with acute compartment syndrome does not support this theory.

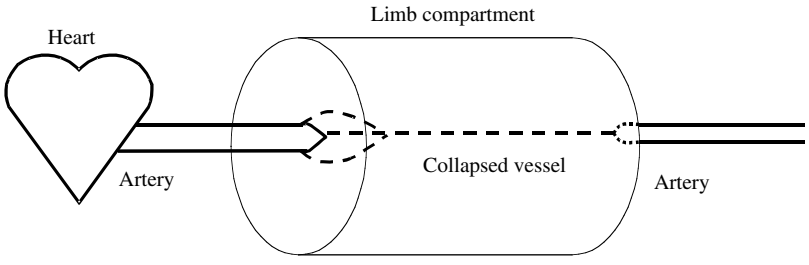

FIGURE 4.3 Schematic illustration of the tidal wave theory. The microcirculation does not remain open long enough during diastole to allow for blood flow. The elevated hydrostatic pressure within the limb compartment forces the vessels to collapse during diastole. A transient pressure increase during systole will only expand the left part of the vessel.

PATHOPHYSIOLOGY OF ABNORMALLY INCREASED INTRAMUSCULAR PRESSURE

Despite distal pulses being palpable in patients with acute compartment syndrome, local nutritive capillary blood flow may be compromised due to increased compartment pressure. The effects of increased intramuscular pressure on arterial blood pressure, venous blood pressure, MBF, muscle oxygenation, and neuromuscular function are discussed.

Arterial Blood Pressure

MAP in the main arteries of the leg is unaffected by an inflated thigh tourniquet to 60 mmHg or by abnormally elevated intramuscular pressure to 40 mmHg in a casted leg that was obstructed by venous stasis.[9,25] Increased intramuscular pressure limits flow mainly through increased critical closing pressure and with only a small change in arterial resistance.[26]

Venous Blood Pressure

Venous pressure cannot be lower than the hydrostatic pressure of the surrounding tissues.[7,26] Therefore, increased tissue pressure induces a local venous hypertension, which decreases the local PP and blood flow according to Equation 4.3 and Equation 4.5 (Figure 4.1). Increased intramuscular pressure that exceeds recumbent tibial vein pressure impairs flow in the tibial veins.[27] There is a close relationship between abnormal venous blood flow and subsequent development of neuromuscular deficit.

Venous hypertension is an important factor for the development of acute compartment syndrome in the lower limbs.[28–30] Jepson described the development of acute compartment syndrome in the leg of dogs after venous obstruction of the dogs' leg. He concluded that contracture deformity depends on a combination of factors, of which the impaired venous flow is the most important.[31]

Muscle Blood Flow

Figure 4.4 summarizes the effects of increased intramuscular pressure on local MBF by the different theories. The critical intramuscular pressure level that ceases local MBF may vary significantly depending on the theory applied.

Factors that allow skeletal muscle to compensate to maintain adequate viability include initial autoregulation of the microcirculation and enhanced venous oxygen extraction. Autoregulation compensates for the decreased blood flow by decreasing vascular resistance.

Muscle Oxygenation

The ischemia-reperfusion injury triggers release of oxygen-derived free radicals originating from endothelial cells. The injury is also aggravated by activation of neutrophils producing free radicals. Neutrophils have also been found to contribute

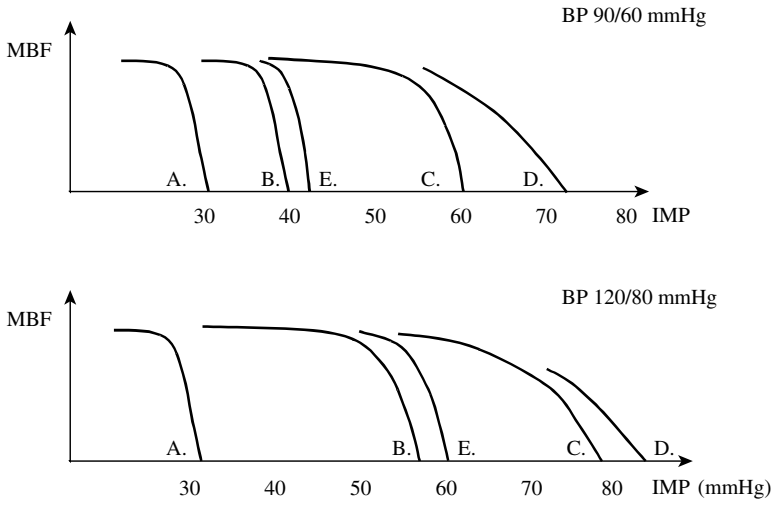

FIGURE 4.4 Schematic curves describing the relationship between IMP and local MBF in a subject with blood pressure of 90/60 mmHg (upper trace) and in another with blood pressure of 120/80 mmHg (lower trace), according to the microvascular occlusion theory (A), critical closing pressure theory (B), tidal wave theory (C), arteriovenous pressure gradient theory (D), and the delta-*p* concept (E).

to microvascular dysfunction and abnormalities of blood flow distribution in experimental studies on acute compartment syndrome.[32]

Intramuscular oxygenation approaches zero when intramuscular pressure reaches MAP locally.[33] Endothelial cells of the capillary bed become dysfunctional, allowing leakage of fluid and plasma proteins into the interstitial space.[34] Increased tissue pressure to between 40 and 60 mmHg impedes tissue oxygenation.[8,35] Total ischemia during 4 h damages 5% of the muscle cells. Total ischemia during 8 h damages 100% of the muscle cells.[2]

Neuromuscular Function

Muscle function depends on the structural integrity of the muscle and on an intact motor nerve and endplate. Once the local MBF is reduced to levels where it no longer meets the metabolic demands of tissues, losses of tissue function and viability ensue.[33] Nerve conduction velocity decreases when intramuscular pressure reaches 40 to 60 mmHg.[8,25,36,37]

HYPOTENSION AND LIMB ELEVATION

The local MBF[10] and PP[9,25,33,38,39] are greatly influenced by limb position. Elevation of extremity is commonly used to control bleeding in traumatized patients and following limb surgery. Limb elevation after prolonged surgery may also provoke acute compartment syndrome.[40–42] How arterial blood pressure, venous blood flow, abnormally elevated interstitial hydrostatic pressure in the compartment, and

neuromuscular functions are affected by limb elevation is discussed in the following section.

Arterial Blood Pressure

Mean arterial blood pressure and local PP decrease significantly by limb elevation. Elevation of a limb with increased intramuscular pressure may induce symptoms of imminent acute compartment syndrome.[9] Studies have shown that elevating a limb can impede local MBF.[43,44] Hypotension of the central circulation due to hypovolemia decreases MAP.

Venous Return

Venous stasis induces edema formation.[45,46] Like venous hypertension, it can be an important factor in the development of compartment syndrome in extremities.[28–31] Limb elevation has also been recommended as a treatment for patients with posttraumatic venous hypertension.[47] Other factors that may decrease venous return from the lower limbs are increased intraabdominal pressure,[48,49] injuries to the pelvis,[50] and extensive thrombosis.[29,51]

Interstitial Hydrostatic Pressure

Abnormally elevated intramuscular pressure in leg compartments does not decrease significantly by limb elevation.[8,9] However, moderate increase of intramuscular pressure due to vein obstruction alone is decreased by limb elevation.[9] The combined effects of increased leg volume by venous obstruction and restricted swelling by plaster cast increase compartment pressure more than what would be expected by a simple summation of the pressures.[8,9,52] The phenomenon of amplification and summation of intramuscular pressure in an obstructed and casted leg has been described in animal experiments[8,52] as well as in human studies.[9,53]

Neuromuscular Function

Intramuscular pressure levels exceeding 35 mmHg combined with limb elevation have been shown to decrease PP to levels that induce neuromuscular dysfunction.[9,33] When intramuscular pressure reaches 40 mmHg in a casted elevated leg with venous obstruction, subjects experience gradual loss of sensation in the foot or throbbing discomfort (Figure 4.5).

SUMMARY

Pathophysiology of acute compartment syndrome is the study of how abnormally elevated intramuscular pressure affects the function of blood flow, muscle, and nerve. Local PP is important to calculate in patients having impending acute compartment syndrome. PP is a function of the difference between MAP and intramuscular pressure at rest. Venous hypertension is an important factor for development of the

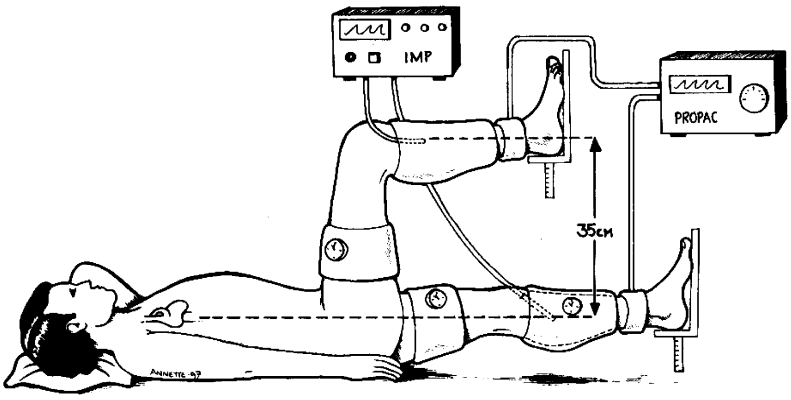

FIGURE 4.5 An experimental model to study the pathophysiology of elevated intramuscular pressure and limb elevation. Blood pressures are measured in the distal part of both limbs and in the upper arm. Venous stasis is applied by thigh tourniquet (60 mmHg). Both legs are casted; one leg is elevated.

syndrome. Limb elevation decreases mean arterial blood pressure. Tissue hydrostatic pressure and local venous pressure are unaffected by limb elevation.

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5 Diagnosis of Acute Compartment Syndrome

INTRODUCTION

Acute compartment syndrome is defined as a condition in which increased interstitial fluid hydrostatic pressure impedes local muscle blood flow and impairs the function of the tissues within a defined osteofascial space.[1] Therefore, in the clinical situation, one should search for signs of increased pressure, decreased blood flow, and impaired function of muscles. The patient's history and the physician's findings at physical examination are the cornerstones in diagnosing acute compartment syndrome. Knowledge of pathophysiology, anatomic compartments, and the nerves passing through them is essential for diagnosing the syndrome.

STAGES OF ACUTE COMPARTMENT SYNDROME

Development of the syndrome may be described in three clinical stages that are related to pathophysiological stages. Stage I is abnormally increased intramuscular pressure. Stage II is inadequate blood perfusion pressure of the compartment. Stage III is impaired function of the muscles and nerves in the compartment (Figure 5.1). Therefore, knowledge of the pathogenesis and pathophysiology of acute compartment syndrome is important to accurately diagnose the condition.

When a patient's symptoms and signs elicited have reached Stage III, clinical diagnosis may be easy to make. However, waiting for all the symptoms to develop might jeopardize the viability of tissues in the compartment. Patients may lose function permanently if the syndrome is treated after neuromuscular dysfunction has occurred.[2] A study found that only 15% of patients who experience neuromuscular dysfunction at the time of diagnosis regained normal function after treatment.[3] Time duration is therefore an important factor. Furthermore, in multiply traumatized patients, it may be difficult to differentiate between dysfunction induced by abnormally increased intramuscular pressure and dysfunction due to nerve injury. The duration of each stage may vary considerably among patients.

Symptoms and Signs of Increased Intramuscular Pressure (Stage I)

History

Patients with abnormally increased intramuscular pressure complain of tightness of the compartment(s) and local pain. The duration of external compression is important to investigate. It may be helpful to ask relatives or paramedics in which position the unconscious patient was found. This helps focus on the affected compartments.

Stage	Pathophysiology	Clinical Symptoms
I	Increased intra-muscular pressure	Swelling Decreased range of motion
II	Decreased muscle blood flow	Ischemic pain at rest Pain at passive stretch
III	Impaired function	Sensory and motor dysfunction

FIGURE 5.1 Three stages of the relationship between intracompartmental pathophysiology and clinical symptoms.

Following external compression as well as successful surgical treatment for arterial occlusion, the limb may initially appear normal. The swelling evolves over time.

Physical Examination

At examination the limb is swollen. Often the skin may reveal erythema over the compressed area. The compartment is tense, tender, and painful at palpation. Usually, there is no subcutaneous pitting edema in acute cases but the skin over the compartment may have a waxy or shiny character. Repeated measurements of the largest circumference over the muscle belly of both limbs with a tape measure are helpful to estimate the magnitude and speed of swelling. The site of measurement should be marked on the skin. The passive range of motion of the joints over which the compartment muscles act should be measured. The range of motion is often decreased before passive stretch of the muscle becomes painful. Findings should be compared with those in the contralateral extremity. The date and hour of the investigation must be documented in the patient's file.

In rare cases, the limb may be extremely swollen and the tissues hard at palpation, but the patient still has no pain. This is a time for close observation of the patient, who may start to experience pain within a short period of time. Intramuscular pressure should be measured if the findings are nonconclusive.

Symptoms and Signs of Inadequate Local Tissue Perfusion (Stage II)

History

At this stage of the syndrome the patient complains of severe increasing pain. The pain intensity is often out of proportion and is sometimes difficult to relate to the primary injury or diagnosis. When the patient's pain does not respond to analgesic medication, the pathophysiological process may have induced tissue ischemia. The patient's lack of responsiveness to pain medication may sometimes be frustrating

for the medical staff. The patient's pain may be very intensive and the objective signs discrete. Some patients with ischemic pain experience and express severe anxiety. This may sometimes interfere with normal pain behavior. We must not primarily blame psychological factors or a low threshold for pain tolerance when patients are experiencing pain with few objective signs for imminent acute compartment syndrome.

Physical Examination

The limb circumference increases further. Affected muscles are weak. The intensity of pain increases momentarily when compartment muscles are exposed to passive stretch. The reason for this is increased intramuscular pressure during passive stretch of a muscle.[4] Pressure in a volume-loaded compartment increases more during passive stretch of a muscle than in a normally hydrated compartment (Figure 5.2). The range of passive motion of the joint over which the muscle(s) function decreases with an increase in compartment volume. Distal pulses are usually normal. Pressure in the compartment is usually not high enough to affect flow in the main arteries. When pulselessness occurs, it is a sign of arterial injury and not an acute compartment syndrome. However, following arterial repair, a reperfusion syndrome may develop if the time for warm ischemia is long enough. Warm ischemia is defined as nonflow in a limb with normal or near-normal body temperature. It is most helpful to document the time period for warm ischemia in managing these patients. If the time period for warm ischemia exceeds 4 to 6 h, most patients require treatment by fasciotomy.

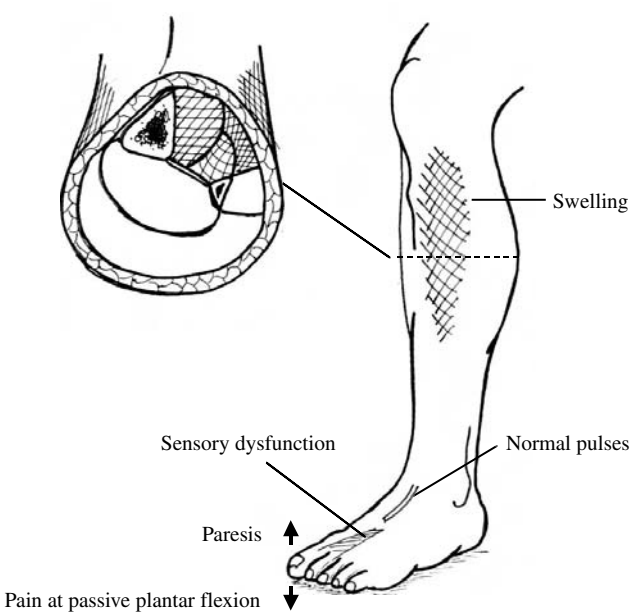

FIGURE 5.2 Clinical findings in patients with acute anterior compartment syndrome of the leg. These patients have calf pain at passive flexion of the ankle joint and the big toe.

Symptoms and Signs of Neuromuscular Dysfunction (Stage III)

The duration of abnormally increased intramuscular pressure is as important as the magnitude of pressure elevation in producing neuromuscular dysfunction.

History

At this stage, the patient experiences loss of sensation over the skin, which is innervated by nerves that pass through the compartment. The patient complains of muscular weakness or even inability to move the joints actively. The patient cannot dorsiflex the ankle joint if the anterior leg compartment is involved nor flex his/her fingers if the volar compartment of the forearm is involved.

Physical Investigation

The patient is unable to activate the muscles of the compartment and has lost sensation for light touch. Passive range of motion of the involved joint decreases further. Compartment muscles are non-responsive to electrical stimulation.

LOCATION OF ACUTE COMPARTMENT SYNDROMES

The following sections present specific etiology and diagnosis of the syndrome in the leg, foot, thigh, gluteal region, lumbar spine, shoulder, upper arm, forearm, and the hand. The symptoms of Stage II and Stage III in different locations of acute compartment syndrome are elucidated. In many patients, several compartments within a limb may be involved. Therefore, symptoms from different locations often may merge together. One example of this is the concurrent compartment syndromes of the foot and leg after trauma.[5]

Leg Compartments

The leg is the most common location for acute compartment syndrome.

Anatomy

The leg contains at least four separate osteofascial compartments (Figure 5.3). The anterior compartment encloses the extensor muscles of the leg and the deep peroneal nerve. It is one of the most nonyielding compartments. The tibialis anterior muscle has an end artery supply, which makes it more vulnerable than the other leg compartments. The lateral compartment contains the peroneal muscles and the superficial peroneal nerve. The deep, posterior compartment contains the ankle joint flexors and toe flexors as well as the tibialis nerve. The existence of additional compartments is a matter of controversy. The superficial posterior compartment consists of the medial and lateral gastrocnemius muscles and the soleus muscle.

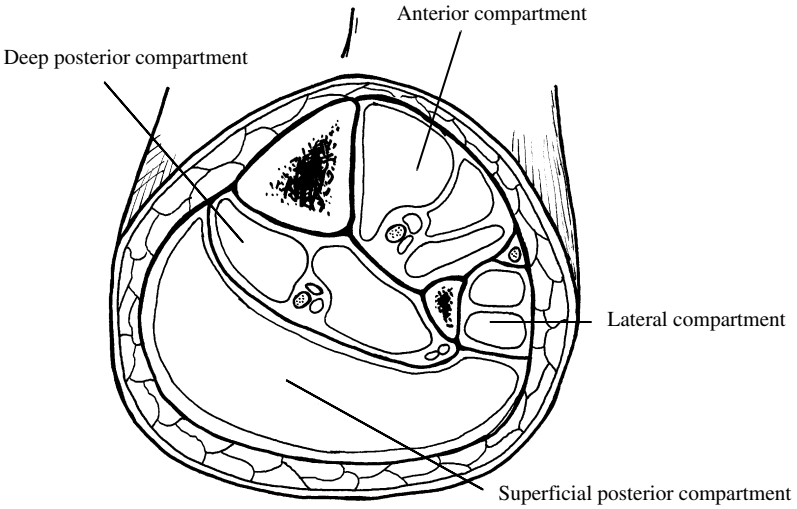

FIGURE 5.3 The four or five compartments of the leg. Some authors believe the tibialis posterior muscle is located in a separate fifth compartment.

Etiology

Fractures are the most common cause of the syndrome. The syndrome has been reported to occur in 3 to 12% of all tibia fractures. Other causes for acute compartment syndrome are discussed in Chapter 3.

Clinical Findings in the Anterior Compartment of the Leg

The leg is swollen and tender lateral to the anterior margin of the tibia. The patient's pain intensity in the anterior compartment increases by passive plantar flexion of the ankle joint and the big toe. Skin sensation is lost over the first interdigital cleft and the patient develops a weak dorsiflexion of the big toe and the ankle joint because the deep peroneal nerve is affected in the anterior compartment (Figure 5.2).

Clinical Findings in the Lateral Compartment of the Leg

The lateral compartment is located in a proximodistal direction between the fibular head and the lateral malleolus (Figure 5.4). It is swollen and tender. By passive supination of the ankle joint by the investigator, the patient experiences increased pain intensity from the lateral compartment. Skin sensation is lost over the dorsum of the foot because the superficial peroneal nerve is compressed in the lateral compartment. Often both the anterior and lateral compartments are involved and the patient develops a drop foot. The condition occurs most often together with acute anterior compartment syndrome. Isolated peroneal compartment syndromes are unusual.[6]

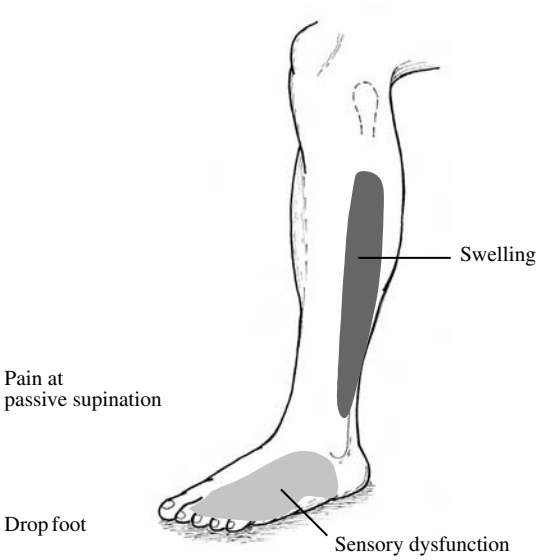

FIGURE 5.4 Symptoms and findings of acute compartment syndrome in the lateral compartment.

Clinical Findings from the Posterior Compartments of the Leg

The calf is swollen and hard at palpation. By passive dorsiflexion of the ankle joint and toes by the investigator, the patient experiences increased pain intensity from the muscles in the deep and superficial posterior compartments (Figure 5.5). The test is performed in a similar way as the Hohman's test for calf thrombosis. During this test, the patient experiences increased pain intensity from the muscles in the posterior compartments when the investigator passively dorsiflexes the ankle joint by pushing the sole of the foot. Skin sensation is lost over the sole of the foot. The ability to grip the investigator's index finger by flexing the toes is weak as well as the strength for plantar flexion of the ankle joint (Figure 5.2).

Foot Compartments

Acute compartment syndrome of the foot usually follows fractures and massive soft tissue trauma to the foot. Early diagnosis and decompression are as important as in other compartments to prevent loss of neuromuscular function.

Anatomy

Nine anatomical compartments in the foot have been identified:[7] medial, superficial, lateral, adductor, the four interossei, and the calcaneal compartment. The first three compartments are divided into three main compartments by a medial and a lateral intermuscular septum that run the entire length of the foot. The adductor and the four interossei compartments are located in the forefoot, and the calcaneal

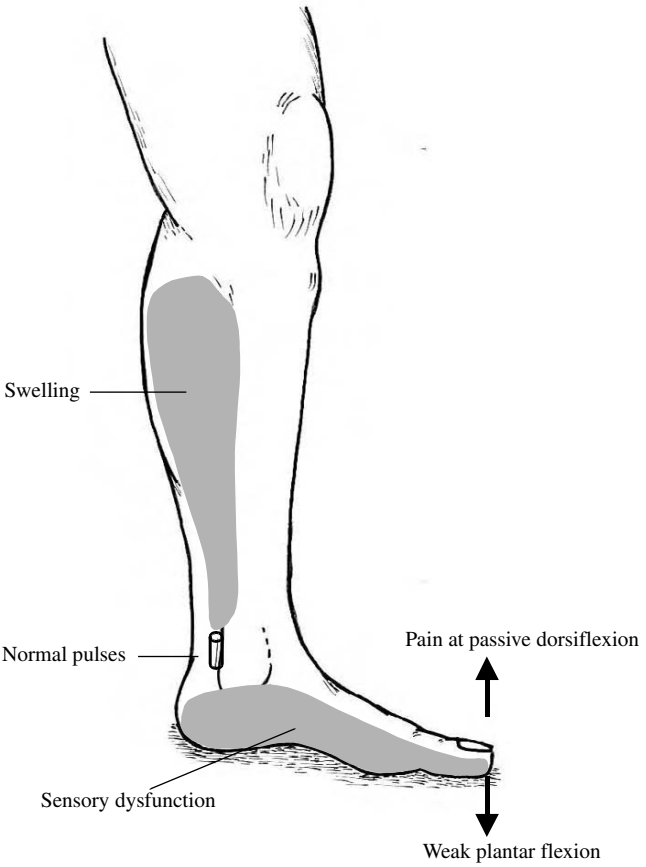

FIGURE 5.5 Symptoms and findings of acute compartment syndrome in the posterior compartments.

compartment is located in the hindfoot.[7] The neurovascular bundle runs through the calcaneal compartment. This may explain some of the confusion regarding reports on descriptions of compartment location in the foot. Some compartments are found only in the forefoot, others in the hindfoot, and only a few of them are located along the whole foot (Figure 5.6). The previously described central compartment is divided by a transverse fascia in the hindfoot into a superficial and deep compartment.

Etiology

Crush injuries to the foot may induce acute compartment syndrome of the intrinsic muscles.[8–11] About 10% of patients with calcaneal fractures may develop elevated hydrostatic pressure in the foot,[11] and about half of these patients (5%) develop claw-toes[11] if left untreated. The syndrome may follow distortion of the foot.[12]

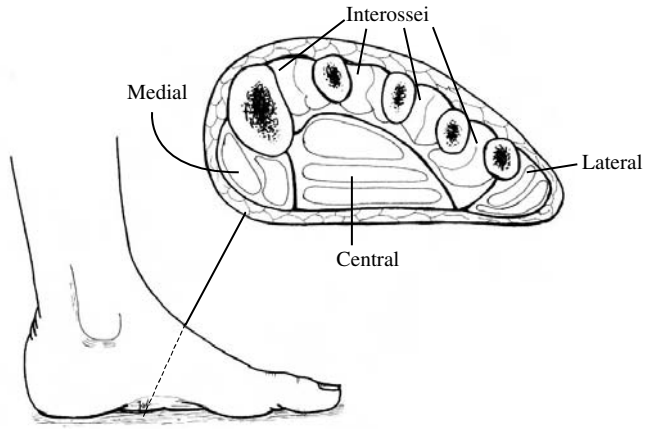

FIGURE 5.6 Cross section of the midfoot. Nine different compartments exist. The calcaneus compartment is located more dorsally toward the calcaneus bone.

Clinical Findings

In clinical diagnosis, it is difficult to separate the involvement of each compartment. Therefore, different clinical findings may not be related to a specific compartment of the foot. The patient has a tense swelling in the arch of the foot, which is tender to palpation. The patient may experience numbness and a tingling sensation in the whole foot.

Pain on passive dorsiflexion of the toes may indicate ischemia in the intrinsic muscles of the foot.[9,13–15] It is the most reliable clinical finding and occurs in 86% of patients.[16] The sign is difficult to interpret, because in many patients this occurs up to 22 months following surgical treatment. Objective motor deficits and loss of pinprick sensation is also difficult to estimate. Patients with crush injuries of the foot may have cyanotic toes and delayed capillary filling time. The end results of an untreated acute compartment syndrome in the foot are a painful dysfunctional extremity with sensory disturbances, stiffness, contracture of the forefoot, and clawing of the toes.

Thigh Compartments

Acute compartment syndrome in the thigh is a rare condition that can be seen in patients with femoral fractures, crush injuries that develop into crush syndrome, and severe contusion of the anterior thigh in contact sports.

Anatomy

The thigh has three compartments: anterior, posterior, and medial. They are separated by the lateral, posterior, and medial intermuscular septa, respectively (Figure 5.7). The anterior compartment includes the quadriceps muscles and the posterior

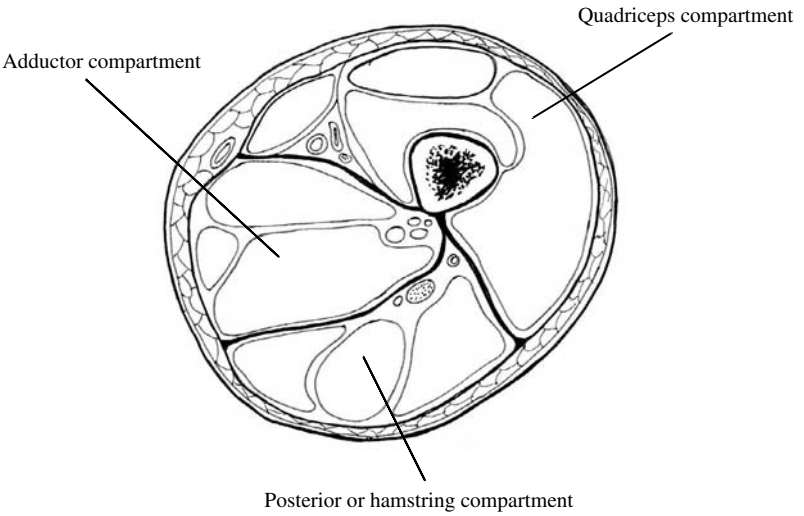

FIGURE 5.7 Cross section of the thigh compartments. The medial compartment encloses the adductor muscles. The vastus medialis obliquus, the rectus femoris, intermedius, and vastus lateralis muscles comprise the quadriceps muscles. The hamstring muscles are located in the posterior compartment.

compartment includes the hamstring muscles. The medial, or adductor, compartment contains the adductor muscles. Sensory distribution of the femoral nerve includes lateral, intermediate, and medial cutaneous nerves of the thigh. The saphenous nerve includes the sensory distribution over the medial parts of the knee and leg.

Etiology

Thigh compartment syndromes may be seen in patients with multiple trauma,[17] thigh contusion during athletic activity,[18–20] reperfusion syndrome following external compression, vascular injury, extensive venous thrombosis,[21] and femur fractures.[22] Many of the patients are multiply injured by high-energy trauma to the thigh[23] with or without femur fracture. The syndrome may follow external compression of the thigh by antishock trousers.[24] It has been reported in patients with systemic hypotension, coagulopathy, and vascular injury. In rare cases, it may be induced by exercise.[25–27]

Clinical Findings

The patient complains of intensive thigh pain. The thigh is swollen and tense. The thigh circumference should be measured every hour initially. Sensory function of the femoral nerve branches and the sciatic nerve may be impaired. The anterior compartment is tested by passive flexion of the knee joint while maintaining the hip in neutral position. This is a sensitive test for increased volume of quadriceps muscles in the anterior compartment. Pain on passive stretching of the muscles in the posterior

compartment is tested for by passively extending the knee joint while holding the hip in 60° of flexion. Even if only one of the compartments is involved initially, other compartments are often affected reactively.

Gluteal Compartments

Early diagnosis of gluteal compartment syndrome is difficult because it is rare and poorly understood. It is important to diagnose the syndrome because the compartments contain large muscle volumes, which may induce massive myoglobulinemi and the crush syndrome. Gluteal compartment syndrome, although a rare condition, should be included in the differential diagnosis of acute posttraumatic sciatic nerve palsy.

Anatomy

The pelvic region can be divided into four muscular compartments: (1) the iliopsoas muscle compartment, (2) gluteus medius and minimus, (3) gluteus maximus, and (4) the anterolateral compartment with the tensor fasciae lata muscle.[28] The iliopsoas muscles lie in an osteofascial compartment consisting of the iliac bone and the iliac fascia. The femoral nerve runs diagonally through the lateral border of the psoas belly. The external rotators of the hip may induce neuromuscular dysfunction by compressing the sciatic and peroneus nerves. At this level, the peroneus nerve runs separately in almost 30% of the population. It may be difficult to differentiate between nerve compression (entrapment) and compartment syndrome in trauma cases.

Etiology

Gluteal compartment syndromes have been reported following trauma in 15% of patients,[28–31] prolonged external compression in drug-induced coma in 50% of patients,[30–33] reperfusion syndrome following vascular surgery and positioning during other surgical procedures in 25% of patients,[30–32,34,35] and other disease-associated conditions.[31,36,37] Gluteal contractures have been reported in children following numerous injections of antibiotics in the gluteal region.

Clinical Findings

Gluteal muscles are swollen and hard at palpation. The gluteal skin is red in patients with long-term external compression. Any passive hip motion is painful, especially flexion and adduction. The proximity to the sciatic nerve can induce neuropathy by compression. Leg and foot sensation may decrease. Patients may experience weakness in all lower extremity muscles and the Achilles reflex may be absent. Dysfunction of the femoral nerve may lead to paralysis of the rectus femoris muscle and loss of sensation in the distal anterior thigh as well as the anteromedial aspect of the leg all the way to the medial malleolus.[28] Later in the presentation of the disease, patients may demonstrate dysfunction of the femoral and sciatic nerves by deficits in the thigh, leg, and foot strength. Sciatic nerve palsy caused by direct contusion is unlikely to give the delayed onset of neuromuscular dysfunction. Most patients

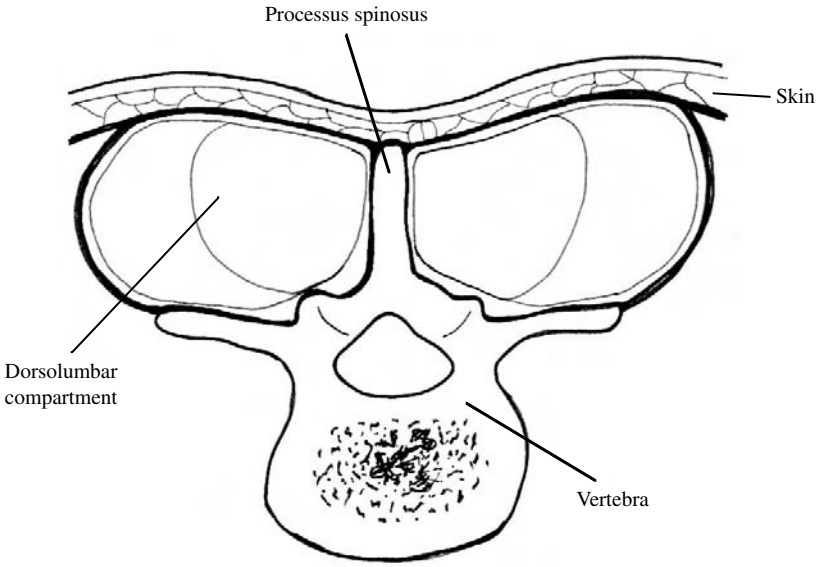

FIGURE 5.8 The dorsolumbar compartments are enclosed in unyielding osteofascial spaces.

have severe elevation of myoglobin in plasma and urine, and elevated plasma creatininphosphokinase (CPK).

Lumbar Paraspinal Compartments

Anatomy

Based on anatomical dissection of the spine, a lumbodorsal compartment has been suggested.[38,39] The erector spinae muscles are located in unyielding osteofascial space bounded anteriorly by the transverse processes and intertransverse ligaments. The lumbodorsal fascia in the dorsal and lateral direction covers the compartments. It is covered medially by both the laminae and the spinal processes with their interconnecting ligaments (Figure 5.8).

Etiology

Acute compartment syndrome in the erector spine muscles and pain in the rectus abdominis muscles was initiated by downhill skiing in one patient.[40]

Clinical Findings

The lumbar paraspinal muscles are rigid and tender. The lumbar lordosis is flattened and the lumbar spine flexed. Straight-leg raising aggravates the back pain. Sensation may be diminished over the lumbosacral area but not in the caudal area.[40] In a biomechanical study, the spinal flexion combined with lordotic flattening was labeled as "Bourbon tube effect."[41]

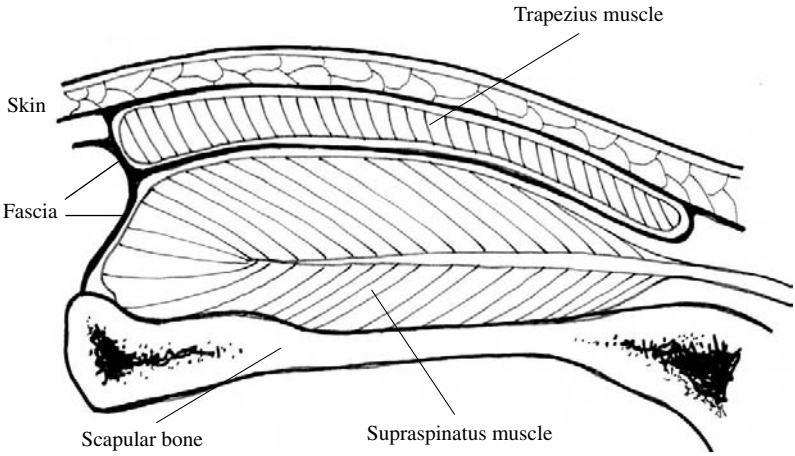

FIGURE 5.9 The supraspinatus muscle is located in an osteofascial space on the scapula, underneath the trapezius muscle.

Shoulder Compartments

Anatomy

Four separate osteofascial compartments may be identified in the shoulder. These are the three muscles related to the scapula,[42] the supraspinatus, infraspinatus, and the subscapularis muscles; the fourth is the deltoid muscle (Figure 5.9).

Etiology

Prolonged external compression in drug addicts and severe trauma to the scapula may induce abnormally elevated intramuscular pressure in the muscles. Vigorous exercise by bayonet fencing training induced symptoms of acute compartment syndrome in the supraspinatus muscle.[43]

Clinical Findings

Local triangular swelling proximal and distal of spinae scapulae indicates the syndrome.[42] Passive inward and outward rotation of the shoulder may induce increased pain intensity due to increased pressure in the supraspinatus and infraspinatus muscles.

Upper Arm Compartments

Anatomy

The upper arm has two compartments: the anterior compartment encloses the biceps muscle, and the posterior compartment encloses the triceps muscle. On the medial side between the compartments, the neurovascular bundle is located in the

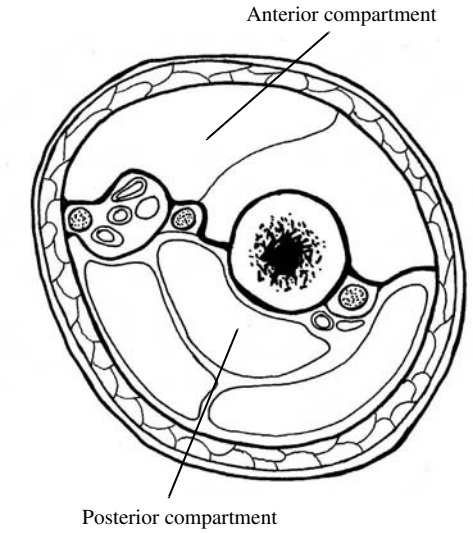

FIGURE 5.10 Cross section of the anterior and posterior compartments of the upper arm. The main neurovascular bundle runs medially between the compartments.

intermuscular septum (Figure 5.10). The fascial boundaries are not as rigid compared to the forearm and leg compartments.

Etiology

Direct trauma or compression of the upper arm in unconscious patients initiate the syndrome.[44–48] It may follow as a complication of an inflated pneumatic tourniquet,[49] angiography, dislocation of the shoulder, and avulsion of the triceps surae muscle.[50]

Clinical Findings

Patients experience pain, swelling, and tenderness of the upper arm. Passive flexion or extension of the elbow induces severe pain, and the range of motion decreases when the syndrome evolves. Sensation and grip strength of the hand may be reduced due to dysfunction of the median nerve.[49] The skin may show delayed capillary refill.

Forearm Compartments

Anatomy

The three major compartments in the forearm are the deep volar, superficial volar, and dorsal. The extensor indices proprius muscle may be located in a separate fourth compartment (Figure 5.11).

Etiology

The syndrome is described after forearm fractures,[51–54] cannulation of arm vessels,[55–57] and prolonged external compression. Between 2% and 3% of all forearm

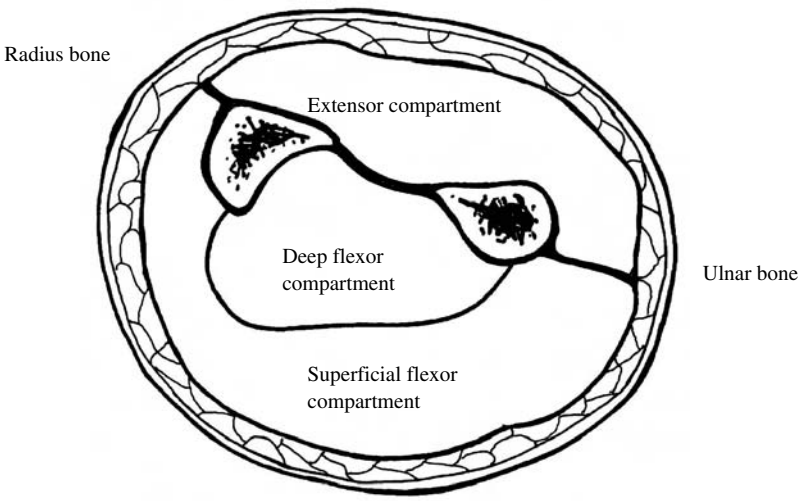

FIGURE 5.11 Cross section of the forearm showing an extensor compartment and two flexor compartments.

fractures develop a compartment syndrome.[54] The etiology of forearm compartment syndrome is injection of drugs in 30%, trauma in 40%, and external compression of the arm in 15%. Less usual etiologies are severe burn injuries and diseases. Thompkins presented a patient with exercise-induced acute compartment syndrome in the forearm extensor compartment following strenuous canoeing.[58]

Clinical Findings

The muscles of the forearm are swollen and tender at palpation. Passive extension of the fingers and the wrist induces severe pain in the volar compartments, and patients may experience weakness of the handgrip. Patients may lose sensation in their fingertips. Passive flexion of the fingers and the wrist induces pain if the dorsal compartment is involved.

Hand Compartments

About half of the patients with acute hand compartment syndrome also have a simultaneous forearm compartment syndrome.[59] This implies that hand ischemia is only one facet of a more complex clinical picture.

Anatomy

The ten compartments of the hand include the four dorsal interossei compartments; the three volar interossei compartments; and the hypothenar, thenar, and adductor pollicis muscle compartments (Figure 5.12). The carpal canal is also a physiologic compartment. Edema and swelling within this space are bound by synovial tissue and cannot escape proximally or distally. Some physicians consider six compartments of the hand.

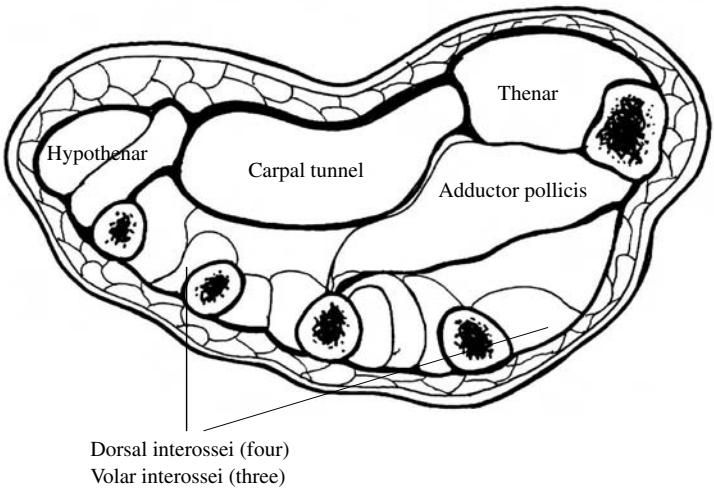

FIGURE 5.12 Cross section through the carpal tunnel and the ten compartments of the hand.

Etiology

The etiology includes multiple fractures and snakebites,[59] crush injuries, burn injuries,[60] and intravenous and intraarterial injections.[56,57]

Clinical Findings

The patient has a painful, tense, and swollen hand. The metacarpophalangeal joints are held extended and the interphalangeal joints flexed. In this position, the interossei muscles are short. The pain intensity increases by passive stretching of the involved intrinsic muscles.[61] However, in many cases, pain cannot be elicited by passive motion of the fingers when only the hand is involved.[59,61] Sensation in the digits is usually normal. Later, and in more extensive cases, compression of the median nerve results in sensory loss.

ADDITIONAL DIAGNOSTIC TOOLS

Diagnosis by clinical examination is sometimes easy and conclusive. However, it requires a conscious patient who can cooperate and an experienced examiner. Therefore, adjunctive diagnostic techniques such as bedside measurements of intramuscular pressure or direct noninvasive nerve stimulation may be helpful diagnostic tools. Other noninvasive techniques that have been considered for diagnosing acute compartment syndrome include MRI studies, pulse oximetry,[62] and sensory testing by vibration exposure.[63]

Intramuscular Pressure Measurement

Abnormally increased intramuscular pressure is the pathogenic mechanism, which induces the symptoms of acute compartment syndrome. Whenever clinical signs and

symptoms are nonconclusive, intramuscular pressure may be measured to confirm or exclude the syndrome. Measurements of elevated intramuscular pressure are also helpful to identify patients who do not primarily require surgical treatment, but initially need nonsurgical treatment. Different methods of intramuscular pressure measurements are presented and discussed in Chapter 16.

Direct Nerve Stimulation

Painful swollen extremities are commonly seen following trauma. Patients who are unable to activate their compartment muscles voluntarily may have a nerve injury, an acute compartment syndrome, painful inhibition of muscle activity, or any combination thereof. The question arises whether the paralysis is due to a primary nerve injury or secondary to abnormally increased tissue pressure. Direct nerve stimulation at the site where the nerve enters the compartment may be a valuable investigation to estimate the pathogenesis of neuromuscular dysfunction. A normal response to electrical stimulation of the compartment nerve indicates that the cause of the paralysis is not a compartment syndrome.[64] No muscular response indicates that the patient has an acute compartment syndrome with no functioning muscle fibers.

Imaging Techniques

Imaging technologies such as standard CAT or MRI for diagnosis of acute compartment syndrome are usually not indicated. Treatment should not be delayed to obtain these studies. They do not add anything essential that cannot be found by the patient's history and by clinical investigation. Imaging techniques may delay diagnosis and treatment of acute compartment syndrome. An arteriogram is indicated when vascular injury is suspected or repair is indicated.

Pulse Oxymetry

The pulse oxymeter measures the arterial oxygen saturation of the arterial blood reaching the compartment, and not the tissue oxygen saturation of the compartment itself. The role of pulse oxymetry in the early detection of acute compartment syndrome has to be determined.[62] However, in patients with chronic compartment syndrome, it has been reported to be useful.[65,66] Near-infrared spectroscopy can detect muscle ischemia, which is caused by acute compartment syndrome despite severe hypotension and hyperemia, making it potentially useful in critically injured patients.[67]

Stimulation by Vibration

Altered perception of vibration stimuli correlated with decreased neutral function in subjects with elevated intramuscular pressure between 35 and 40 mmHg.[63] Alterations in vibratory sensory testing as measured with a 256-cps tuning fork was found to be an early and reliable indicator of abnormally elevated compartment pressure.

TABLE 5.1
Typical Findings of Acute Compartment Syndrome, Arterial Injury, Venous Occlusion, and Neurapraxia[a]

Symptoms	ACS	Arterial Injury	Venous Occlusion	Neurapraxia	Inflammatory Reaction
			Stage I		
Increased pressure	++	0	+	0	+
Swelling	++	0	+	0	+
Erythema	+	0	0	0	++
			Stage II		
Pain at rest	++	++	+	0	++
Pain with stretch	++	+	+	0	+
Decreased passive ROM	++	0	0	0	+
Intact pulses	++	0	++	++	++
			Stage III		
Paresthesia	++	0	0	++	0
Paresis	++	0	0	++	0

Note: ACS = acute compartment syndrome; ++ = frequently or always; + = sometimes or to a certain extent; 0 = seldom or never; ROM = range of motion.

[a] Symptoms are elicited by the increased intramuscular pressure in Stage I, by the impeded blood flow in Stage II, and by impaired neuromuscular function in Stage III.

DIFFERENTIAL DIAGNOSIS

It may be difficult to separate symptoms elicited from injury to arteries, veins, and nerves in patients who are multiply injured. Table 5.1 summarizes some of the findings of the different conditions. All the listed findings of Stage I to Stage III are positive only in patients with acute compartment syndrome. Patients with arterial injury have local pain at rest and no arterial pulses distal to the injury. Patients with venous occlusion have swollen legs, and may have pain at rest and pain with passive stretch of muscles, e.g., a positive Hohmans sign in the leg. Patients with neurapraxia have disturbances of sensitivity and motor weakness distally, whereas patients with inflammatory reaction have mild symptoms of compartment syndrome listed in Stage I and Stage II. Only patients with acute compartment syndrome and neurapraxia have Stage III symptoms.

SUMMARY

The patient's history and findings at physical examination are the cornerstones in diagnosing acute compartment syndrome. Development of the syndrome may be described in three clinical stages that are related to the pathophysiology of the

syndrome. In Stage I, patients experience swelling and decreased range of movement due to increased intramuscular pressure. In Stage II, the pain becomes ischemic due to impaired muscle blood flow. Passive stretch of affected muscles induces severe pain. In Stage III, neuromuscular impairment gives motor and sensory dysfunction. Intramuscular pressure recording and subsequent calculations of local perfusion pressure are helpful to identify patients with the syndrome.

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6 Treatment of Acute Compartment Syndromes

INTRODUCTION

The quality of functional results is related to the promptness of treatment. Therefore, treatment of acute compartment syndrome must not be delayed. The goal of treatment is to restore a normal neuromuscular function. This is achieved by normalizing the local perfusion pressure, which is obtained by decreasing the abnormally elevated intramuscular pressure in the compartment by fasciotomy. Early active treatment to reduce edema following surgery is important. The wound is closed by secondary wound closure in most patients with residual swelling after fasciotomy.

GENERAL PRINCIPLES FOR TREATMENT

Early countermeasures to prevent swelling after trauma and to maintain local perfusion pressure are important. Local blood perfusion can be restored in the compartment by the following six steps.

Step 1. Intramuscular pressure decreases by cutting all dressings that give external compression.
Step 2. Local perfusion pressure in the leg increases when elevation of the injured limb is concluded.
Step 3. Mean arterial pressure increases by treating hypovolemia.
Step 4. Surgical treatment by acute fasciotomy normalizes intramuscular pressure.
Step 5. Treatment of postischemic reperfusion prevents crush syndrome from developing.
Step 6. Postischemic swelling following fasciotomy may be reduced by early active edema reduction. Each of the steps is discussed in detail.

STEP 1

By cutting all dressings that give external compression, intramuscular pressure decreases to levels that allow for normal circulation (Figure 6.1).[1,2] Elevated intravenous pressure decreases with decreased intramuscular pressure. Intravenous pressure in a compartment can never be lower than intramuscular pressure.[3]

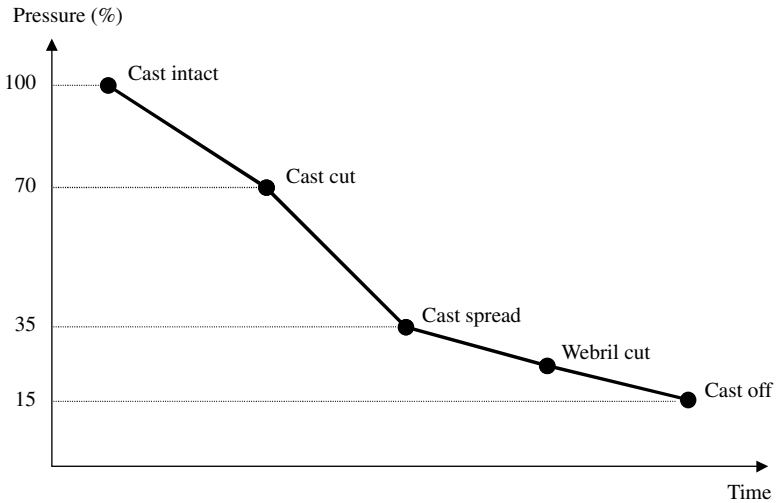


FIGURE 6.1 Decrease in intramuscular pressure in a swollen limb by stepwise removal of plaster cast and bandages. The absolute initial pressure may vary between 60 and 100 mmHg.

Step 2

Correct treatment by Step 2 requires considering how abnormally increased intramuscular pressure, local intravenous pressure, and mean arterial pressure are affected by limb elevation. Abnormally elevated intramuscular pressure in a supine subject does not decrease by elevation of the affected limb.[4–7] Patient's tolerance for increased intramuscular pressure decreases by limb elevation. Local intravenous pressure in the compartment cannot be decreased by limb elevation, because of venous congestion. Venous pressure always exceeds local tissue hydrostatic pressure.[3] Mean arterial blood pressure decreases by limb elevation.[4,8] Therefore, limb elevation decreases local blood perfusion pressure in patients with impending acute compartment syndrome (Figure 6.2). Limb elevation is harmful when intramuscular pressure is abnormally increased and may even, on theoretical grounds, elicit an iatrogenic acute compartment syndrome.[4,8]

Step 3

It is important to maintain the mean arterial pressure by treating the hypovolemia. The interstitial space is three times larger than the intravascular space in the human body. Large amounts of fluid may rapidly shift from the intravascular space to the interstitial space in traumatized patients. Fluid shift from the intravascular space into the interstitial space may cause a predisposition to hypovolemia.

Step 4

Decompression by fasciotomy is based on typical clinical symptoms and signs. It normalizes compartment pressure by increasing the compartment size. Residual

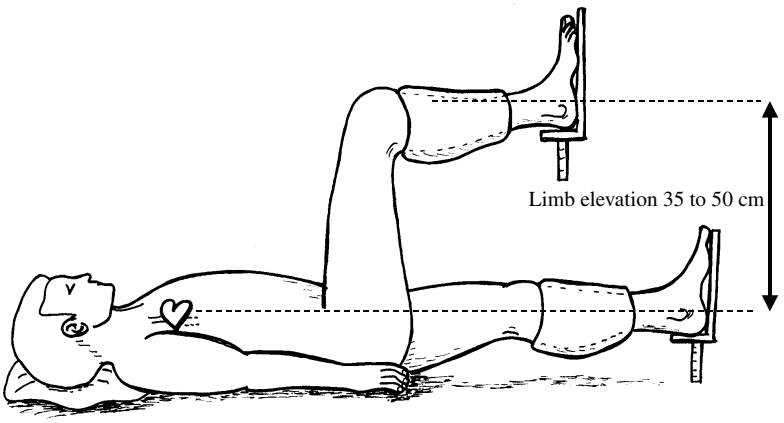


FIGURE 6.2 Reduction of local perfusion pressure by 50% if the limb is elevated 35 to 50 cm, due to decreased mean arterial pressure by 25 to 38 mmHg in the elevated compartment.

muscle tension may be treated by epimysiotomy.[9] Techniques for most locations are discussed later.

Step 5

Postischemic reperfusion is commonly seen after orthopedic, vascular, or reconstructive plastic surgery. Limb swelling, muscular dysfunction, or muscle necrosis may complicate treatment by fasciotomy for acute compartment syndrome. Further damage of muscle tissue may occur on postischemic reperfusion.[10–12] It is believed that the tissue damage seen after extended periods of ischemia is a consequence of both ischemia and reperfusion and not a consequence of the reduced blood flow alone.[10–13] Oxygen free radicals may play a critical role in ischemia-reperfusion injury. Free radicals are atoms or molecules with one or more unpaired electrons, making them extremely reactive and cytotoxic. Therefore, reoxygenation may induce multifold cellular and vascular permeability alterations. Treatment of ischemic and reperfused tissue with different antioxidants and scavengers may have a salvaging effect in skeletal muscle.[14]

In an experimental study, bleeding from the muscle following fasciotomy was no guarantee for tissue viability.[15] The results were explained by an abnormal intramuscular revascularization that distorted the entire intravascular system. The condition was termed concealed pressure-ischemia contracture. The muscle might survive partly, but with patchy nonfunctioning areas within it.

Step 6

Edema reduction should be initiated as soon as possible following fasciotomy. Concentric muscular activity and passive muscular stretch may reduce limb edema. Other methods include intermittent external compression, limb elevation, and hyperbaric oxygenation. In uncomplicated cases, wound closure is possible during the third to fifth day after fasciotomy. Methods for edema reduction are discussed in Chapter 18.

INDICATIONS FOR SURGICAL TREATMENT BY FASCIOTOMY

The characteristic symptoms and signs of an acute compartment syndrome are the best indications for decompression by fasciotomy. However, symptoms and signs may be difficult to interpret and a definite diagnosis cannot always be made on clinical grounds alone.[16,17] Confusion concerning the indications for decompression exists. The concept of a critical intramuscular pressure value above which surgical decompression should be performed is controversial. Some authors advocate the use of absolute intramuscular pressure values.[18,19] Others relate intramuscular pressure to diastolic blood pressure.[20] The author's opinion is that calculations of local blood perfusion pressure are the best adjunct parameter to a thorough history and clinical examination. Calculations of perfusion pressure are based on measurements of intramuscular pressure and mean blood pressure in the arm, as discussed in Chapter 4 on pathophysiology.

Local perfusion pressure below 40 mmHg in traumatized legs impairs muscle blood flow and impedes normal cellular metabolism.[21] Perfusion pressure is the best parameter to study because it takes into account all the important parameters that affect tissue nutrition and viability.

TREATMENT BY FASCIOTOMY

Decompression should be done in a bloodless field whenever possible. The limb should be kept elevated for 1 or 2 min. No exsanguination method should be used. The thigh tourniquet should be inflated to 100 mmHg above systolic blood pressure. The surgical procedure seldom takes more than 5 to 10 min to perform during tourniquet ischemia. Whenever possible, fasciotomy should be done in a bloodless field by using a thigh tourniquet. An ipsilateral femur fracture or traumas to the soft tissues of the thigh are contraindications for the use of a thigh tourniquet. Surgery performed without a bloodless field takes much longer time and increases the risks for iatrogenic tissue damage, especially to the nerves.

The goal of surgical treatment is complete opening of all tight fascial envelopes. Limited skin incisions or subcutaneous fasciotomy to treat the syndrome must be avoided for several reasons. Limited exposure may not allow for complete decompression of all the compartments and "blind" surgical procedures are prone to iatrogenic tissue damage. The skin envelope contributes to external compression and a significantly elevated intramuscular pressure in patients with acute compartment syndrome.[22] The postischemic hyperemia following fasciotomy increases the volume of the limb further. Limited skin incisions may prevent a normal postoperative limb swelling caused by dysfunctional endothelial cells.

After fasciotomy is completed, the tourniquet is released. The tourniquet and the elastic support under the tourniquet, including all bandages on the thigh, are removed to prevent venous stasis. The viability of the tissues is judged after 5 to 15 min of reperfusion. An initial conservative attitude to muscular excision is recommended. The satellite cells are myogenic cells underneath the basal lamina of the

muscle fiber. They have a great potential to regenerate muscle tissue.[23–26] When they are excised, initially no muscle regeneration occurs.

Delayed treatment of acute compartment syndrome is a clinical challenge. Several authors have reported the results from treatment of patients with high injury severity score (>30), a delay of more than 35 h to treatment, and an estimated ischemic time of 56 h.[27–29] It was recommended that these patients not be treated surgically until there was a demarcation of a gangrenous part. Even extensive myonecrosis is not an indication for immediate surgical treatment. Finkelstein and co-workers concluded that the sequel of infection in a crushed limb after delayed fasciotomy is much worse than the late muscle contracture that may result from muscle fibrosis.[27] Sheridan and Matsen reported an infection rate of 46% and an amputation rate of 21% after late fasciotomy.[29]

Fasciotomy of the Foot

Between five and nine functional compartments have been described in the foot. Three of the compartments run the entire length of the foot (medial, lateral, and superficial). Five of them are located in the forefoot (one adductor and four interossei) and one is located deep in the hindfoot (calcaneal).

There are two approaches to these compartments: the dorsal and the medial. The dorsal approach to fasciotomy is performed with two longitudinal incisions centered over the second and fourth metatarsals. The medial incision provides good exposure. It can be extended proximally to decompress the tarsal tunnel and give access to the posterior tibial neurovascular bundle. The 5- to 7-cm skin incision starts on the medial side of the heel, 3 cm from the planta pedis and 4 cm from the back of the heel. It runs distally parallel to the sole (Figure 6.3). The medial compartment is opened and the abductor hallucis brevis muscle is reflected superiorly. The medial intermuscular septum is opened longitudinally. Skin closure can usually be performed 5 to 7 days after fasciotomy.

Fasciotomy of the Leg

Several techniques for decompression of leg compartments have been described.[30,31] They allow for a complete fasciotomy of all compartments. They must also allow for a postischemic hyperemia and swelling due to reperfusion.[32–34] If only the anterior or lateral compartment is affected without any swelling or tenseness of the posterior compartments, fasciotomy of the two may be enough. If two or more compartments are involved in the leg, fasciotomy of all four compartments is recommended. The recommendation is based on the clinical observation that sometimes the posterior compartments may swell later than the anterior compartment. By decompressing all leg compartments, no doubt about remaining acute compartment syndrome exists.

The Parafibular Approach

All compartments of the leg may be decompressed through a single 20- to 30-cm skin incision over the lateral compartment, 1 cm behind the anterior intermuscular septum (Figure 6.4).[30,35] The superficial peroneal nerve in the peroneus tunnel behind

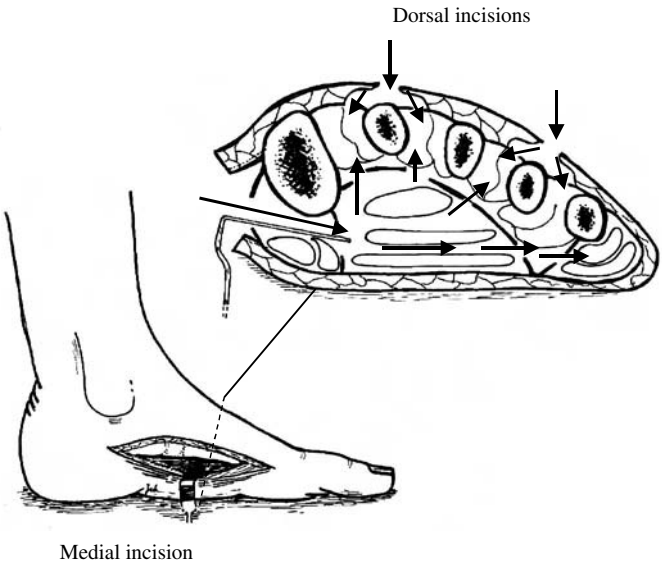


FIGURE 6.3 Fasciotomy of foot compartments. All nine compartments of the foot can be decompressed through a medial skin incision. By two dorsal incisions centered over the second and fourth metatarsals, the interosseus compartments are decompressed.

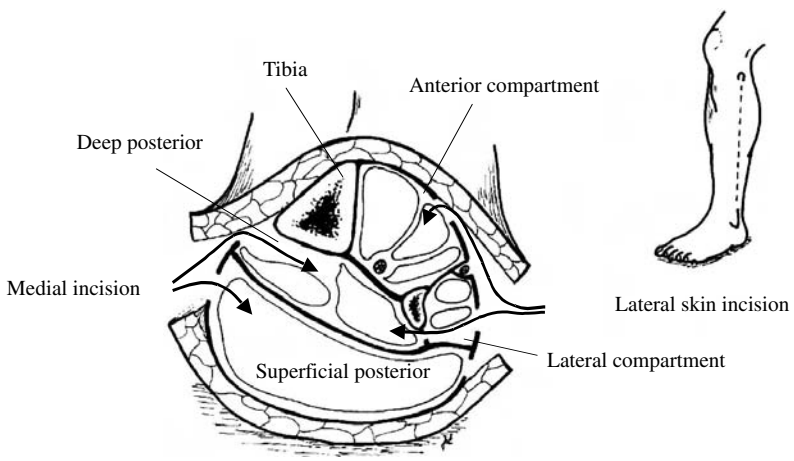


FIGURE 6.4 All leg compartments may be decompressed by a long, single skin incision extending from the fibular head to the lateral malleolus.

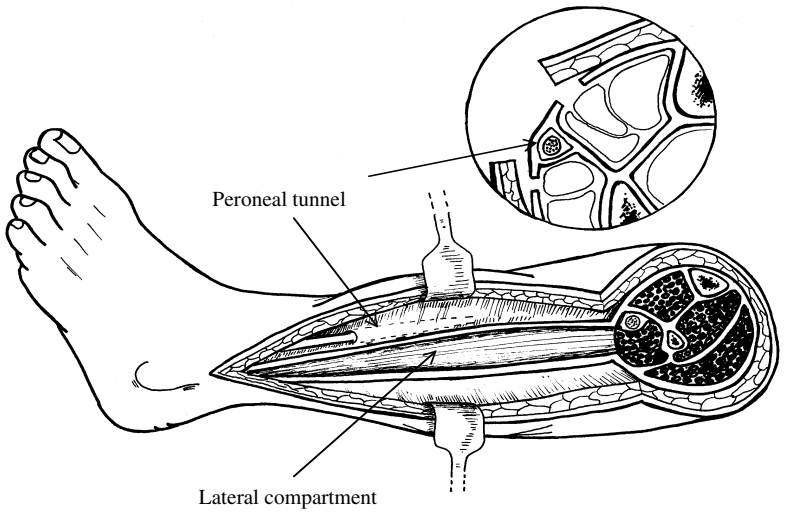


FIGURE 6.5 The peroneal nerve often runs in fascial tunnel before it pierces the fascia about 10 to 12 cm proximal of the lateral malleolus.

the anterior intermuscular septum 10 to 12 cm proximal of the lateral malleolus is identified and protected. If the nerve passes through a peroneal fascial tunnel, it may be decompressed by opening the tunnel all the way until it enters into the muscle belly proximally.[36] The lateral compartment is opened 1 cm behind the anterior intermuscular septum (Figure 6.5). The fascia over the anterior compartment is exposed by subcutaneous dissection toward the anterior ridge of the tibia. It is divided longitudinally over the proximal muscle bellies and distally all the way to the retinaculum. The line of fascial incision follows 1 or 2 cm lateral of the anterior margin of the tibia. The deep posterior compartment is opened from behind the fibula by following the posterior intermuscular septum and releasing the soleus muscle attachment from the proximal third of the fibula.

Double-Incision Fasciotomy

Double-incision fasciotomy has been advocated by Mubarak et al.[31] It is performed through a lateral and a medial skin incision. The lateral incision is identical to the parafibular approach described previously.

The superficial and deep posterior compartments are opened by a 20- to 30-cm-long skin incision 4 cm behind the posterior margin of the tibia. By this skin incision, damage to the branches of the saphenous nerves and veins is avoided to some extent. By dissecting close to the fascia toward the posteromedial margin of the tibia, the nerves and veins may be reflected anteriorly. One half of the skin on the anterior circumference of the leg should be left, and the second half of it on the posterior side. The fascia covering the soleus and gastrocnemius muscle bellies is opened. More distally and about 1 cm from the posterior margin of the tibia, the fascia over the flexor digitorum longus muscle is divided. If the soleus muscle has

a very distal attachment to the tibia, it is often necessary to detach the muscle between 3 cm and up to 8 cm from the tibia in a proximal direction to allow for full decompression of the muscles in the deep posterior compartment, including the posterior tibialis muscle. Others have suggested limited skin incisions of 15 cm[31] and even subcutaneous fasciotomy.[37] However, skin as a limiting factor for decompression in patients with acute compartment syndrome has been reported.[22] Therefore, limited skin incisions should be avoided. Furthermore, subcutaneous fasciotomies of the posterior compartment are too traumatic to the muscles, nerves, and veins. This author strongly discourages blind surgery through small skin incisions in this location.

If the patient has a contusion or other severe trauma to the pretibial soft tissues, surgery by double incision may create an anterior distally based cutaneous lambeau. This may lead to increased risk for skin and soft tissue necrosis and wound-healing problems. Therefore, this author prefers the lateral, single parafibular approach in such cases. Double-incision fasciotomy may be used in cases when surgery in bloodless field is not possible, e.g., in patents with ipsilateral femur fractures. It is a good alternative for surgeons who are not familiar with the single, lateral incision technique, which is also technically more demanding.

Transinterosseous Decompression

Through a similar skin incision as described for the parafibular fasciotomy, the lateral compartment is opened behind the anterior intermuscular septum. The superficial peroneal nerve and the peroneus longus muscle are reflected anteriorly. The posterior superficial compartment is opened by splitting the posterior intermuscular septum. The anterior compartment is opened anterior to the anterior intermuscular septum. By reflecting the extensor digitorum longus muscle and the anterior tibial neurovascular bundle forward and medially toward the tibia, the interosseous membrane is exposed. Over the tip of a right-angled clamp a puncture of the interosseous membrane is extended distally close to the fibula.[38]

Disadvantages of this technique include bulging of swollen muscles from the deep posterior compartment through the fascial incision. Shear forces at the foramen of the interosseous membrane may compress the vessels. This may induce a venous stasis of the anterior compartment because the vasculature is an end artery system. Furthermore, the interosseous membrane is a part of the stability of the proximal and distal syndesmosis between the tibia and the fibula.

In most cases of trauma, a four-compartment fasciotomy is necessary. If the syndrome develops in one compartment, other compartments are prone to reactive swelling. The symptoms of an impending syndrome may also be delayed in other compartments.

Fibulectomy

Decompression by fibulectomy[39–42] is not recommended because of its inferior results and increased risks for disability. During this procedure, the peroneal vessels and their paired venae comitantes may be damaged.[38]

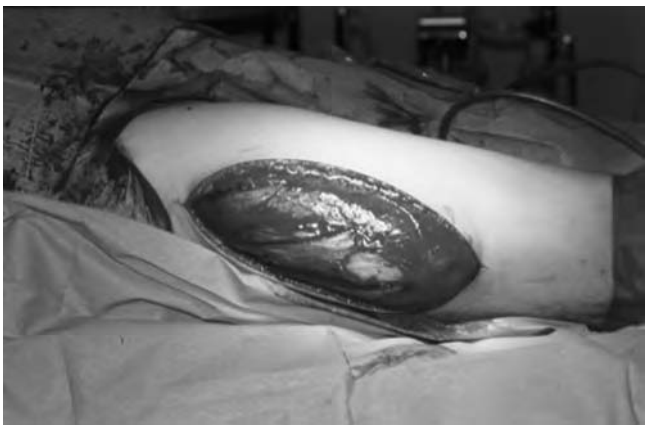


FIGURE 6.6 Intraoperative photo of the lateral thigh. The skin incision should be complete from the greater trochanter to the lateral femoral condyle. (See color insert following page 106.)

Fasciotomy of the Thigh

There are three compartments of the thigh: anterior, posterior, and medial. The psoas muscle may be considered as a separate compartment.[43] Patients with compartment syndrome of the thigh are decompressed by a lateral approach (Figure 6.6).[44] The anterior and posterior compartments are decompressed by incision of the fascia lata and the lateral intermuscular septum after retraction of the vastus lateralis muscle (Figure 6.7). The technique has been used by several authors.[45–49]

Fasciotomy of the Gluteal Compartments

The gluteal compartment syndrome is uncommon. It must be considered as a potential complication of trauma to the pelvic ring. A conservative approach has been advocated. Five out of six patients with swelling of the gluteal muscle compartments, rhabdomyolysis, myoglobinuria, and sciatic nerve palsy of the contralateral leg were treated conservatively. Symptoms resolved in all of them.[50] Isolated gluteal compartment syndrome does not increase the risk for crush syndrome.[51] If the patient has clinical signs of simultaneous active bleeding, arteriography of the pelvis may be performed before a fasciotomy.[52]

Skin incision to decompress the gluteal compartments may be a curved incision parallel to the iliac crest or a posterior curved incision from the posterior iliac spine to the greater trochanter.[53] A third alternative is exposure with a double curved incision that runs from the posterior iliac spine over the greater trochanter down to the level of inferior gluteal fold. The incision is then brought medially beneath the buttocks and down to the midposterior thigh (Figure 6.8).[54] The tensor fascia lata is opened by fasciotomy.[51,55] The gluteus maximus muscle is decompressed by multiple epimysiotomies.[56] The gluteus medius is decompressed by epimysiotomy.[51] In patients with sciatic or peroneal nerve palsy, the sciatic nerve must be inspected intraoperatively to exclude direct damage to the nerve.

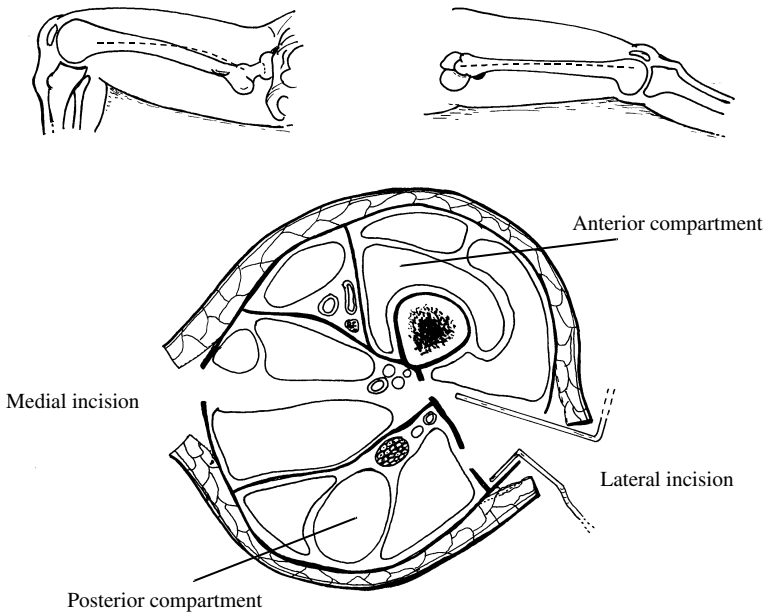


FIGURE 6.7 The three thigh compartments may be decompressed through a single, straight lateral skin incision or through an additional medial skin incision.

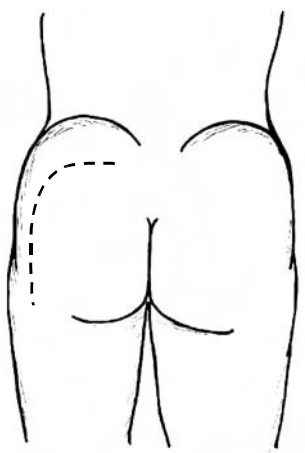


FIGURE 6.8 Gluteal compartments can be decompressed by a curved incision parallel to the iliac crest to the greater trochanter. The gluteal muscles may need multiple fasciotomies and epimysiotomies because they are multipennated.

Fasciotomy of the Dorsolumbar Compartment

Fasciotomy may be done through separate skin incisions between 2 and 4 cm lateral to the spinous processes. A midline skin incision may also be used.[57]

Fasciotomy of Shoulder Muscles

The shoulder includes several compartments. The supraspinatus, infraspinatus, and subscapularis muscles are enclosed in osteofascial compartments, whereas the trapezius and deltoid muscles have more compliant locations. The possibility of a compartment syndrome of the shoulder has been suggested.[58] However, there are few reports of the syndrome in this location.[58–60] The supraspinatus muscle generates high intramuscular pressure during contraction.[61] However, this is not a sign of volume load of the muscle, but rather a sign of its muscular architecture and force generation.[62]

Fasciotomy of the Upper Arm

The upper arm has two compartments: the extensor compartment enclosing the triceps muscle and the flexor compartment enclosing the biceps muscle bellies. The compartment is less rigid than other leg and arm compartments. A longitudinal incision on the medial aspect of the arm from the shoulder to the elbow over the medial intermuscular septum will allow for decompression of both the biceps and triceps compartments (Figure 6.9). Delayed primary closure may be performed after 4 or 5 days.[63]

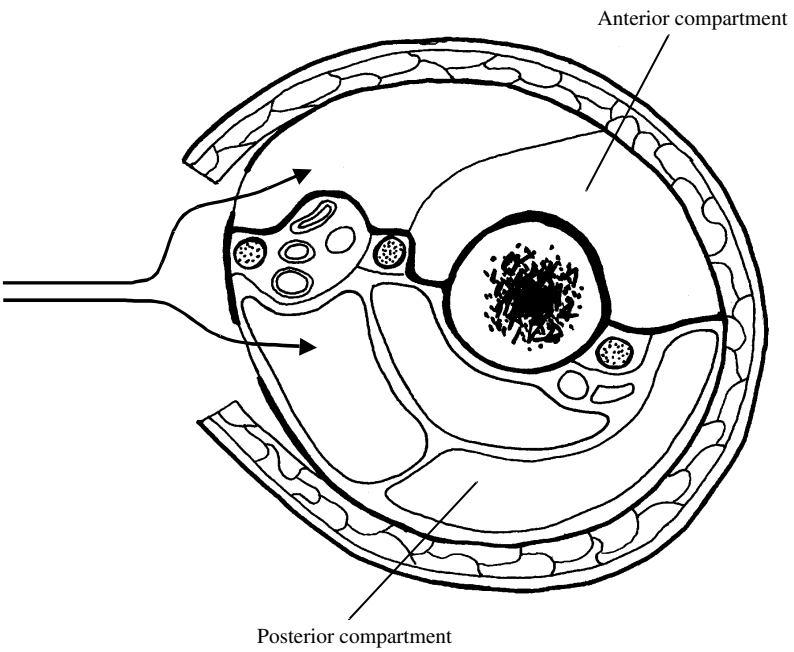


FIGURE 6.9 The anterior and posterior compartments of the upper arm can be reached by a skin incision over the medial neurovascular bundle.

Fasciotomy of the Forearm

Four compartments of the forearm have been described: the superficial and deep volar, the dorsal, and the extensor indicis proprius muscle, which is located in a different fascial envelope and may need a separate fasciotomy. Surgery should be done in a bloodless field whenever possible.

Fasciotomy of the Volar Compartments

The volar incision according to Gelberman[64] originates 2 cm proximal to the medial epicondyle. It is extended obliquely across the antecubital fossa and further distally to reach the volar aspect of the forearm. The incision is extended ulnar to the palmaris longus tendon to avoid injury to the cutaneous branch of the median nerve. It crosses the wrist crease at an angle to avoid exposure and transmission of tensile forces during wound healing. The incision is extended into the midpalm (Figure 6.10). A curvilinear skin incision has also been described. Finally, the volar aspect of the forearm can be decompressed by a straight volar incision on the ulnar side, which starts proximal to the elbow and radial to the biceps tendon.[64] The superficial flexors are decompressed by a complete fasciotomy (Figure 6.11A). The deep flexors are exposed by retracting the superficial flexors (Figure 6.11B). The tourniquet used for a bloodless field is removed and muscles are mobilized, inspected, and judged for viability signs. Individual muscles may need epimysiotomy. Liquefied muscles should be removed at this time. The median nerve in the carpal tunnel is decompressed to avoid an edge effect on the nerve at its proximal entrance in the tunnel. The median nerve may also need proximal decompression at the lacertus fibrosis and at the pronator teres muscle as well.[65] The ulnar nerve is decompressed when indicated by clinical signs by opening the Guyon's tunnel.

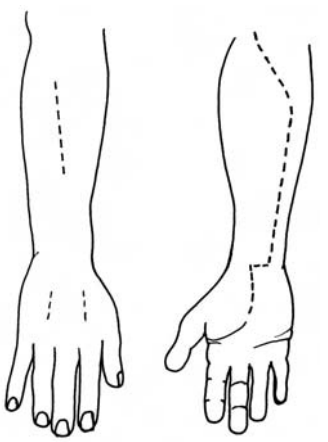


FIGURE 6.10 Suggested skin incisions to decompress the compartments of the forearm and the interossei muscles of the hand. The carpal tunnel must be decompressed in all cases of acute compartment syndrome of the forearm.

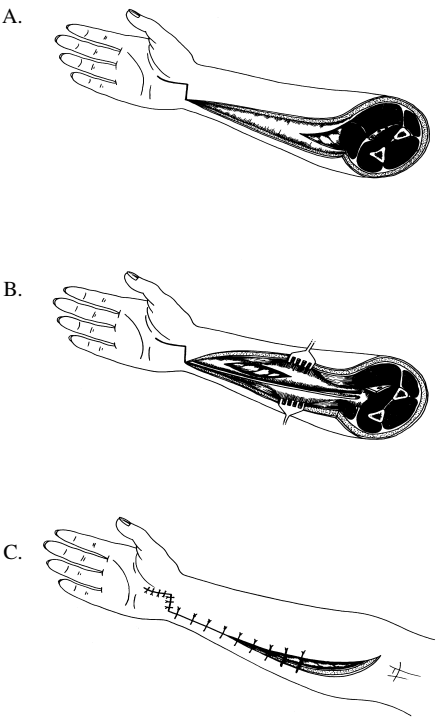


FIGURE 6.11 Decompression of the volar compartments of the forearm and secondary suture of the wound. (A) The carpal tunnel and the superficial flexors are decompressed. (B) By retracting the superficial flexors, the deep compartment is decompressed. (C) The skin over the carpal tunnel should be sutured primarily whenever possible.

Fasciotomy of the Dorsal Compartment

A straight longitudinal incision is carried from 5 cm distal to the lateral epicondyle and extended about 10 cm distally. The fascia covering M. extensor indicis proprius may need a separate fasciotomy.

Fasciotomy of the Hand

The hand includes six to ten compartments. The ten compartments include the hypothenar, thenar, adductor pollicis compartments, the four dorsal interossei compartments, and the three volar interossei compartments. All compartments with abnormally increased intramuscular pressure must be treated. Besides, most patients with acute compartment syndrome of the hand must have a carpal tunnel decompression and sometimes also decompression of the ulnar nerve.[66] The dorsal interossei muscles may all be reached through two dorsal skin incisions, one over the second metacarpal bone and another over the fourth metacarpal bone (Figure 6.10). It is important to decompress the carpal tunnel and Guyon's tunnel in all cases of sensory dysfunction.

POSTOPERATIVE MANAGEMENT

Edema Reduction

Intensive edema-reducing therapy is important following surgery by fasciotomy. Edema-reducing methods are outlined in Chapter 18. They include active muscle contractions, passive muscle stretch, intermittent external compression, limb elevation, and hyperbaric oxygenation.

Following decompression, the limb is immobilized. Early active exercises to reduce edema formation are important. Following successful edema reduction, the wound may be closed by secondary closure after 3 to 4 days in many patients.[35,67] Patients with acute compartment syndrome of the leg and soft tissue trauma to the ipsilateral thigh are more difficult to treat. These patients may need repeated wound adaptation 5 to 7 days after trauma. Secondary wound closure is also more difficult in patients with diabetes and in those who cannot walk or activate their limb muscles for other reasons. Continuous epidural anesthesia should therefore be used cautiously. This author's experience is that this treatment of pain induces muscular dysfunction that impairs edema reduction. Alternative pain treatment should be considered in these patients if muscular activity cannot be maintained. Edema reduction by concentric muscular activity is a prerequisite for successful early secondary wound closure.

Secondary Wound Closure by Dermatotraction

Creep occurs when skin is stretched under a constant force. The skin will continue to extend if the force is kept constant. Stress relaxation occurs when the skin is stretched for a given distance, which is held constant. Several techniques are available to facilitate the closure of wounds following fasciotomy for acute compartment syndromes, such as (1) progressive closure by wire sutures or tape, (2) ETE tension bands,[67,68] (3) dermatotraction by Sure-Closure®,[69] (4) a rod-tensioning device,[70] or (5) Kirschner wires.[71]

Secondary Closure by Wire Sutures

Secondary wound closure by wire sutures may be done on the third or fourth day following surgery if treatment by edema reduction has been successful.[35] Placement of static tensioning devices across the wound includes tape[30] or sutures,[35] which are replaced or tightened. Intramuscular pressure must not exceed 30 to 35 mmHg at secondary wound closure.[35] Treatment in this way makes skin transplantation unnecessary. The results of secondary wound closure are cosmetically more appealing than skin transplantation (Figure 6.12). The wound heals quicker and the time for in-hospital stay is shorter.

Silicon or Rubber Bands

Elastic tensioning devices have the advantage of providing continuous pull on wound edges. They may be tightened without replacement and without the need for anesthesia.[72] Tightening is performed by pulling each segment of the vessel loop. The

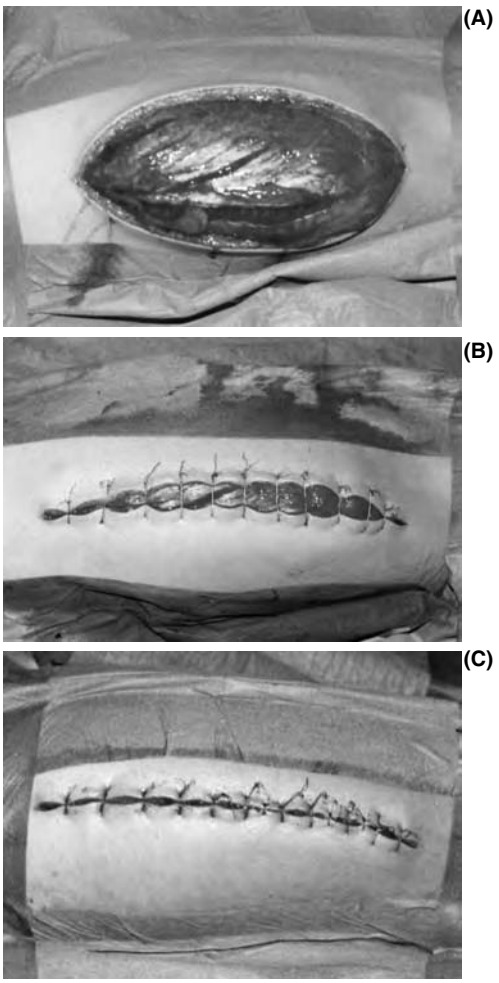


FIGURE 6.12 Secondary wound closure by wire sutures in a patient 3 days after fasciotomy for acute compartment syndrome of the thigh. (A) The lateral thigh wound 3 days after fasciotomy. (B) Wire sutures are applied 3 days after fasciotomy. (C) On Day 5, the sutures are tightened to completely close the wound. (See color insert following page 106.)

excess of the loop may be tied to the last staple. By using an elastic vessel loop shoelace, delayed primary closure is achieved within 9 days.

External Tissue Extender (ETE)

Each ETE unit consists of a silicon band connected to two friction stoppers. The units may be connected to each other by hinges (Figure 6.13). The silicon bands can be tightened twice daily without anesthesia. In this way, the wound edges are approached. When the wound gap is between 10 and 15 mm, it can be closed by ordinary sutures. The ETE technique is a valuable tool in patients who have difficulties in reducing their

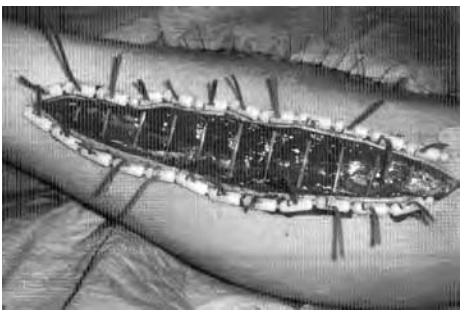


FIGURE 6.13 Dermatotraction by external tissue extender (ETE) in a patient treated by parafibular fasciotomy due to acute leg compartment syndrome. Treatment by external tissue extender was initiated on Day 3 after decompression.

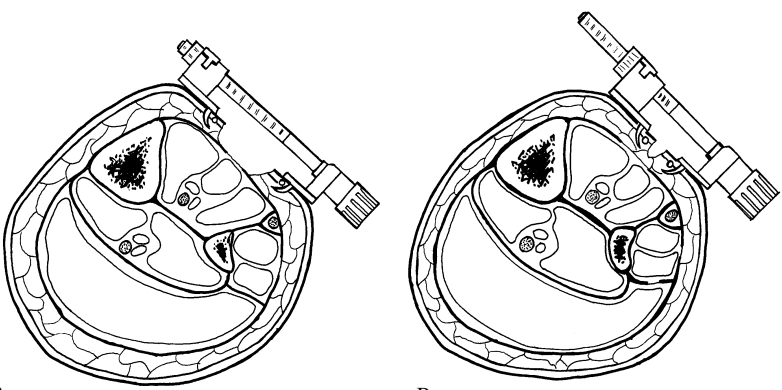
limb edema. The ETE device decreases limb volume by increasing the interstitial hydrostatic pressure to levels that do not impair local perfusion pressure.[35,67]

Sure-Closure

The device consists of two 7.5-cm-long pins, each of which is threaded through the dermis on either side of the wound. Two U-shaped arms are hooked to the pins (Figure 6.14). The distance between the wound edges is decreased and a threaded screw that passes through the arm increases the tension. By using a tension of 3 N or less, intramuscular pressure in the leg remains under 30 to 35 mmHg.[73]

Other Methods of Dermatotraction

A simple method using 2-mm Kirschner wires through the skin has been described (Figure 6.15).[71] The skin edges can also be approached by another technique that



A. B.

FIGURE 6.14 (A) Schematic illustration of secondary wound closure by Sure-Closure. (B) The tensile forces acting over the wound edges can be kept constant at a desirable level by a threaded screw.

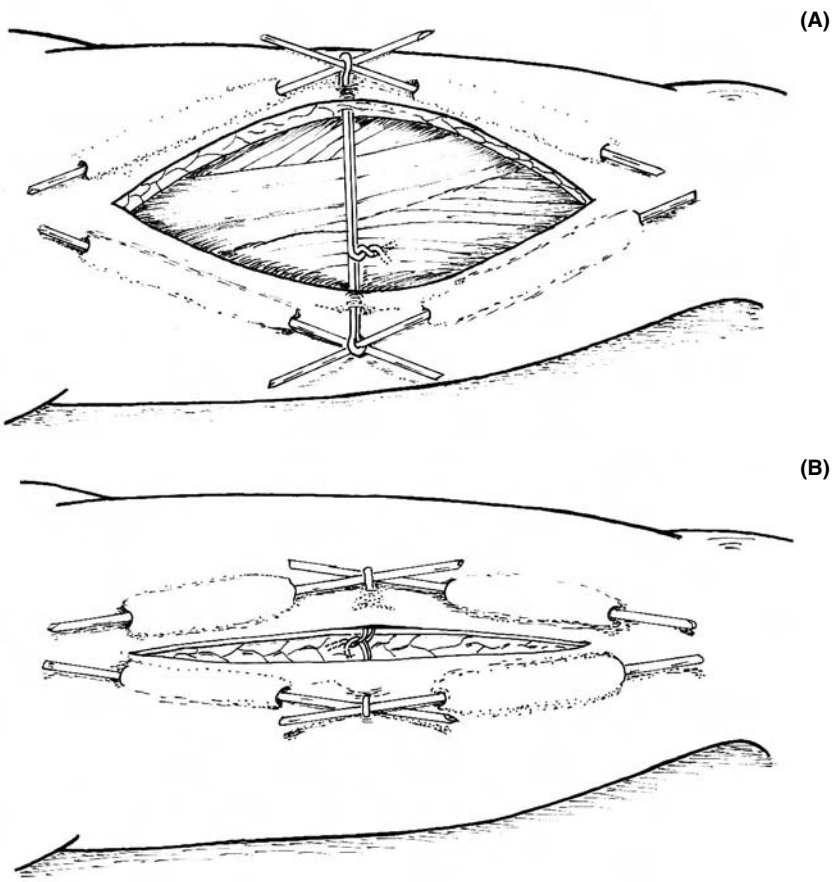


FIGURE 6.15 (A) Subcutaneously inserted Kirschner wires, which are caught by a wire suture at the crossing point. (B) At this time the skin edges are approached. The sound may be closed by ordinary stitches.

includes a rod-tensioning device combined with a transparent polyurethane adhesive sheet that sticks firmly to the skin and to the rod, but not to the moist wound. Each day the rod must be lifted and turned one complete turn and then laid back in the rack, which prevents unrolling.[70]

Wounds that are treated by delayed closure are as strong as primary closed wounds. This means that an ischemic wound can safely be left open for 3 days before suturing without affecting the biomechanical properties.[74]

Vacuum-Assisted Wound Closure

The wound is exposed to subatmospheric pressure of about 125 (50 to 200) mmHg. A sterile polyurethane sponge is cut to fit with the entire wound surface. An adherent plastic sheet is placed to cover the wound and the tubing, which is connected to a reservoir and a pump. Edema fluid is removed from the extravascular space. The

mechanical tension stimulates proliferation of granulation tissue. Following fasciotomy, a higher proportion of vacuum-treated wounds undergo primary closure, making skin grafting unnecessary.

Skin Grafting

Skin grafting gives poor coverage and sensation to the wound, and it is less appealing cosmetically. In most cases, skin grafting can be avoided following fasciotomy for acute compartment syndrome. The key point is to reduce the posttraumatic limb edema early after fasciotomy.

Role of Hyperbaric Oxygenation

Hyperbaric oxygen therapy provides hyperoxygenation of the tissues. Oxygen dissolves in plasma in direct proportion to its partial pressure. Treatment by hyperbaric oxygenation is therefore a supplement of the oxygen-carrying capacity of red blood cells. Tissues can remain viable without hemoglobin-borne oxygen if the patient breathes pure oxygen at 2.4 atm absolute pressure. Treatment by oxygen induces mild vasoconstriction and simultaneously elevates partial pressure of oxygen levels. Vasoconstriction lowers capillary pressure, altering the Starling equilibrium in a way that promotes absorption of interstitial edema. Injury induced by ischemia-reperfusion can be reduced by hyperbaric oxygenation treatment.

Hyperbaric oxygen therapy has been used as an adjunctive treatment in patients with ischemic injury following trauma. The high oxygen tension in arterial blood achieved with hyperbaric oxygen therapy increases the wound oxygen tension in previously hypoxic wounds.[75] Hyperbaric oxygenation restores oxygen tension to normal and enhances healing. It reduces postischemic edema[76] and skeletal muscle necrosis in skeletal muscle injury[77] and in acute compartment syndrome.[78,79] It has been suggested that treatment by hyperbaric oxygenation can arrest the progression of abnormally increased intramuscular pressure during the lag phase in acute compartment syndrome.

When tissue oxygenation levels decrease, the ability of leukocytes to handle infection is markedly reduced. A tissue oxygen tension of 30 mmHg (4 kPa) is required for fibroblast function during wound healing. Thus, treatment by hyperbaric oxygenation may maintain tissue viability during an initial hypoxic phase, prevent infection, promote healing, and restore function of the injured tissues. It improves metabolic restitution in postischemic skeletal muscle.[80]

Hyperbaric oxygenation treatment enhances the recovery of blood flow and functional capillary density in postischemic tibialis anterior muscle.[81] It improves wound healing. Hyperbaric oxygenation is recommended as a useful adjunct in management of severe (Grade III) crush injuries of the limbs in patients over 40 years of age.[82] Hyperbaric oxygenation can be used in patients with obvious necrosis at the time of decompression, when massive edema exists and in patients who have impaired ability to reduce edema by other means, e.g., patients with neuropathy.

SUMMARY

The goal of treatment of acute compartment syndrome is to restore normal neuromuscular function. This is achieved by normalizing the local perfusion pressure, which is obtained by decreasing the abnormally elevated intramuscular pressure. The following six easy steps may treat the syndrome. Local perfusion pressure in the compartment can be restored by (1) cutting all dressings that give external compression, (2) concluding limb elevation, (3) increasing mean arterial pressure by treating hypovolemia, and (4) normalizing intramuscular pressure by fasciotomy. Whenever possible, fasciotomy should be performed in a bloodless field. The tourniquet should be removed from the limb immediately after pressure is deflated. (5) The fifth step includes treatment of postischemic reperfusion and the crush syndrome that may follow fasciotomy by early active edema reduction. (6) The sixth step is prescribing treatment for edema reduction. Concentric muscular activity is one of the most powerful edema-reducing mechanisms. This should be taught to the patient. Following successful edema reduction, the wound may be closed 3 to 5 days after fasciotomy in most cases. In some cases, closure by dermatotraction is very helpful. Skin grafting can be avoided in most patients. Hyperbaric oxygenation is recommended as a useful adjunct in management of severe crush injuries, especially in patients over 40 years of age.

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7 Acute Abdominal Compartment Syndrome

INTRODUCTION

Today more patients survive the initial phase of high-energy multitrauma that once was fatal. Therefore, patients with multifactorial organ dysfunction, including patients with abnormally elevated intraperitoneal hydrostatic pressure, are more likely seen. Abnormally elevated intraabdominal pressure may cause acute abdominal compartment syndrome. Diagnosis and treatment of the syndrome are difficult because patients have multiple injuries and present a complex clinical picture. The elevated intraabdominal pressure induces respiratory, cardiovascular, renal, and visceral dysfunction. It even affects the central nervous system in humans.[1,2] Acute abdominal compartment syndrome is lethal if left untreated.

Elevated intraabdominal pressure, increased intraperitoneal hydrostatic pressure, and intraabdominal hypertension have been used synonymously. They represent clinical conditions that are initially benign and reversible. When intraabdominal pressure is abnormally elevated or intraabdominal hypertension acts during an extended period of time, the condition may evolve to an acute abdominal compartment syndrome. The syndrome is irreversible and requires surgical treatment by decompression. The mortality rate among patients with acute abdominal compartment syndrome is up to 60%.[3] In a review, Schein and co-workers reported on 45 patients with acute abdominal compartment syndrome; 19 of these (42%) died.[4]

DEFINITION

Acute abdominal compartment syndrome is defined as increased intraperitoneal hydrostatic pressure to levels that impair local blood perfusion pressure and induce dysfunction of the intra- and retroperitoneal organs. The elevated intraabdominal pressure induces respiratory, cardiovascular, and renal dysfunction. Acute abdominal compartment syndrome is the end result of a progressive elevation of intraabdominal pressure that leads to multiple organ dysfunction.

Intraabdominal hypertension has been defined as intraabdominal pressure exceeding 15 to 18 mmHg (= 20 to 25 cm H_2O).[5] It is a preliminary stage that, when left untreated, may lead to a clinically manifest acute abdominal compartment syndrome. Intraabdominal hypertension can be considered an early indication of acute abdominal compartment syndrome.

Incidence

The incidence of acute abdominal compartment syndrome varies with clinical setting and definition of the syndrome. In a study, the syndrome was diagnosed in 21 of 145 patients (14%) with abdominal trauma and with injury severity score exceeding 15 points.[6] In this study, 60% of patients had blunt trauma and 67% required abdominal packing during the initial operation. In other studies, the incidence of the syndrome is reported to be 4% in patients with repair of aortic rupture[7] and 15% in patients who required laparotomy and packing.[3]

Historical Perspectives

The associations between increased intraabdominal pressure and dysfunction of different abdominal and thoracic organs have been known for more than 100 years. Weber in 1851 and Donders in 1854 suggested that physiological oscillations of intrathoracic and intraabdominal pressures during respiration assisted venous return from the abdominal to the thoracic cavity.[8] Later, Marey in 1863 and Bert in 1870 demonstrated the association between respiration and intraabdominal pressure.[9,10] In 1878, Quincke showed that abdominal pressure in patients with ascites exceeded atmospheric pressure.[8] He also described that increased abdominal pressure impaired venous return from the viscera. Emmerson concluded that distension of the abdomen by fluid or gas raises abdominal pressure.[8]

In 1890, Heinricus described pulmonary dysfunction secondary to increased intraabdominal pressure in an experimental study, and in 1900 Schaefer pointed out that the tone of the abdominal wall muscles is regulated by the respiratory center.[8,11] Intraabdominal pressure exceeding 20 to 33 mmHg killed small animals by fatiguing the diaphragm and diminishing venous return to the heart.[8]

The effects of increased intraabdominal pressure on renal function were first described by Wendt in 1873 and later by Bradley and Bradley in 1947.[12,13] They reported that increased intraabdominal pressure induced oliguria. It has been shown in experimental studies that constant distension of the abdomen by inflating a balloon, which had been surgically inserted into the abdomen, caused death.[14] Renal decapsulation was first performed by Harrison in 1896.[15]

In the 1940s, mortality rates following treatment of large omphaloceles by forceful closure of the abdominal wall was reported. The abdominal crowding induced respiratory failure and cardiovascular collapse. Later, in the 1960s and 1970s, physicians became more aware of the potentially hazardous effects of increased intraabdominal pressure during laparoscopy.[16] The term *acute abdominal compartment syndrome* was first used by Kron et al. in 1984.[17] Currently, the syndrome refers to the consequences of abnormally elevated intraabdominal pressure regardless of cause.

ETIOLOGY AND PATHOGENESIS

Acute abdominal compartment syndrome may follow posttraumatic hemorrhage and abdominal surgery. The mechanisms that induce acute abdominal compartment

syndrome are usually multiple and the pathogenetical factors are additive.[18,19] The syndrome has also been documented in patients with tense ascites, necrotizing pancreatitis, megacolon, and retroperitoneal hematoma.[7,20,21] Three different mechanisms may increase intraabdominal pressure to abnormal levels: (1) increased intra- or retroperitoneal volume, (2) external compression, and (3) decreased size of the abdominal compartment.

Increased Intraabdominal Volume

Hemorrhage, edema, bowel distension, obstruction of mesenteric veins, tense ascites, and abdominal packs, as well as peritonitis and tumor cause increased intraabdominal volume.[3,22–26] Severe bowel edema may follow prolonged ischemia, e.g., following surgery on the abdominal aorta.[7,27,28] Laparoscopy with CO_2 pneumoperitoneum has been shown to have adverse effects on cardiovascular and renal function in experimental[29] and clinical studies.[30–32] Acute abdominal compartment syndrome can occur with no abdominal injury in patients with severe hypovolemic shock[33] and in patients with coagulopathy.[28]

External Compression

External compression of the abdomen increases intraabdominal pressure. This may be caused by pneumatic antishock garments[34,35] and by tight abdominal closures.[3,22,26] External compression by military antishock trousers may also increase intraabdominal pressure.[36–38]

Decreased Compartment Size

Repair of abdominal wall defects or large incision hernias may increase intraabdominal pressure by decreasing the compartment size. Decreased abdominal volume may be due to loss of the abdominal wall and to increased tension of the abdominal wall muscles. Increased intrathoracic pressure may also depress the diaphragm and thereby decrease the abdominal volume, which results in increased intraabdominal pressure.

Elevated intraabdominal pressure is seen in patients with burn injuries with abdominal eschars.[34,35] Acute abdominal compartment syndrome was reported in two patients with more than 70% of the body surface burned.[39] Patients with circumferential torso burns are at increased risk for the syndrome.

PATHOPHYSIOLOGY

Hydrostatic pressure in the peritoneal cavity interacts with pressure in the thoracic cavity, retroperitoneal space, and lower extremities. Understanding the mechanical and physiological interaction between increased intraperitoneal pressure and other body compartments is helpful in the clinical diagnosis of patients with severe abdominal injury (Figure 7.1).

Intraabdominal pressure exceeding 10 to 25 mmHg decreases cardiac output, induces respiratory dysfunction, and declines renal function. Multiorgan system failure evolves if the pressure is not promptly relieved.[18] Abnormally elevated

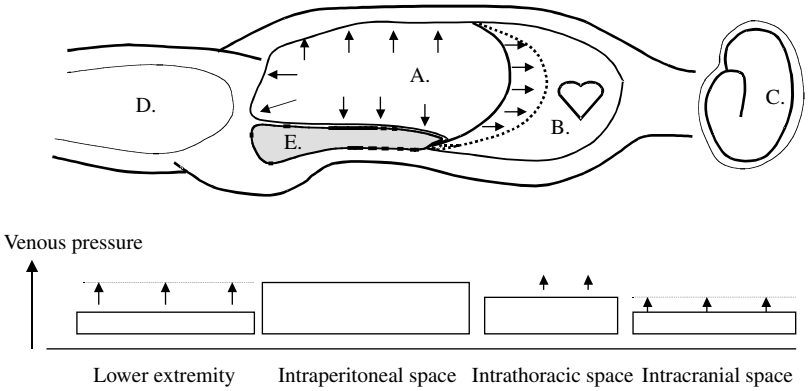

FIGURE 7.1 The intraperitoneal compartment (A), the intrathoracic compartment (B), the intracranial compartment (C), the extremity compartments, (D) and the retroperitoneal compartments (E). The figure illustrates how elevated intraabdominal pressure is transmitted to the other compartments by pushing the diaphragm upward. The elevated intraperitoneal venous pressure is also transmitted by the waterfall mechanism (pooling of blood) to all the other body compartments. Patients with abnormally elevated intraperitoneal hydrostatic pressure have pulmonary and cardiovascular dysfunction. Intracranial and extremity pressures increase due to venous stasis.

intraabdominal pressure impairs renal blood flow, pulmonary function, and cardiac function. It may cause bowel ischemia and affect intracranial pressure.

Increased intraabdominal pressure reduces abdominal wall blood flow, which leads to ischemia and edema.[40] Both the venous return and cardiac output fall as intraabdominal pressure rises as little as 10 mmHg.[41] The effects of increased intraabdominal pressure on respiratory and cardiovascular, renal, visceral, and neurologic function are discussed next.

Respiratory Function

Elevated intraabdominal pressure pushes the diaphragm upward (Figure 7.1). The total lung capacity and functional residual capacity decrease by 40% when intraabdominal pressure increases from about 2 to 12 mmHg.[42] This may lead to compression of the lung tissue and atelectasis, which in turn may create a shunt of unoxygenated blood.[18,41,42] The volume and compliance of the thoracic cavity decrease. The end inspiratory pressure increases. The dynamic and static compliances of lung tissue decrease. Tissue oxygenation decreases and carbon dioxide increases.[43] The pulmonary end expiratory pressure required to deliver a fixed tidal volume increases significantly by intraabdominal pressure above 25 mmHg. Pulmonary wedge pressure increases with increasing intraabdominal pressure.[44] It has been suggested that the high pulmonary elastance seen in experiments of increased intraabdominal pressure is due to decreased chest wall compliance associated with decreased intrathoracic volume because of the elevated diaphragm.[7] Peak inspiratory pressure is high during mechanical ventilatory support.[18] Increased intraabdominal pressure is a major determinant of respiratory dysfunction in diaphragmatic rupture.[45]

Cardiovascular Function

Cardiac Function

Intraabdominal pressure of 10 to 15 mmHg decreases cardiac output significantly.[41] By 20 mmHg, cardiac output decreases by 15% in normovolemic dogs and by 50% in hemorrhaged dogs.[46] The elevated intrathoracic pressure impairs the stroke volume, decreases venous return, and decreases cardiac filling pressure as well as increases vascular resistance of the systemic circulation.[41,47,48] Intraabdominal pressure of 40 mmHg produced by inflation of a pneumatic antishock garment decreases cardiac output.[45] In summary, cardiac depression is related to increased peripheral resistance[46] and diminished venous return,[41] or both.

Arterial Pressure

The mean arterial pressure of the lower extremities is not affected by elevated intraabdominal pressure. In an experimental study, the mean arterial pressure did not change significantly when intraabdominal pressure was elevated to 20 mmHg during pneumoperitoneum.[49] Little difference between brachial and femoral arterial pressure was observed when intraabdominal pressure was increased up to 70 mmHg.[50] This indicates that mean arterial pressure in the lower limbs is not decreased by abnormally elevated intraabdominal pressure.

Venous Return

Increased intraabdominal pressure is transformed to all intraabdominal and retroperitoneal veins. Increased intraabdominal pressure is also transformed into the retroperitoneal space (Figure 7.1). About 90% of the increased intraabdominal pressure is reflected in femoral venous pressure.[50,51] This means that changes of inferior vena cava pressure parallel intraabdominal pressure. Venous return depends on the resistance of the veins and on the upstream driving pressure for venous return. It has been shown to be impaired at an intraabdominal pressure of 15 mmHg[46] and 30 mmHg.[52] Rubinson and co-workers showed with angiographic observations in experimental studies that the inferior vena cava was obstructed at the diaphragm when the hydrostatic pressure difference was 28 mmHg.[52] Venous pressure in the limbs increases to levels above intraabdominal venous pressure.

In normovolemic patients, cardiac output usually does not fall significantly until intraabdominal pressure is 40 mmHg.[53] This pressure level increases pressure in the vena cava inferior and venous return from the lower extremities decreases.[54] The mechanism for this is the waterfall phenomenon (Figure 7.1).[55,56] In hypovolemic conditions, even lower abdominal pressures can be harmful because both the inferior vena cava and the arterioles are collapsed at lower pressures.

Perfusion Pressure

Elevated intraabdominal pressure decreases perfusion pressure of the intraabdominal organs. Elevated tissue hydrostatic pressure and venous pressure are the causes for the decreased perfusion pressure.

Renal Function

Postoperative anuria or oliguria in a patient after closure of a tensely distended abdomen may be caused by abnormally elevated intraabdominal pressure. Intraabdominal pressure of 20 mmHg impairs renal function by reducing glomerular filtration rate and renal blood flow.[47] Intraabdominal pressure of 30 to 40 mmHg may induce anuria.[18,27,29,47,57] Renal vascular resistance increases over 500% when intraabdominal pressure is elevated from 0 to 20 mmHg.[18,57] The dysfunction is reversible.

It has been reported that 80% of the inflated pressure of the antigravity suit is transmitted to the retroperitoneal space.[36] Harman et al. showed that impaired renal function was not due to urethral compression.[47] They observed no significant difference in any parameters of hemodynamic or renal function in dogs with and without urethral stents. Therefore, urethral compression was excluded as a cause of renal dysfunction. One reason for this may be that the renal collecting system can generate hydrostatic pressures up to 90 mmHg. It has been suggested that direct compression of the kidneys by increased intraabdominal pressure may elevate cortical pressure, leading to an acute renal compartment syndrome.[20] Sugrue and co-workers found an association between increased intraabdominal pressure and renal impairment in patients admitted to an intensive care unit after laparoscopy.[58]

Visceral Abnormalities

The gut is the most sensitive organ to elevated intraabdominal pressure. Elevated intraabdominal pressure may reduce visceral blood flow, induce ischemia, and cause organ dysfunction.[48] Intraabdominal pressure of 20 mmHg decreases mesenteric arterial blood flow significantly. At an intraabdominal pressure of 40 mmHg, the pH falls to levels indicating severe mucosal ischemia which may be as important as the pulmonary, cardiac, and renal changes.[44] In an experimental study, all viscera except the adrenal glands showed a marked reduction in blood flow beyond that attributable to the decrease in cardiac output.[48] Caldwell and Ricotta found that the changes in organ blood flow were more marked than could be accounted by changes in cardiac output alone.[48]

Despite normal systemic hemodynamics, splanchnic ischemia may occur with slightly elevated intraabdominal pressure. Such ischemia may be associated with an increased incidence of multiple organ failure and mortality.[59] Intestinal ischemia has been reported during prolonged laparoscopy in patients with normal central hemodynamics.[60,61] Splanchnic hypoperfusion and gut acidosis commence at lower intraabdominal levels and long before the manifestations of abdominal compartment syndrome become clinically evident.[5] Intestinal low-grade ischemia is seen at an intraabdominal pressure of 15 mmHg and is associated with bacterial translocation.[62]

Neurologic Abnormalities

Increased intraabdominal pressure elevates intracranial pressure and reduces cerebral perfusion pressure.[1,2,63] Bloomfield et al. also showed that the increased intracranial pressure was due to elevated intrathoracic pressure.[1] The change of intracranial

pressure is induced by elevated intrathoracic pressure and central venous pressure (Figure 7.1). Intracranial pressure and hemodynamic parameters normalized when pleural pressure was kept at atmospheric level.

Reduced intraabdominal pressure by decompression normalized intracranial pressure in these experiments. In another study, sternotomy and pleuro-pericardiotomy prevented the rise of intracranial pressure.[19] Therefore, patients with both increased intraabdominal and head injury may be at risk for profound injury to the central nervous system.

Abdominal Wall Abnormalities

The compliance of the peritoneal cavity is 20 times greater at atmospheric pressure compared to that at an intraabdominal pressure of 40 mmHg.[50] Laplace law may be applied to the abdominal cavity. The tension of the abdominal wall is described by the relationship $T = p \times r$. Tension of the abdominal wall (T) is directly proportional to the intraabdominal pressure (p) and the radius (r) of the abdominal cavity. Increased intraabdominal pressure may cause abdominal muscle ischemia, increased risks for infection, and herniation.[40]

MEASUREMENT OF INTRAABDOMINAL PRESSURE

Intraabdominal pressure may be measured directly with an intraperitoneal catheter attached to a transducer. Indirect methods to estimate intraabdominal pressure include urinary bladder pressure, gastric pressure, and pressure in vena cava.

A grading system based on intraabdominal pressures has been proposed. Grade I is intraabdominal pressure between 10 and 15 cm H_2O, Grade II is 15 to 25 cm H_2O, and Grade III is between 25 and 35 cm H_2O.[16] This grading system has been correlated to clinical findings and suggested treatment.

Direct Measurements

Intraabdominal pressure can be measured directly by a fluid-filled intraperitoneal catheter connected to a transducer or, if not available, a manometer.[4,8,17,18,40,64–67] Blunt techniques similar to measurements of intramuscular pressures may be used.[40,64–66] The CO_2 insufflators for laparoscopy can be used to automatically increase and measure intraabdominal pressure.

Urinary Bladder Pressure

Intraabdominal pressure may be indirectly determined by measuring urinary bladder pressure by a Foley catheter.[17] Bladder pressure closely reflects intraperitoneal pressure.[68] However, patients with small neurogenic bladder or intraperitoneal adhesions as well as patients with obesity, pregnancy, or ascites may give unreliable estimates of intraabdominal pressure.[20]

The bladder is drained and then filled with 50 to 100 mL of sterile saline through the Foley catheter. The tubing of the collecting bag is clamped. The catheter is connected to a pressure transducer. The symphysis pubis is the zero

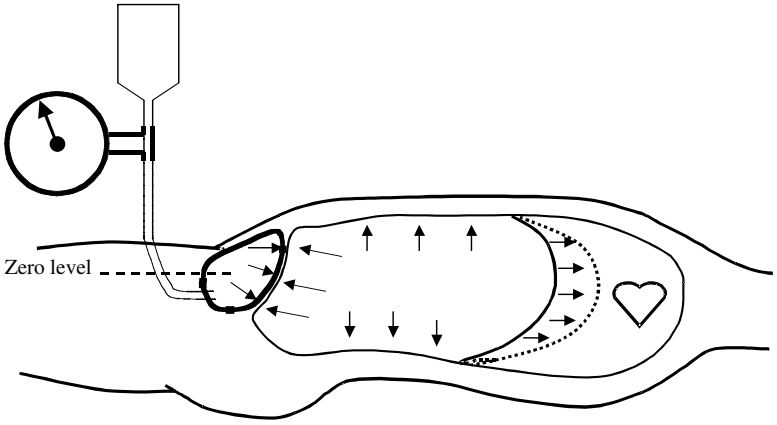

FIGURE 7.2 Indirect determination of intraperitoneal pressure by measuring urinary bladder pressure. The bladder is drained and then filled with 50 to 100 mL of sterile saline through the Foley catheter. The tubing of the collecting bag is clamped. The catheter is connected to a pressure transducer. The symphysis pubis is used as the zero level.

reference (Figure 7.2). The technique shows a high degree of correlation with direct measurements of intraabdominal pressure up to 70 mmHg.[43,68]

Intragastric Pressure

A nasogastric tube may be used to measure pressure in the stomach as an estimate of intraabdominal pressure. Hydrostatic pressure can be obtained after installing 50 to 100 mL of saline in the stomach.[18,69] A gastric balloon filled with air may also be used for pressure recordings.[70] Extracorporal transducers are leveled at the midaxillary line to avoid hydrostatic artifacts from the tubing. Human studies have shown an acceptable correlation of gastric pressure to pressure in the urinary bladder.[69,70]

Pressure in the Inferior Vena Cava

No human studies have validated its use. The method is associated with significant risks. However, it has been shown in an experimental study that pressure in the femoral vein catheter correlates well with intraabdominal pressure measured directly and with urinary bladder pressure.[71]

CLINICAL MANIFESTATIONS

Symptoms of increased intraabdominal pressure include respiratory, cardiovascular, and renal dysfunction (Figure 7.3). Trauma patients who require abdominal packing, patients with coagulopathy, and those with profound hypothermia are at high risk. In Stage I, the abdomen is swollen and the circumference is increased as a sign of increased volume. The distended abdomen becomes tense. Venous stasis is seen in the lower extremities. The symptoms of Stage I may start at an intraabdominal pressure of 10 to 20 mmHg.[16,59] In Stage II, the abnormally elevated intraabdominal

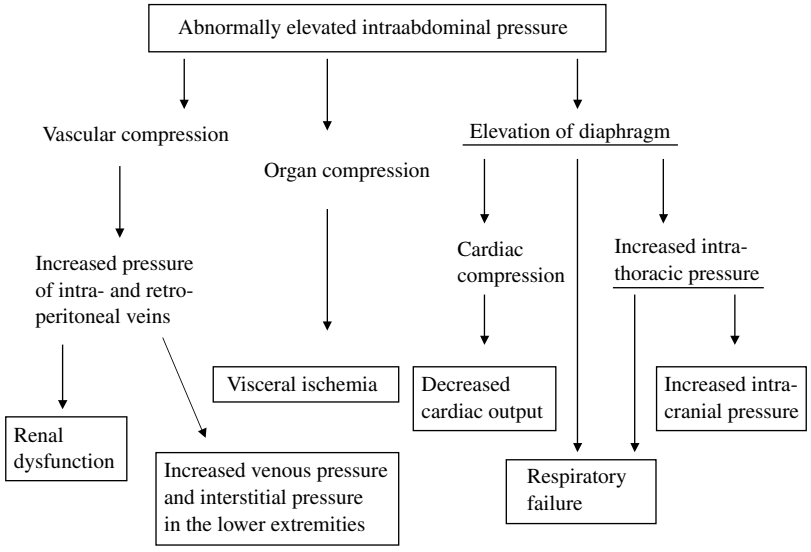

FIGURE 7.3 Algorithm illustrating how abnormally elevated intraabdominal pressure induces renal dysfunction, respiratory failure, decreased cardiac output, and visceral ischemia.

pressure affects the microcirculation of the intra- and retroperitoneal organs. Progressive oliguria, in spite of adequate cardiac output, and hypoxia with respiratory dysfunction evolve in the patient. In Stage III, symptoms of intra- and extraabdominal organs occur. These include respiratory, cardiovascular, renal, and visceral dysfunction. Most studies of the relationship between elevated intraabdominal pressure and organ dysfunction have been performed on hemodynamically stable patients or in animal experiments. They indicate that intraabdominal pressure exceeding 20 to 25 mmHg in trauma patients may need surgical treatment.

Respiratory Symptoms

Patients have difficulty breathing. They become tachypnoic. Most of them are critically ill and require artificial respiration. Increased intraabdominal pressure is associated with elevated diaphragm[7,41] and increased pulmonary airway pressure, with decreased arterial oxygen tension and hypercarbia. Pulmonary compliance is low and patients require positive end expiratory pressure to maintain oxygenation.[18] Chest x-ray shows small lung fields and elevated diaphragms.

Cardiovascular Symptoms

Patients have metabolic acidosis, tachycardia, low stroke volume, and elevated central venous pressure. The cardiac output decreases due to increased peripheral resistance and due to decreased venous return (Figure 7.1). Pulmonary wedge pressure is increased. Large volumes may be contained in the lower limbs due to venous stasis created by pressure on the inferior vena cava. Intraabdominal pressure of 14 mmHg increases the femoral venous pressure from 10 to 18 mmHg.[54] This level

of pressure reduces or even eliminates pulsatility in the common femoral vein, indicating proximal venous stasis.

Renal and Visceral Symptoms

Oliguria and anuria occur when intraabdominal pressure exceeds 25 to 30 mmHg. Prolonged low-grade visceral ischemia is associated with bacterial translocation, sepsis, and multiorgan failure.

TREATMENT

Respiratory, cardiovascular, and renal dysfunctions are difficult to manage unless the intraabdominal pressure is reduced.[7,20] Surgical treatment of the abdomen by celiotomi is the best way to normalize elevated intraabdominal pressure.[19] Prophylactic mesh fascial closure at the initial laparotomy may reduce the incidence of acute abdominal compartment syndrome, adult respiratory distress syndrome, and renal failure. Abdominal decompression produces polyuria and improved creatinin clearance.

Nonsurgical Treatment

Central venous pressure as well as pulmonary artery occlusion pressure (or pulmonary wedge pressure) are normal or elevated. Therefore, it is easy to assume that the patient has an adequate volume of the intravascular space. However, cardiac filling pressure to determine intravascular volume status is not reliable in these patients.[72] Patients with abnormally elevated intraabdominal pressure should be treated by adding volume when their cardiac output is low.

Arterial blood gas measurements demonstrate hyperemia, hypercardia, and acidosis, which all reflect physiological derangement. Mechanical ventilation with a positive end expiratory pressure inhibits metabolism of lactate.[41,53] However, it may cause further physiological abnormality if the increased intraabdominal pressure remains elevated. Intravascular volume should be restored and oxygen delivery maximized.[47] Hypothermia and coagulopathy must be corrected.

Surgical Treatment

Surgical and nonsurgical treatment for acute abdominal compartment syndrome must be executed simultaneously. It has been suggested that an urinary bladder pressure exceeding 25 mmHg associated with unexplained oliguria is an indicator for decompression.[17,19] Surgical treatment is conducted by abdominal decompression. After decompression, the abdomen is left open. Decompression of the abdomen is associated with a substantial improvement in survival rates. Escharotomy or tangential excision of abdominal burns may be helpful in burn patients with abdominal scars.[39]

Results

Surgical decompression of the peritoneal cavity reduces intraabdominal pressure and reverses this derangement.[19,73] The central venous pressure, urinary output,

ventilatory pressure, carbon dioxide tension, and oxygenation are normalized. The decompression improves acute respiratory failure.[7,18,43] Abnormally elevated peak airway pressures (>80 cm H_2O) may improve to normal levels.[16] The elevated pulmonary airway pressure and P_aCO_2 normalize soon after abdominal decompression. However, pulmonary oxygenation may take 18 to 36 h to return to normal.[7] Renal function returns to normal following decompression of patients with increased intraabdominal pressure due to postoperative hemorrhage.[28]

Adverse effects of surgical decompression have been described. These include severe hypotension immediately after the abdominal cavity is opened.[3,5,25] This may be explained by a sudden decrease of systemic vascular resistance or by a reperfusion syndrome.[3] Therefore, volume loading before abdominal decompression is advocated.[5,74] Anaerobic metabolites reach circulation by the reperfusion. Cardiac asystole following abdominal decompression has been described in one fourth of the patients with acute abdominal compartment syndrome.[3] Asystole has been reported in 12 to 25% of the patients following abdominal decompression.[5,74]

Wound Closure

Primary closure of abdominal wall may not be possible in patients with massive visceral edema and large intra- and retroperitoneal hematoma. When the abdomen cannot be closed without acceptable tension, it must be left open. The skin may be closed by sutures or towel clips to protect the bulging viscera. It may be covered with sterile plastic drape.[1] Secondary wound closure may be performed 7 to 10 days following the primary insult when edema is reduced and renal function normalized.[5] A silicon rubber sheet can be used to close abdominal wall in patients where primary abdominal closure was impossible or where it resulted in compromise in respiratory or renal function.[57] Synthetic mesh grafts[26] and plastic or rubber sheets have been used to close and reconstruct abdominal wall.[3,75]

CHRONIC ABDOMINAL COMPARTMENT SYNDROME

In patients with gradual increase of intraabdominal pressure, various organ systems are able to compensate for the increased intraabdominal pressure. The acute symptoms from multiple organs do not occur in these patients. The chronically elevated intraabdominal pressure may give rise to recurrent symptoms in these patients, which could be labeled chronic abdominal compartment syndrome.

Chronic elevation of intraabdominal pressure, as seen in obese patients, may produce obesity hypoventilation syndrome. Chronic elevation of intraabdominal pressure has been suggested to cause benign intracranial hypertension in excessively obese patients.[76,77] Obese patients may have elevated intraabdominal pressure, which increases pleural pressure, cardiac filling pressure, and impedes venous return from the brain, leading to intracranial pressure associated with pseudotumor cerebri.[77]

SUMMARY

Acute abdominal compartment syndrome is caused by abnormally elevated intraabdominal pressure. Elevated pressure induces dysfunction of intraabdominal and extraabdominal organs. The three pathogenetical mechanisms that cause the syndrome are increased volume of the intraperitoneal contents, external compression, and decreased size of the intraperitoneal cavity. Intraabdominal pressures of 10 to 25 mmHg decrease cardiac output and venous return from the lower extremities, and induce respiratory, renal, and visceral dysfunction. The intrathoracic and intracranial pressures increase. Intraabdominal pressure may be directly determined by an intraperitoneal catheter or indirectly by urinary bladder pressure measurements. Abnormally elevated intraabdominal pressure, estimated as urinary bladder pressure exceeding 25 mmHg, associated with unexplained oliguria is an indicator for decompression. The syndrome is fatal if left untreated. Surgical decompression by celiotomi reverses the symptoms and normalizes the circulation and the function of abdominal organs.

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8 Ischemic Contractures

INTRODUCTION

Untreated acute compartment syndrome evolves to ischemic contracture of affected tissues. After 4 h of total ischemia, 5% of muscle fibers become necrotic. After 8 h of ischemia, all muscle fibers are damaged. The muscle becomes fibrotic. This leads to loss of joint motion and imbalance of forces acting over the joint. Peripheral nerves may become compressed secondarily by the muscle fibrosis, which may induce further neuromuscular dysfunction. The limb dysfunction induces disability (decreased activity) and handicap (decreased participation). The aims of the clinical investigation of these patients are to document which muscles and nerves are affected and to what extent the tissues are damaged. The aim of this chapter is to discuss general principles of diagnosis, classification, and treatment of ischemic muscle contractures. Congenital and nonischemic reasons for contractures such as neuromuscular and inflammatory diseases are not included.

Definitions

Volkmann's contracture is a limb deformity that represents one of the final stages of muscle and nerve fibrosis following an untreated acute compartment syndrome. Muscular contracture may also occur as a secondary response to primary muscle ischemia or from a direct irreversible ischemic nerve injury.

In papers prior to 1972, acute compartment syndrome was classified as Volkmann's contracture in the *Index Medicus*. Acute compartment syndrome was classified as early contracture and the established ischemic contracture as late contracture.[1] Many cases previously termed as early contracture are today diagnosed as acute compartment syndrome.

Background

Von Volkmann first described the clinical entity in 1881.[2] He proposed that the syndrome was caused by tight dressings (external compression) of traumatized limbs. The contracture was considered to be due to ischemia caused by prolonged blocking of the arterial supply. Von Volkmann also thought that the simultaneous venous stasis accelerated progress of the paralysis. The contracture was described in terms of rigor mortis. Today, the contracture is regarded as the result of ischemic injury to selective tissues. Therefore, the condition will be labeled as ischemic contracture in this chapter. The ischemic contracture of Volkmann may be regarded as the local tissue complications following an untreated acute compartment syndrome.

Epidemiology

In a study on tibial shaft fractures, 1% of the patients with minor severity of injury and 22% of patients with major injury had signs of contracture.[3] In that study, patients with severe limitations of ankle and foot movement comprised one third of the patients with disability. Forearm fractures account for 15%[4] to 20%[5] of contractures.

In the review of Volkmann's ischemic contracture seen at the Mayo Clinic during 1935 to 1954, the time elapsed from injury until the patient was seen at the clinic varied from 4 h to 27 years.[1,6] Prior to 1935 the incidence of ischemic contracture was 0.18 per thousand new registrants. From 1935 to 1954, the incidence decreased from 0.08 to 0.03 per thousand new registrants.[5] Later, soft tissue injuries and crush injuries without associated fractures represented a larger proportion of cases. Ischemic contracture may be the reason for persistent stiffness of the ankle and foot in 1 to 10% of these patients.

ETIOLOGY AND PATHOGENESIS

Every acute compartment syndrome that is left untreated evolves into an ischemic contracture. Direct local injury, proximal vascular injury, arterial occlusion, or any combination of a no-flow situation (Figure 8.1) may initialize ischemic contracture. A direct injury leading to impairment of the arterial supply of a specific muscle may also induce a contracture state. Venous obstruction is an important pathogenic factor.[7] In experiments where only the veins were tied, pathologic changes were found in every instance.[8] Within an hour after vein

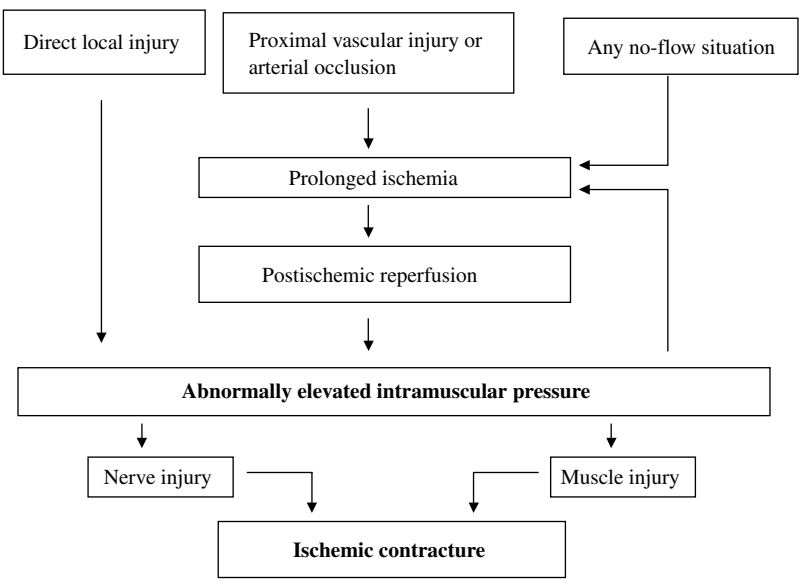

FIGURE 8.1 Pathogenesis of ischemic contractures following trauma is abnormally elevated intramuscular pressure. Muscle contracture may be induced by injury to muscle and nerve.

obstruction, the muscle became swollen, hard, and dark blue. Contracture followed always after these experiments. Experimentally induced contracture after only arterial ligature appeared later than that produced by acute venous obstruction.[8] Other pathogenetical mechanisms suggested for ischemic contracture include the theory of spasm of arteries and their collateral branches induced by the sympathetic nervous reflex system.

Muscle contracture may follow in patients with fractures treated without bandages. It may occur in patients with tibial fractures without pain. The risk for contracture increases if patients are not treated within 12 to 24 h of ischemic muscular pain. Muscle contracture may result from direct fibrosis of the small muscles of the hand following burn injuries.[9]

Direct injury of the tissues in a compartment may induce ischemia due to an abnormally elevated intramuscular pressure. Arterial injury or arterial occlusion may give ischemia distal to the lesion. In these cases, tissue pressure is most often normal initially in the affected part of the limb. However, following restoration of the arterial occlusion, a reperfusion syndrome may occur, which may induce abnormally increased intramuscular pressure. Direct ischemic injury to the nerve tissue may also induce muscular contracture.

The pull on plantar flexor muscles by dorsiflexion of the ankle joint increases intramuscular pressure to levels that may induce ischemia in the posterior compartment of the leg.[10,11] Immobilization of the leg and foot in plaster cast, keeping the ankle joint in slight plantarflexion, diminishes the risks for increased intramuscular pressure in all compartments.[11,12]

PATHOPHYSIOLOGY

Ischemic insults to muscle tissue may recover completely and induce gangrene or contracture. Gangrene involves all tissues including the most distal. In contrast, contracture is due to ischemia of selective tissues such as muscles and nerves of the affected compartments. Tissues distal to the compartment are not affected initially. They are not ischemic.

Pathologic changes of muscle contracture include edema, disappearance of nuclei and sarcolemma, loss of muscle striation, and hyalin degeneration with atrophy.[13] Muscle necrosis may be partly reversible if the local circulation can be restored before fibrous replacement has occurred.[14] Five percent of the muscle cells become necrotic after 4 h of total ischemia. After 8 h of total ischemia, 100% of the cells are necrotic.[15] They may become dysfunctional due to the proximal nerve and muscle insult. Some areas of the necrotic muscle may regenerate whereas others heal by fibrosis, which gradually causes a contracture. Skeletal muscle fibers may regenerate from satellite cells of a dying fiber if the basal lamina of the fiber is intact. The mechanism for activation is autonomous to the fiber and can occur without nerve or blood supply.[16] The number of satellite cells decreases with age and the capacity to regenerate skeletal muscle also appears to decrease with age. When muscles become ischemic, they lose their elasticity by degeneration of the fascicles. In the residual state, the muscle becomes rigid and contracted.[13]

GENERAL PRINCIPLES OF DIAGNOSIS, CLASSIFICATION, AND TREATMENT

Symptoms

Patients with ischemic contracture may experience pain, paresthesia, hypoesthesia, and paresis. They also experience cold, cyanosis, swelling, and joint stiffness. The range of motion amplitude is decreased. Muscles become hypotrophic and the affected limb is weak.

Some patients may recall severe pain in their initial history of trauma. The pain may have been resistant to pain medication during that time period. Some patients may have experienced pain for several weeks or months after the acute untreated compartment syndrome.[17]

Clinical Findings

The purpose of the clinical investigation is to determine the extent of muscle and nerve damage. Findings at clinical investigation include loss of motor and sensory function, decreased range of motion, and deformities of fingers and toes. These findings form the basis for choice of treatment. Patients may have difficulties in reaching a neutral position of the joint. Subluxation of involved joints may occur. After the initial stages of the syndrome, the clinical picture can vary considerably. Patients express difficulties to grip with their hands or stand on their feet.

Muscle Tissue

The retraction of flexor muscles at the distal joints in the limbs is the most striking feature of ischemic contracture. The tenodesis effect implies that a contracted finger or toe is extended if the wrist or ankle joint is flexed (Figure 8.2). Conversely, if the wrist or ankle joint is extended, the position of the finger or toe becomes more flexed. The involvement of nerve damage contributes to the clinical variety of functional impairment. Muscular imbalance develops by long-standing muscle palsies. This may create articular stiffness and subluxation of the involved joints. The active and passive range of motion of joints is reduced.

Nerve Tissue

Nerve lesions represent another important feature of the clinical picture. This includes motor deficit of the leg and foot muscles. Electromyographic investigation is a valuable aid in difficult cases. Sensory deficits are seldom present without motor involvement. In their series, 60% of patients with motor deficit had normal sensitivity.[18] Muscular hypotrophy, dry cyanotic skin, and dystrophic nails are common findings in these patients.[18] The sudomotorfunction is impaired.

Classification

Classification of contractures is essential in determining the method and timing of treatment. Several classifications have been suggested. Contractures have been

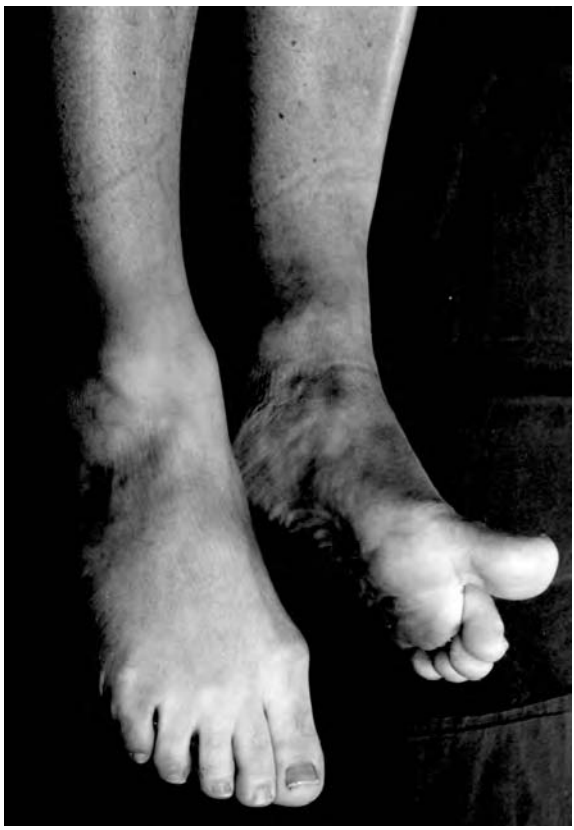

FIGURE 8.2 Example of ischemic contracture of the extensor muscles of the anterior compartment of the left leg in a patient with acute anterior compartment syndrome 1 year earlier. The extensor hallucis longus muscle is fibrotic. When the patient plantarflexes the foot, the metatarsophalangeal and interphalangeal joint of the left big toe becomes hyperextended (tenodesis). The ankle joint flexion is impeded.

classified into three grades[19] or four grades[1] based on severity. They have also been classified into three types,[20] four types,[21] or five stages[18] based on severity and tissue involvement. Classification must be based on a thorough clinical investigation and is most important before determining treatment. A three-grade general classification of ischemic contractures — mild, moderate, and severe contractures — has been used in this chapter whenever possible.

Mild Contracture

This is a localized type in one or a subtotal number of muscles in the compartment. Force generation of affected joints is normal or slightly decreased. There is normally no sensory dysfunction. As a sign of muscular retraction, a tenodesis effect is always present (Figure 8.2).

Moderate Contracture

Most muscles of the compartment are involved, both deep and superficial muscles. All fingers or toes are contracted. Sensory dysfunction is common. Articular stiffness due to secondary contractures of joint tissues is mild.

Severe Contracture

All muscles are necrotic and sensory dysfunction is severe. Secondary joint contractures are commonly seen, with contracted capsulae and ligaments. Articular stiffness due to secondary contractures of joint tissues is severe. The joint forces are not balanced around the neutral axis, which leads to subluxation and joint deformation. Dystrophic changes of other tissues such as skin and nails are seen.

Treatment

The overall goal of treatment is to regain active and passive range of motion around the neutral position of the joint. This includes the ability of patients to stand on plantigrade feet, to participate in common activities of daily living, and to use the pinch and grasp grips of their hands.

The principles for treatment of the upper and lower limbs are basically the same and may be divided into three phases. The first phase is treatment by physiotherapy and by supportive and corrective orthoses. This is achieved by serial casting or dynamic splinting, or both. Often, patients need night splinting after the contracture has been corrected. During this treatment, patients with sensory nerve dysfunction have increased risks of developing skin ulcerations due to pressure. Muscle strengthening is an important part of the exercise program. Treatment by growth hormone may increase growth of regenerating skeletal muscle after ischemic necrosis and diminish atrophy of skeletal muscle after denervation.[22]

In the second phase, deformities are corrected by surgery of soft tissues. More severe contractures that are not responsive to the methods previously described may need surgical treatment such as lengthening or transfers of tendons. Nerve compressions due to fibrotic muscles giving sensory or motor dysfunction should be treated by decompression as soon as possible. Expected results following decompression of nerves include increased sensitivity, increased muscle strength, and decreased pain.

The third phase involves corrective bone and joint surgery as well as soft tissue releases. The best time for surgical treatment is when the patient has had the peak recovery, usually 3 to 6 months from the initial trauma. Delaying surgery for more than 1 year is seldom necessary. In the following section, symptoms, clinical findings, and general principles of treatment of muscles and nerves of separate compartments of the upper and lower extremities is discussed.

CONTRACTURES OF LEG MUSCLES

The relative involvement of different leg compartments and the degree of muscular contracture in each compartment create a variety of different clinical pictures. There-

fore, clinical findings and treatment of contracture in each of the main four compartments in the leg are described separately. Muscular retraction may be demonstrated by measuring the total passive range of motion deficit. The total deficit is the clinical reflection of the muscular retraction in the leg. The severeness of the contracture can be estimated by range of motion scores of involved joints. Stiffness of the foot and ankle correlate with the severity of the leg injury and occur in about 20% of major injuries.[3] One third of patients with limitations of foot and ankle movement had clinical evidence of ischemic contracture in that study.

In most patients, contractions affect mixed compartments. Therefore, the treatment must be individualized. Many of the patients need complex surgical procedures including tendon transfers, tenotomies, and arthrolysis to gain improved function. Arthrodesis of selected joints may be necessary.[23] The goal of the treatment is to regain active and passive range of motion around the neutral position of the involved joints. This includes the ability of the patient to stand on a plantigrade foot. In severe cases, a derotating three-dimensional wedge osteotomy of the distal part of the tibia has been suggested.[24] Nerves entrapped by fibrotic tissue must be decompressed early in the rehabilitation period.

Contracture of the Anterior Compartment

Symptoms

Patients experience decreased ability to dorsiflex the ankle joint and toes. They have gait difficulties due to a drop foot. Plantarflexion of the ankle joint is diminished. The toes are painful and have decreased range of motion.

Clinical Findings

Patients may have sensory dysfunction over the first interdigital cleft. The extensor muscles may be weak. During maximal plantarflexion, the big toe extends as a sign of tenodesis (Figure 8.2). This is also true for passive flexion of the ankle joint.

Treatment

In mild cases, a splint may be sufficient. In more severe cases, the posterior tibial tendon may be transferred through the interosseous membrane to the dorsum of the foot. The tendon is transected at its insertion into the navicular bone. It is retracted proximally and brought anteriorly through the interosseus membrane. The distal aspect of the tendon is attached to the middle cuneiform bone through a separate incision.[25] An alternative method is to transfer the peroneus longus or brevis muscle to Os cuneiforme III[23] or cuneiforme II.[26] However, this alternative is often not available because the peroneal muscles are also affected in many of the patients with contracture of the anterior compartment.

Reconstruction with the peroneus longus tendon is an alternative for patients with no remaining muscles in the anterior compartment. The tendon is brought through the anterior intermuscular septum and transferred to the tendons of the tibialis anterior muscle and to the long extensors of the toes.[27]

Contracture of the Lateral Compartment

Symptoms

Patients experience local pain and sensory dysfunction of the superficial peroneal nerve. The contracture results in a supination of the ankle joint due to forces generated by the posteromedial structures of the leg.

Clinical Findings

Patients may have sensory dysfunction over the dorsum of the foot, except for the first interdigital cleft. The peroneal muscles are weak.

Treatment

Patients with mild symptoms are treated by a splint. Partial transfer of the anterior tibial tendon to the lateral part of the dorsum of the foot or to the peroneus brevis tendon insertion may restore balance of the ankle joint.

Contracture of the Posterior Compartments

Symptoms

Patients complain of painful rigid toes and pain in the forefoot. Patients may have flexion contractures of the toe joints, a cavus foot, and a fixed equinovarus deformity. The toe deformities, which include claw toes or mallet toes, are painful and may create skin problems. The ankle joint hurts.

Clinical Findings

Patients have a short cavus foot with clawing of the toes. They may have an equinovarus deformity of the forefoot (Figure 8.3 and Figure 8.4). The distance between the lateral malleolus and the Achilles tendon may be shorter.[24] This may be explained by dorsiflexion of the talocrural joint combined with adduction in the midtarsal joint. This angulation forces the patient to rotate the leg outward to parallel the feet during walking (Figure 8.3 and Figure 8.4).

Involvement of the distal part of the deep posterior compartment leads to claw toes,[26] due to contracture of the flexor hallucis longus and the flexor digitorum longus muscles. When all muscles of the superficial and deep posterior compartments are involved, the foot shows a fixed equinovarus deformity.

Contracture of the posterior tibialis muscle causes a medial and plantar pull on the navicular bone. These forces induce flexion contracture in the talocrural joint.[24] The active and passive range of motion is reduced. The subthalar joints may be completely rigid, and the navicular and cuboid bones may be subluxated. The talus and calcaneus bones are dorsiflexed in these patients. If no weight bearing is allowed, there is no counterforce to the pull of the tibialis posterior muscle. This effect will also be strengthened by contracture of the flexor hallucis longus and flexor digitorum muscles, which are responsible for the toe deformity.

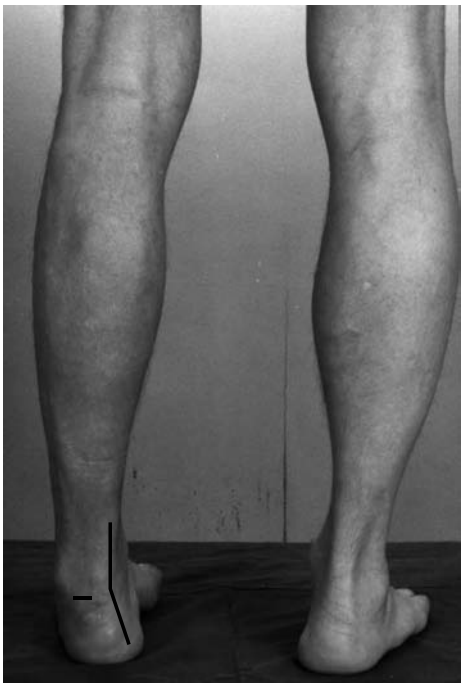

FIGURE 8.3 Ischemic contracture following untreated acute compartment syndrome of the left leg. The left side shows a short cavus foot with an equinu-varus deformity. The distance between the lateral malleolus and the Achilles tendon (black bar) is shorter. (See color insert following page 106.)

Treatment

The aims of the treatment are to restitute nerve function, release joint contractures, and replace lost muscle function to balance the forces over the ankle joint. Treatment must be individualized and may include neurolysis; tendon release; and lengthening, tendon transfer, arthrolysis, and arthrodesis.

Impaired passive dorsiflexion of the ankle joint may be increased by Z-platy of the Achilles tendon. This may be combined with capsulotomy of the ankle joint and lengthening of other flexor tendons. Claw toes and mallet toes may be treated by tenotomies of the flexor hallucis longus and flexor digitorum longus tendons. In addition to soft tissue releases, correction by a wedge osteotomy of the foot may be a necessary treatment in patients with fixed deformation of the foot. In severe cases, Karlström et al. suggested treatment by a derotation three-dimensional wedge osteotomy of the distal part of the tibia.[24]

CONTRACTURES OF FOOT MUSCLES

Untreated acute compartment syndromes of the foot or foot and leg compartments may induce complex posttraumatic deformities. This has been labeled the “short

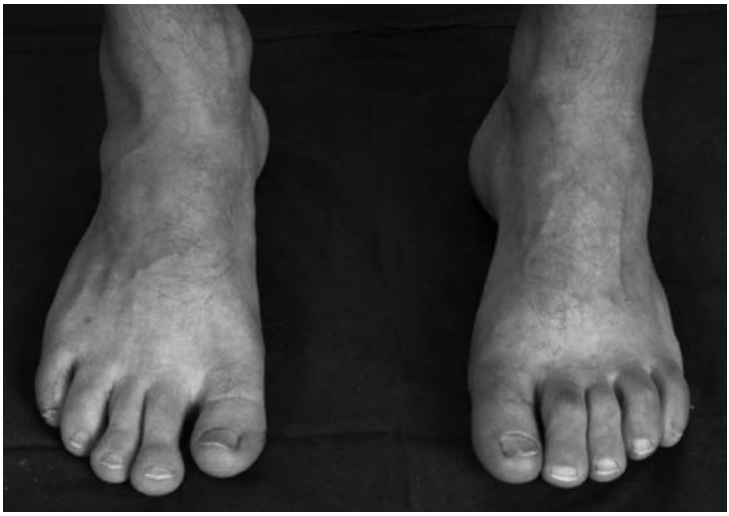

FIGURE 8.4 Mild claw toe deformities of dig. II to IV in a patient with ischemic contracture of the flexor digitorum longus muscle and intrinsic dysfunction. Soft tissues along the longitudinal arch are hypotrophic. The foot is shorter and has a cavus deformity. (See color insert following page 106.)

foot syndrome." It includes a contracted pes equinovarus foot with clawing of the toes. Contracture of the short toe flexors of the foot is seen after fractures of the calcaneus and crush injury of the foot. Assessment by sonography or MRI may demonstrate scarred, necrotic musculature in the involved compartments. The muscular imbalance over the affected joints forces them into subluxation or luxation. Treatment may require complex soft tissue release, muscle and tendon transfer, lengthening of tendons, and release of intrinsic muscles. The etiology of clawed toes was not known (or idiopathic) in 80% of patients in an operated series.[28]

Anatomy

Four to nine anatomical compartments of the foot have been described (Figure 8.5). Each of the compartments may develop fibrotic muscle tissue in response to untreated acute compartment syndrome or injury. The intrinsic muscles of the foot flex the metatarsophalangeal joints and extend the proximal and distal interphalangeal joints. Many factors such as extent of muscle necrosis in the foot and leg and nerve dysfunction determine whether the toes will be clawed or develop another deformity. Furthermore, the clinical picture of the foot deformity may be more complicated when contracture of leg muscles coexists.

Symptoms

Patients with ischemic contracture of foot muscles experience pain, paresthesia, hypoesthesia, and paresis. They also experience swelling and stiffness of toe joints. The range of motion amplitude of the foot and toe joints is decreased. Claw toe is

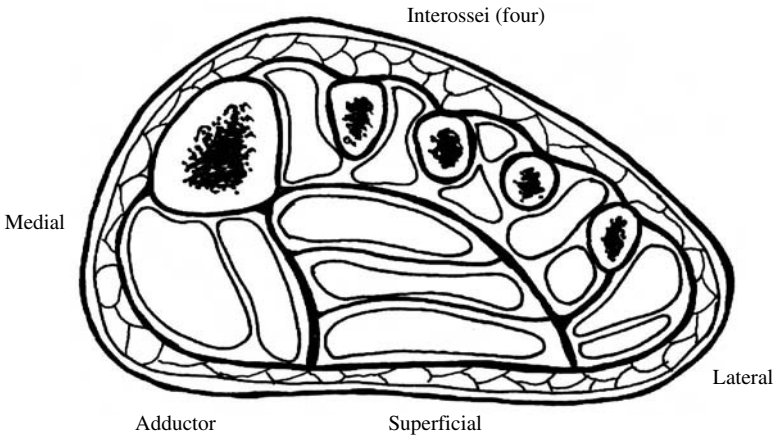

FIGURE 8.5 Compartments of the foot. Nine compartments of the foot have been described. The calcaneus compartment is located more dorsal, closer to the calcaneal bone, compared to this section of the foot.

a symptom and not an entity. Clawing of the toes implies that they are painful and can no longer fulfil their normal dynamic function.

Clinical Findings

Three types of toe deformities are defined. The terms *hammertoe* and *claw toe* have been used interchangeably in the literature. Claw toe has been used to describe the distal changes in patients with neuromuscular diseases. The claw toe deformity involves the metatarsophalangeal joint, which is hyperextended (Figure 8.6). Hammertoe is used to describe an abnormal flexion posture of the proximal interphalangeal joint in one or more of the lesser four toes. The deformity is fixed. It is not possible to passively correct it to the neutral position. Mallet toe is a flexion posture of the distal interphalangeal joint.

The clinical manifestation is clawing of the toes or formation of hammer toes. Hammer toes may be due to untreated compartment syndrome of the intrinsic muscles of the foot.[23] The loss of intrinsic muscle function leads to muscle imbalance over the toe joints. Patients with claw toes (intrinsic minus deformity) have extension of the metatarsophalangeal joint, flexion of the proximal interphalangeal joint, and extension of the distal interphalangeal joint (Figure 8.6). The toe deformity is similar following contracture of the flexor digitorum longus, but the distal interphalangeal joints are flexed and the other joints of the toes may even be in neutral position.[23] Curly toes differ from claw toes by having the metatarsophalangeal joint in a neutral position. A cavus foot deformity may be due to both intrinsic and extrinsic contracture.

Treatment

Treatment of the contracted foot includes complex soft tissue release, muscle and tendon transfer, tendon-lengthening procedures, and intrinsic releases to correct the

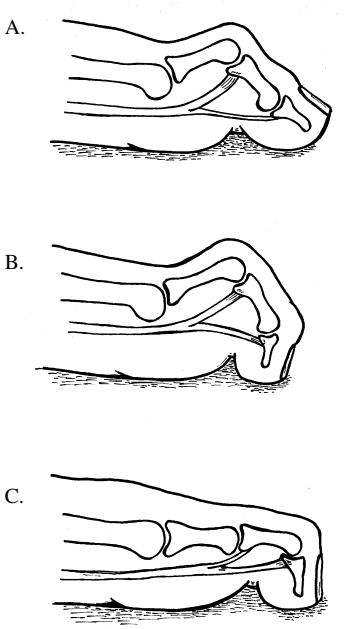

FIGURE 8.6 Three types of toe deformities may be found at clinical investigation. (A) The hammer toe deformity is due to contracture of the intrinsic (short flexor) muscles of the foot. (B) The claw toe deformity is due to a mixed contracture of the intrinsic and extrinsic flexors. (C) The mallet toe is due to isolated contraction of the extrinsic muscles of the foot (long flexors).

deformity.[23] Pes cavus may be treated by a wedge osteotomy. Toe deformities can be treated with conventional methods for foot reconstruction.

CONTRACTURE OF THIGH MUSCLES

Most patients described in the literature are young children who developed contractures following multiple intramuscular injections in the thigh.[29–31] Causes other than abnormally elevated intramuscular pressure may have induced the contracture in these cases. The contracture may appear in three forms: the vastus lateralis form, the rectus femoris form, and a combined form. The symptoms and clinical findings vary according to the degree to which the different muscles are involved.

Symptoms

Patients complain of limited flexion of the knee joint due to contracture of the quadriceps muscles. They experience pain on walking during an extended period of time, abnormal gait pattern, and inability to sit cross-legged and in the Japanese style.[32] Patients have difficulties in squatting. Contractures of selective thigh muscles may induce anterior knee pain due to patellar luxation or subluxation.[33]

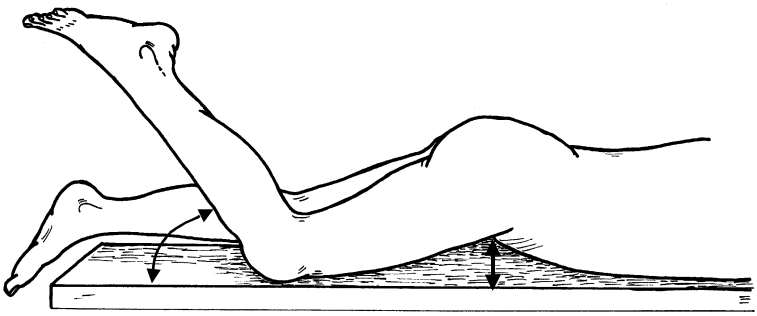

FIGURE 8.7 The Ely's test. During passive knee flexion in a prone patient, the pelvis begins to elevate from the table. This is due to a contracted rectus femoris muscle. The angle of knee flexion at which this occurs is expressed as the pelvic elevation angle.

Clinical Findings

Flexion of the knee joint is restricted if the vastus lateralis is affected. If the rectus femoris muscle is affected, knee flexion on full extension of the hip joint is restricted.[32] Patients have a positive Ely's test, which means that the pelvis will rise off the table or bed on knee flexion in the prone patient. The angle of knee flexion when the pelvis is elevated from the table is defined as the pelvis elevation angle (Figure 8.7).

The severity of rectus femoris contracture may be graded into four stages according to the knee total range of motion. Stage I includes patients with less than 31°, Stage II a range of motion between 31 and 60°, Stage III between 61 and 90°, and Stage IV more than 90°. The contracture is more severe when the angle is smaller.

By measuring the range of motion of the knee, first with the hip flexed and then again with the hip extended, the contribution of the rectus femoris muscle to the contracture can be demonstrated and evaluated.[34]

Treatment

Patients with a mild contracture may be treated by stretching during the early phase of the condition. In established moderate contractures, surgical treatment is indicated. In severe cases, passive stretching and physiotherapy are unlikely to produce lasting improvements.[30,35]

Surgical treatment for quadriceps contracture includes proximal muscle release, Z-plasty of the rectus femoris muscle, V–Y lengthening, and distal releases of the quadriceps muscles.

Proximal Release

Several alternatives to performing proximal releases are described.[35] It may be performed through a Smith–Pedersen anterior ileofemoral approach. Division of the fascia lata and proximal release of the quadriceps muscles along the tro-

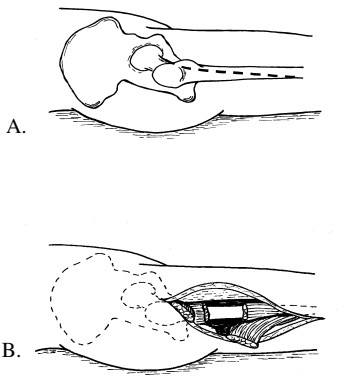

FIGURE 8.8 Drawing of the surgical procedure of proximal release of quadriceps muscles. (A) Skin incision. (B) The iliotibial band and tensor fasciae latae are cut. Vastus lateralis is detached from its origin at the greater trochanteric line and allowed to slide distally. The rectus femoris muscle may be released through the same incision. (Redrawn after Sengupta, S., *J Pediat. Orthop.,* 5, 187, 1985.)

chanteric line and lateral intermuscular septum are performed (Figure 8.8). With the subject in supine position, a longitudinal incision is made over the fibrotic part of the rectus femoris muscle.[36] With the affected leg hanging down from the operation table and creating tension of the muscles, a transverse incision is made in the fibrotic part of the rectus femoris muscle. Additional transverse incision of the fibrotic iliotibial muscle and other quadriceps muscles may be performed while the knee is further flexed. In severe cases, the procedure is combined with release of the sartorius muscle and the tensor fasciae lata.[37] Postoperatively, the hip is kept fully extended and the knee flexed 90° in a spica for 2 to 3 weeks.

After surgery, the knee is kept in maximal flexion for 3 or 4 weeks in a plaster cast. When the cast is removed, vigorous quadriceps exercises are important to improve the extensor lag. Knee-stretching exercises must be continued throughout the growing period.

Z-Lengthening

The importance of Z-lengthening of the rectus femoris muscle has been emphasized.[29] Satisfactory active and passive range of motion following Z-plasty was described in three cases. After surgery, patients were immobilized in a cast for 6 weeks.

V–Y Lengthening

V–Y lengthening of the quadriceps muscle tendon according to Thomsen procedure is an alternative method if all quadriceps muscles are affected. The method is not suitable in patients with selective contractures.

Distal Quadricepsplasty

This is a soft tissue operation in the distal one third of the thigh. An incision is made along the lateral border of the rectus tendon in the distal third of the thigh. The fascial sleeve is opened. Attachment of the fascia lata to the patella is divided.[33]

Results and Complications

Sasaki et al. saw nerve palsies in 15% of the patients.[32] The femoral lateral cutaneous nerve may be affected in patients treated by release of the pelvic origin and the saphenous nerve after release of the muscle belly.[32] Other complications include patellar luxation. Williams described patients who could not flex their knee joint more than 30° if the patella was forcefully held in midline.[33] Further knee flexion was possible only if the patella was allowed to dislocate.

Jackson and Hutton compared treatment by proximal release and distal quadricepsplasty.[34] Patients treated by proximal release showed better results. Knee flexion was 80° and no knees demonstrated an extension lag. Distal quadricepsplasty gave an extension lag in 70% of patients after an average follow-up of 10 years. It has been concluded that surgery should be avoided in patients who are younger than 5 years,[32,38] and it has been recommended in patients over 6 years[32] and 10 years of age.[38] Following these procedures, knee flexion may increase by 40 to 80°.

CONTRACTURE OF GLUTEAL MUSCLES

This is an unusual condition. Patients may experience symptoms of sciatica without low-back pain. They have atrophy of the buttock. In children, range of motion of the hip joints is severely decreased in flexion and abduction. Based on data from clinical investigation and neurography, it is possible to classify stages of severity of the contracture.

Tight structures may be surgically released. A normal range of motion was reported 1 year after surgery in children.[39]

CONTRACTURE OF HAND MUSCLES

Bunnell first described ischemic contracture in the hand in 1948.[40] It can involve the whole hand or isolated compartments of the hand. Selective paralysis may be difficult to detect early. Often, hand ischemia is a part of a more complex clinical picture including compartment syndrome of the forearm, stretch injury of the brachial plexus, arm fractures, and limb ischemia due to prolonged external compression in patients with drug-induced coma or crush injury. The intrinsic muscles of the hand participate in a complex finger joint motion with the extrinsic tendon system. They are most important to maintain a normal balance over the finger joints. Ischemic contracture of the muscles result in a significant loss of hand function. Superimposed nerve damage will add to hand dysfunction. Ischemic contractures of the hand may be classified as mild, moderate, or severe.[40]

Anatomy

The hand has six to ten compartments. There are four dorsal and three volar interosseus muscles of the hand. Each dorsal interosseus muscle has two heads, except the third, which has one head. The lumbrical muscles arise from a tendon and insert onto the deep flexor tendon.

Symptoms

Patients may complain of pain, weakness, numbness, stiffness, and anesthesia. They may experience difficulties in grasping things of certain sizes. The range of motion of the joints does not allow the hand to be opened sufficiently for grasping.

Clinical Findings

Muscle Tissue

In late cases with fibrosis and shortening of the interossei muscles, the metacarpophalangeal joints are flexed and the interphalangeal joints are extended. This condition has been labeled *intrinsic plus* position.[41] The hand assumes the same position which hyperactivity of the intrinsic muscles would induce. The end result is a disabling deformity with loss of the pinch and grasp grips.

The intrinsic test is useful for diagnosis of intrinsic contracture in the hand.[40,42] If the metacarpophalangeal (MCP) joint is passively held in extension, the interphalangeal joints lose flexion ability (Figure 8.9). Active and passive flexion ability of the interphalangeal joint increase if the MCP joint is held in flexion. The proximal interphalangeal (PIP) joint flexion will be less when the metacarpophalangeal joint is extended compared to when it is flexed. This is a positive intrinsic test (Figure 8.8). If there is an extension contracture of the PIP

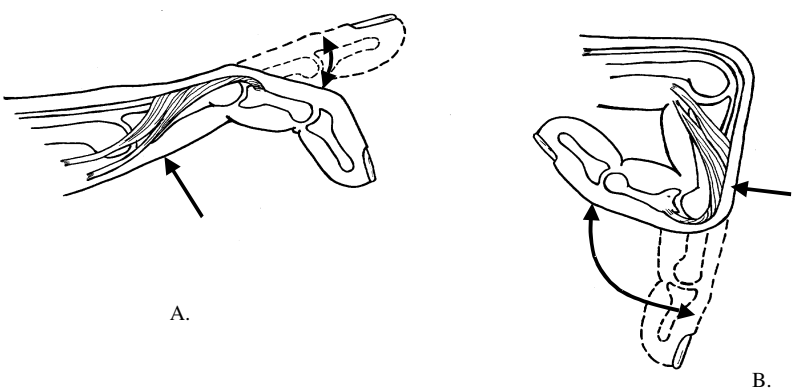

FIGURE 8.9 The intrinsic tightness test is positive if the interphalangeal joint has decreased range of motion when the MCP joint is held in extension (A) and increased range of motion when the MCP joint is flexed (B).

joint due to tight extensor tendons, PIP joint flexion will be greater when the MCP joint is flexed. However, capsular or intraarticular adhesions may limit the range of motion of the proximal interphalangeal joint and thereby an underlying intrinsic contracture.

Nerve Tissue

Sensation in the digits is usually normal. However, if the fibrosis involves the carpal and Guyon's tunnel, compression of the median and ulnar nerves may result in sensory loss as well as motor dysfunction.

Treatment

In mild cases of interosseus contracture, an elastic splint extending the MCP joints may be enough.[40] Several surgical methods have been described, including interosseous stripping, sectioning of the lateral bands, and release of the extensor component of the intrinsic muscles on the interphalangeal joint. The aims of the procedures are to restore the pinch and grasp functions, preserve finger extension and flexion, as well as abduction–adduction functions of the intrinsic muscles at the MCP joints.[42]

Interosseous Stripping

All intrinsic muscles are detached from the carpal bones with small periosteal sleeve.[40] The hand is immobilized in the claw hand position, that is, extended MCP joints and flexed IP joints until muscles are reattached in the more distal site. However, the functional results have been reported as inferior.[42] This operation is suitable for patients with moderate contractures. The prerequisite to perform this operation is that the intrinsic muscles are not too fibrotic.

Sectioning of Lateral Bands

In severe cases where the intrinsic muscles are too fibrous to function, Bunnell recommended tenotomies of the lateral bands.[40] The skin incision is made over the dorsal aspect of the three finger webs. It may be necessary to cut collateral or volar ligaments as well. The procedure is less traumatic. Recurrences of the deformity have been reported.[42]

Release of Extensor Component

Littler modified Bunnell's method of lateral band section by releasing the extensor component of the intrinsic muscles on the interphalangeal joint.[43] The flexor component was preserved on the MCP joint. The extensor hood at the MCP joint consists of three structures: the tendon to the long extensor muscles, the transverse fibers, and the oblique fibers of the intrinsic muscles. The oblique fibers extend the interphalangeal joints. By excising the oblique fibers, the extensor contracture of the proximal interphalangeal is released. The transverse fibers may still preserve their flexion action on the MCP joints, which will prevent hyperextension in MCP

joints. Compensation for intrinsic muscle loss is possible through sublimis tendon transfer for either adduction or opposition.[44]

Distal Intrinsic Release

Distal intrinsic release may be performed in patients with limited flexion of the PIP joints. A longitudinal incision is made along the dorsal midline of the distal half of the proximal phalanx. The lateral band and the oblique fibers are resected at the distal third of the proximal phalanx. The severe intrinsic contracture that involves the meta-carpophalangeal and interphalangeal joints is treated by resection of the lateral tendons of all interossei at the level of the MCP joints. A temporary arthrodesis by a K-wire through the metacarpophalangeal joint may be necessary after surgery.

After surgery the hand is splinted with the MCP joints at 180° and the IP joints activated for free range of motion from the first postoperative day.[42]

CONTRACTURE OF THE THUMB CLEFT

The thumb web space includes the soft tissues located between the metacarpals of the index finger and the thumb. In most cases, contracture of the thumb is associated with neuromuscular injury of the forearm. The thumb is often involved and has its own form of intrinsic contracture. Not only contracture of the muscles but also cicatrization of the skin over the web space and inappropriately applied plaster cast to immobilize the wrist may cause contracture.

Symptoms

Adduction and flexion contracture of the thumb is most disabling. Patients experience problems with gripping and difficulties in opposing the fingers because of the rotated thumb.

Clinical Findings

The deformity is characterized by a reduced cleft that separates the thumb and the fingers. The MCP joint is strongly flexed and the interphalangeal joint is hyperextended. The thumb may also be rotated externally and bound firmly to the second metacarpal. Thereby, the thumb loses its ability to oppose the fingers. The typical finding may be summarized as a “thumb in the palm” sign.

Treatment

Surgical treatment by excising the fascia and stripping of the contracted muscles from bones and tenotomies are described.[40] The first interosseous muscle may be detached from both metacarpals, and the adductors from the thumb may be released from the third metacarpal. A tendon transfer for opposition of the thumb may be necessary. In severe cases, excision of the involved muscles in the thumb web may be required. Tendon transfers and arthrodesis can be performed as indicated.[45]

CONTRACTURE OF FOREARM MUSCLES

Symptoms

Patients with ischemic contracture experience pain, paresthesia, hypoesthesia, and paresis. They also experience swelling and joint stiffness. The range of motion amplitude of the wrist joint is decreased, which gives a lack of grip capability.

Clinical Findings

In patients with ischemic contracture of the forearm, the wrist is flexed, the MCP joints are hyperextended, and the interphalangeal joints are strongly flexed.[42]

Muscle Tissue

Muscular retraction may be demonstrated by measuring the total passive range of motion deficit. The total deficit is the clinical reflection of the muscular retraction in the forearm and in the hand. The summation of values from each of the joints involved may range from 30 to 200°. Muscles become atrophic.

Nerve Tissue

Nerve lesions represent another important feature of the clinical picture. This includes motor deficit of the hand muscles, e.g., contracture of thenar muscles giving impaired oppositional function. Electromyographic investigation is a valuable aid in difficult cases. Sensory deficits are seldom present without motor involvement. Up to 60% of patients with motor deficit may have normal sensitivity.

Joints

Articular stiffness is difficult to assess initially. The thumb becomes stiff in adduction and the wrist in flexion. Permanent flexion of the wrist along with shortening of the volar ligaments and capsulae are difficult to estimate initially.

Miscellaneous

Dry cyanotic skin and dystrophic nails are common findings in patients with ischemic contracture.[18]

Treatment

The goal of the treatment is to regain active and passive range of motion around the neutral position of the joint. This includes ability of the patient to increase the hand grip function for common activities of daily living. One should wait at least 3 months for evidence of spontaneous recovery in the forearm muscles before surgical treatment is initiated.[46] Meanwhile, the patient should be treated by physiotherapy, dynamic orthoses to stimulate function, and static orthoses to adjust malalignment. Patients with contractures classified as mild or moderate may regain function by relatively simple procedures.

Mild Contractures

Treatment of mild type of contractures within less than a month after injury is a combination of physical therapy including functional training and dynamic orthosis. In limited injuries older than one month, dissection or excision of the affected area is sufficient.[20] More extensive mild contractures may need treatment by a muscle sliding operation. This is performed by releasing the flexor muscles from their origin. In mild cases, tenotomy may suffice.

Moderate Contracture

Patients with moderate contracture may need a muscle-sliding operation, tendon transfer, or a combination of the two. The muscle-sliding operation is simple and may be combined with a tendon transfer as a second procedure. One disadvantage with the muscular release is the decrease in grip strength, particularly in flexion of the distal interphalangeal joint.[20] The timing of tendon transfer after excision of necrotic muscle should be made on the merits of each patient, and depends on the extent of joint contracture and nerve dysfunction.

Severe Contracture

Patients with severe contractures may improve by different palliative surgical procedures, such as restoration of finger flexion, tenodesis, and arthrodesis. Eichler and Lipscomb[5] preferred an early muscle-sliding operation combined with neurolysis of the median and ulnar nerves as described by Page.[47] Early excision of necrotic muscles and neurolysis may restore sensation and function of the intrinsic and extensor muscles. Tendon transfer is performed as a secondary procedure after the injury to the skin and fractures are healed. Patients with severe contractures may be treated by pedicle graft, tendon transfer, tenodesis, arthrotomy, arthrodesis, and osteotomy.

Mixed Contractures

In patients with mixed contractures of the hand and forearm, the timing of different surgical procedures must be regarded.[21] Patients must have a good hand sensitivity, a good passive range of motion in the distal joints, and proximal muscles to stabilize the arm. It is important to restore an optimal muscle tension during surgery to avoid postoperative weakness. Muscular activation must provide full finger flexion and extension in any wrist position. The basis for this is to restore the resting sarcomere length during surgery. The maximum grip strength following this treatment is up to 50% compared with the contralateral side.[48]

FREE TISSUE TRANSFERS

Encouraging results of treatment by free muscle transfer from one site to another with complete separation from the donor site has been reported in patients with ischemic muscular contracture and motor loss in the arm.[49]

Muscle transplantation may be used in patients with significant functional deficit due to major loss of skeletal muscle. Patients' primary functional complaint is lack of grip capability. These patients have lost all flexor muscles but have some remaining extensor function. The median and ulnar nerves need to be intact. The hand must have good sensitivity and preserved intrinsic function.[48] Other procedures discussed previously, such as tendon transfers, must have been ruled out before transplantation is considered.

Free transfer of vascularized skin, nerve, and muscle tissue has shown promising results. Circulation can be restored to the transplanted muscle by microvascular anastomosis of the artery and veins of the muscle to suitable vessels in the recipient site. Transfer of the lateral head of the pectoralis major muscle to the forearm flexor has been suggested.[50,51] Free vascularized superficial radial nerve graft to replace the median nerve has been reported.[20,50] Reinnervation by suturing an undamaged motor nerve in the recipient site to the nerve of the transplanted muscle may result in muscular contraction activity after 2 to 4 months. Nerve repair and placement of the muscle at optimum tension are the most important operative steps.[48] Patients may obtain a maximum grip strength that is 50% of normal grip strength.

SUMMARY

Ischemic contracture may be due to an untreated acute compartment syndrome. Muscle fibrosis leads to loss of joint motion and imbalance of muscle forces acting over the joint. It may compress peripheral nerves. Measurements of strength and range of motion of the whole limb are the most important investigations. The contracture may be additionally assessed by MRI, sonography, and conventional radiography. Treatments of contracture include complex soft tissue release, transfer of muscles and tendons, lengthening of tendons, osteotomy, and arthrodesis. Entrapped nerves are decompressed early. The goal of treatment is to regain active and passive range of motion around the neutral position of the joint. This includes the ability of the patient to stand on plantigrade feet, to use the pinch and grasp grips of their hands, and to participate in activities of daily living. The aims of treatment include restitution of nerve function, release of joint contractures, and replacement of lost muscle function to balance the forces over the joint. The first phase is treatment by physiotherapy and supportive and corrective orthoses. In the second phase, deformities are corrected by surgery of soft tissues. The third phase includes corrective bone and joint surgery as well as soft tissue releases. The best time for surgery is when the patient has had peak recovery, usually between 3 and 6 months after the start of contracture.

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9 The Crush Syndrome

INTRODUCTION

The term *crush injury* includes and describes a variety of localized trauma states associated with injuries to bones and soft tissues. Crush injury is a traumatic injury severe enough to threaten tissue nutrition and viability. It involves transfer of large amounts of energy at the accident to the injured tissues.

The term *crush syndrome* describes the systemic manifestations induced by the influx of disintegrated products such as myoglobin, potassium, and phosphorus from muscle necrosis. Patients with major trauma may therefore suffer from two conditions: (1) the local crush injury to the soft tissues, including muscle tissue, and (2) the secondary systemic effects of destructed muscle tissue, which is labeled crush syndrome. This chapter focuses mainly on crush syndrome seen in patients with acute compartment syndrome.

Definitions

Crush syndrome is defined as the general manifestations of skeletal muscle injury. Systemic manifestations of the syndrome include hypovolemic shock, electrolyte derangement, and renal dysfunction. Electrolyte imbalance may even induce cardiac arrhythmia.

Rhabdomyolysis denotes injury to the skeletal muscle cells that liberate their intracellular contents into the extracellular fluid and the circulation. It may evolve to a crush syndrome.

Historical Review

The first reports on rhabdomyolysis appeared in the German literature in 1881.[1] Some consequences of crush injury were recognized in the beginning of the 20th century. They were documented in civilians buried during the 1909 Messina earthquake. Von Colmers in 1909 and Frankenthal in 1916 described muscle necrosis in soldiers buried under debris as a result of mine explosions.[2] Crush injuries were also described after mining accidents and mob stampedes.

In 1910, Myer-Betz described a syndrome of muscle pain, swelling, and weakness along with discoloration of the urine.[3] Later, it was shown that the discoloration was caused by myoglobinuria. In 1917, Hackardt first described anatomic changes of the kidney in patients with muscle necrosis.[4] Bywaters and Beall first used the term *crush syndrome* in 1941.[5] Bywaters estimated that the crushing injury accounted for about 5% of all casualties during the night raids on urban areas during World War II.[2]

MYOGLOBIN

Myoglobinuria means that myoglobin is present in the urine. Myoglobin is the red pigment of skeletal muscle and acts as a store of oxygen. The molecule has a weight of 40,000 Da. It is filtrated in the proximal tubule but cannot be reabsorbed in the distal tubule, which leads to renal insufficiency due to tubular obstruction. The molecule is a ferrous protoporphyrin globin complex containing one iron atom per molecule compared to the four found in hemoglobin. Myoglobin normally comprises 1 to 2% wet weight of skeletal muscle. In highly trained athletes, it may approach twice as much.

RHABDOMYOLYSIS

Rhabdomyolysis is commonly seen in a variety of clinical conditions such as primary skeletal muscle disease and systemic diseases that affect skeletal muscle[6] and following excessive exertion.[7,8] Many cases of rhabdomyolysis, but not all, evolve to a crush syndrome. Major rhabdomyolysis may be a serious and potentially fatal condition. It is mainly seen in young men. Exertional rhabdomyolysis has been reported in physically active individuals. Untrained individuals have larger increase in postexercise creatine phosphokinase in serum than do trained individuals.[9]

Patients have muscle soreness, stiffness, and local swelling. Clinical findings include muscular tenderness and limited range of motion. In laboratory studies, the concentrations of creatin phosphokinase in serum and myoglobin in urine are elevated. These patients need immediate fluid resuscitation to restore a normal blood volume and urinary output. Abnormalities of electrolyte concentrations must be corrected. Diuretics, mannitol, or bicarbonates diuresis may be necessary in severe cases. Thus, many cases evolve to a crush syndrome.

ETIOLOGY

Muscle injury in patients with crush syndrome may be induced by mechanical forces, acute traumatic peripheral ischemia, or a combination of both. Pelvic and limb fractures are commonly associated injuries of the crush syndrome. The syndrome may be induced by reperfusion following external compression,[10,11] overuse of skeletal muscle, heat, alcoholism, viral infections, metabolic disorders, myopathies, drugs,[12–14] toxins, and hypokalemia.[15] It may occur in patients in knee-chest position for lumbar spine surgery,[16,17] and following urologic surgery in the lithotomy position.[11,18] Viral rhabdomyolysis is an acute condition that presents as a viral syndrome with severe myalgia.[6,19] Exertional rhabdomyolysis was diagnosed in patients who presented with muscle soreness, weakness, and swelling following strenuous physical activity.[7,8,20–22] It was caused by repetitive exercises in about one half of all patients with rhabdomyolysis.[8]

It is important to appreciate whether the muscle is mechanically damaged or ischemia is the primary cause. This may have a major influence on the choice of treatment. Most trauma patients with the crush syndrome have some form of massive external compression for several hours or longer. Knezevich and Torch described a

patient with acute compartment syndrome who developed renal failure and a crush syndrome in association with streptococcal toxic shock-like syndrome.[23]

Even if the acute compartment syndrome is treated adequately by decompression, some patients may develop rhabdomyolysis with the crush syndrome and its systemic manifestations of hypovolemia, acute renal failure, and circulatory insufficiency. Crush syndrome may occur in top athletes with acute compartment syndrome of large muscle groups such as thigh muscles.

PATHOGENESIS

The crush syndrome is caused by leakage of myocyte contents into the circulation. Disintegration of muscle tissue and the influx of myoglobin, potassium, and phosphorus into the circulation cause the manifestations. Disintegration of muscle fibers releases osmotically active particles that promote a fluid shift from the intravascular space to the interstitial space of a compartment. Each millimole of osmotically effective particles per kilogram of tissue water exerts a pressure of 19.5 mmHg. This process may create further ischemia and muscle necrosis. Necrotic muscle from crush injury has 25% of its pigment and phosphorus, ca. 30% of its potassium and creatine, and only 5% of its acid-producing substances (e.g., glycogen).[2]

PATHOPHYSIOLOGY

The pathophysiology of the crush syndrome may be based on a model of ischemia-reperfusion injury. It may also be due to mechanical forces or external compression, or both.[24] This implies that muscle ischemia induced by arterial and venous obstruction, alterations of capillary permeability, and cellular and interstitial edema may cause the syndrome.

Reperfusion following prolonged external compression of a limb may lead to edema formation and abnormally elevated intramuscular pressure, which may induce secondary ischemia. It has been suggested that most of the damage to the skeletal muscle fibers occurs during reperfusion rather than during ischemia.[25–27]

The interstitial space is about three times larger than the intravascular space. Therefore, hypovolemia may develop fast if intravenous substitution is insufficiently supplied. Massive transudation and extravasation of intravascular fluid initiate hypovolemia, which may lead to hypovolemic shock. Vasoconstriction is unable to compensate for the rapid leakage of plasma, and blood pressure falls.[28]

Acute renal failure following traumatic injury to skeletal muscle is associated with hypovolemic shock, impaired renal blood flow, decreased glomerular filtration rate, and intratubular obstruction by myoglobin precipitation and vasoactive quinines from injured skeletal muscle cells that may directly damage the kidney.[29] Therefore, acute renal failure as a result of rhabdomyolysis is more likely to occur in trained individuals with a large muscle mass, because at trauma the circulation is exposed to larger quantities of myoglobin and other intracellular components.

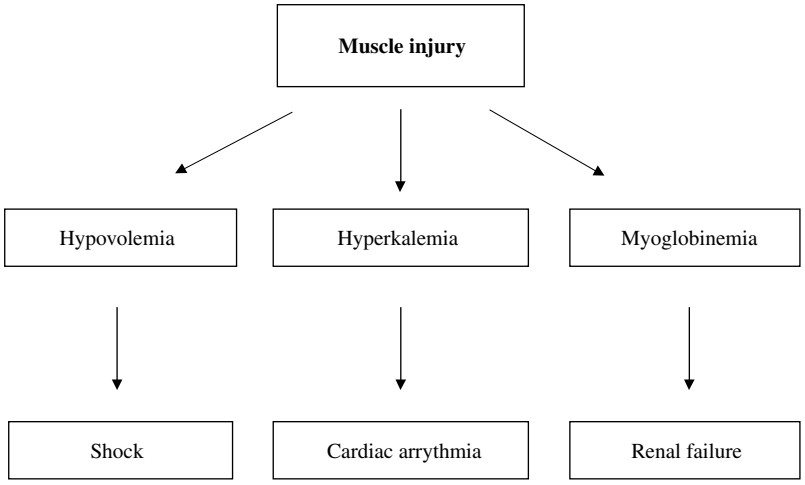

FIGURE 9.1 Clinical symptoms induced by muscle injury, which lead to the crush syndrome.

CLINICAL FEATURES

Symptoms and Signs

The clinical symptoms include hypovolemic shock, hyperkalemia, and acute renal dysfunction (Figure 9.1). Patients have dark urine. Even cardiac arrest may occur after crush injury.[30]

Skin

Well-localized skin erythema on extremities and buttocks may occur after prolonged local external compression. Symptoms may start 30 min after release of the external compression. The injured limb is swollen due to edema, which makes the skin tense and shiny.

Muscle

The muscles are affected by sudden swelling of the involved limb. Extravasation of intravascular fluid into the interstitial space may initiate hypovolemia, which may lead to hypovolemic shock. In nontraumatic cases, patients with rhabdomyolysis experience weakness, myalgia, and tenderness in the affected muscles. Limb movement may cause severe pain. It may even cause respiratory dysfunction if the diaphragm is involved. Similar symptoms may follow exertional rhabdomyolysis.[31] Muscular rigor is seen in patients whose limbs are compressed for 10 to 27 h, but not in any patients compressed for 8 h or less.[32] Once muscular rigor is evident, a large fraction of the affected muscle is necrotic. Palpation of a local induration is diagnostic.

Nerve

Patients generally have lost sensitivity and motor function distal to the injury. Patients with gluteal compartment syndrome involved often experience sciatic nerve dysfunction.[11,33] In most cases, it is impossible to tell whether the muscle, peripheral nerves, or both are affected.

Laboratory Findings

Laboratory findings include hyperkalemia, acidosis, hypocalcemia, and hyperphosphatemia released by the damaged muscle fibers. The degrees of creatinine phosphokinase, potassium, and phosphorus levels in serum are good predictors for acute renal failure in rhabdomyolysis, as well as low serum levels of albumin and the presence of dehydration.[34] Athletic patients with exertional rhabdomyolysis may have hyperuricemia. Finally, rhabdomyolysis of large muscle groups and associated capillary endothelial cell dysfunction leak albumin into the interstitial space of the muscle tissue, leading to hypoalbuminemia. Administration of albumin may increase the associated interstitial edema.[29] Creatine phosphokinase is an enzyme released by muscles under ischemia or necrosis. Peak serum concentration of creatine kinase and the number of injured extremities are good estimators of the severity of the crush syndrome.[35] Creatinine phosphokinase levels seen in patients with crush syndrome are much higher than those expected from surgical treatment alone.

Classification

Based on the severity, the syndrome may be classified into three stages.[10] Patients in Stage I have elevated muscle enzymes in serum and myoglobinuria. Patients in Stage II additionally have oliguria and may be hypotensive. Patients in Stage III have the full clinical picture of crush syndrome, including shock, oliguria, electrolyte derangement, and even cardiac arrhythmia.

TREATMENT

Treatment is directed to substitute volume, correct deranged electrolyte balance, and prevent infection. Rapid institution of forced diuresis and alkalization of the urine to prevent renal dysfunction are of primary concern. Failure to recognize and to respond appropriately to the condition may have fatal consequences in up to 25% of the reports in a literature review[11] and in 12% of patients with rhabdomyolysis.[6]

Adequate cushioning of the patient in the proper position at surgery can prevent the patient from developing the crush syndrome following prolonged operation. The lithotomy position with elevated limbs should be interrupted as soon as possible, especially in elderly patients with impaired peripheral circulation. This position should not be used more than 2 h.[11,36]

Nonsurgical Treatment

If multiple large muscle groups are involved in an acute compartment syndrome, the risk for circulatory hypovolemia and even a hypovolemic shock increases. The

initial period of shock is treated by giving adequate volumes of fluid and plasma. It may be necessary to administer 10 to 12 L of normal saline in the first 12 to 24 h to stabilize the circulation and urine production.[29,37] Mannitol may be administered as a single 25-g dose intravenously. Renal failure is the single most important factor when evaluating the prognosis of the patient with a crush syndrome. Large amounts of myoglobin in the circulation may be filtrated in the kidneys and occlude the distal tubule, which leads to renal insufficiency. Therefore, metabolc acidosis should be corrected by adding sodium bicarbonate to alkalize the urine and increase solubility of myoglobin in the urine. Most authors recommend alkalization of the urine in patients with major rhabdomyolysis whereas some authors do not.[29]

Treatment of hypocalcemia per se is not required because it usually corrects itself spontaneously. Calcium salts may be administered as a means to treat hyperkalemia. Treatment of hyperkalemia is especially important because of its potential for cardiotoxicity and tendency to induce arrhythmia. Finally, intravenous administrations of glucose and insulin lower the potassium level temporarily.[28]

In earlier times,infection was a leading cause of death. Currently, the problem is better controlled by antibiotic therapy combined with incision and drainage of abscesses, adequate debridement of necrotic liquefied muscle tissue, and treatment by antibiotics. More than 50% of open fractures are contaminated following treatment by debridement.[38]

Surgical Treatment

Successful treatment of acute compartment syndrome by fasciotomy may induce myoglobinemia. Decompression by fasciotomy may be a prerequisite to diminish the systemic effects of further muscle necrosis. In most cases, fasciotomy is indicated. Surgical treatment includes debridement, repair of damaged vessels, stabilization of bones, and repair of soft tissues.

Treatment by fasciotomy is controversial if delayed for more than 24 h in patients with lower-extremity crush injury. These patients suffer increased morbidity and mortality compared with those treated nonoperatively.[32,39] The muscle may bleed but must not be viable. Reis et al. reported that fasciotomy had no influence on the survival of muscle, but converted a closed injury into an open one with all the negative consequences it implies.[32]

LIQUEFACTION AND CALCIFICATION

Late sequels of untreated leg compartment syndrome are cystic degeneration, liquefaction, and calcification of the soft tissues. Patients present limb deformity following myonecrosis of leg compartments. Cystic degeneration is an uncommon sequel of acute compartment syndrome. Each of the conditions are defined as a painless, enlarging mass in a muscle compartment following untreated acute compartment syndrome that presents many years after the trauma. The pathogenesis of the process is not clear. It has been described in the superficial posterior[40] and anterior[41] compartments of the leg.

Plain radiology may reveal a calcific shell. The differential diagnoses are abscess; malignant tumor; and infection due to parasite, fungi, or mycobacterium infection.

Recommended treatment is excision and primary closure[40] or repeated needle aspiration of the mass.[42] Packing the wound and delayed closure may lead to secondary infection, chronic sinus formation, and limb amputation.

SUMMARY

Crush syndrome is a life-threatening general complication to acute compartment syndrome and other conditions, which causes leakage of myocyte contents into the circulation. Large amounts of hydrogen and potassium ions may be released from the injured muscle cell and create hyperkalemia, which together with acidosis increase the risks for cardiac arrhythmia. Clinical symptoms include hypovolemic shock, acute renal dysfunction, and deranged electrolyte balance (hyperkalemia and abnormally elevated creatinin phosphokinase levels). This life-threatening condition of the patient is treated before, during, and after fasciotomy by adequate volume substitution and buffering of the acidosis. Massive extravasation of fluid may require large volumes of fluid substitution during the first 12 to 24 h of treatment to avoid hypovolemia and oliguria. The patient needs an indwelling urethral catheter, and the intravenous fluid administration must ensure a urinary output exceeding 100 mL/h in the adult. If Volkmann's ischemic contracture is regarded as the local manifestation of an untreated acute compartment syndrome, the crush syndrome may be regarded as its systemic manifestation.

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10 Etiology and Pathogenesis of Chronic Compartment Syndrome

INTRODUCTION

Exercise-related muscular pain is seen among athletes and occupational patients who are exposed to repetitive muscular activity. The painful conditions have been termed chronic injury, chronic strain, chronic myalgia, repetition strain injury, overuse injury, and cumulative trauma disorder.[1] Up to 15 to 20% of employees are at risk for developing these conditions. A variety of pathologies on muscle-tendon units have been suggested. Most patients with muscular diseases have exercise-related pain and weakness. Symptoms may include anything from benign delayed onset muscle soreness to exertional rhabdomyolysis.

Chronic leg pain is one of the most common complaints by athletes. Each of the tissues, including bone, periost, muscle, fascia, tendon, vessels, nerves, and skin, may elicit pain. These tissues are closely related, especially in the leg and forearm. It may be difficult to distinguish from which tissue the pain comes. Chronic anterior compartment syndrome as a cause for anterior leg pain was confirmed only in 10 to 20% of the patients, who were suspected on clinical ground to have the syndrome.[2] Possible pathogenetical mechanisms of chronic compartment syndrome may be classified as (1) increased volume of compartment contents, (2) decreased compliance of the compartment, and (3) external compression or any combination thereof.

Historical Background

The anterior tibial syndrome was first described independently by Severin and Vogt in 1943.[3,4] The anterior tibial syndrome was defined as the triad of (1) necrosis of the anterior tibial muscle, (2) paralysis of the extensor digitorum brevis muscle, and (3) anesthesia of the skin over the first interdigital cleft of the foot.[5,6] Presently, anterior tibial syndrome should rather be regarded as an untreated acute compartment syndrome of the anterior compartment of the leg. The terminology *march gangrene* was used synonymously with anterior tibial syndrome.[7] In a review of the literature, Bradley in 1973 reported that 33% of all anterior tibial syndromes were induced by exercise.[8] Sixteen percent of these patients had a long history of recurrent exercise-induced pain in the anterior compartment of the leg predating the episode.

Carter first suggested a reversible form of anterior tibial syndrome in 1949.[5] The chronic, recurrent, reversible form of anterior tibial syndrome was first

described by Mavor in 1956.[9] He described a 24-year-old professional football player with anterior pain in both legs. The onset coincided with more intensive training. The patient described the pain as tightness rather than a cramp, and it forced him to rest. He had no pain at rest after walking or moderate exercise. The presence of small fascial defects over the anterior compartment indicated the possibility of increased tension of the fascia due to excessive intramuscular pressure. Mavor treated one patient with chronic anterior compartment syndrome by incising the fascia over the anterior compartment of the leg. The incision, which included the fascial gaps, was repaired by a 5 × 16 cm graft of the fascia lata.[9] Others questioned the existence of this syndrome.[10,11] However, French and Price in 1962 and later Reneman in 1968 confirmed the existence of the syndrome by measurements of intramuscular pressure,[12,13] and muscle blood flow.[12] To summarize the historical perspectives, the concept of chronic compartment syndrome in the anterior compartment of the leg is considered to have originated from the reversible exercise-induced anterior tibial syndrome.

INCREASED VOLUME OF THE COMPARTMENT

Although the etiology of the syndrome is not very well known, the pathogenesis of the syndrome is fairly well known. Abnormal volume increase of compartment contents may be caused by exercise, trauma, muscular hypertrophy, venous hypertension, inflammatory changes of muscle tissue, muscle rupture due to repetitive overloading, or by any combination thereof. Symptoms of the syndrome may also follow blunt trauma and tumor expansion (Figure 10.1).

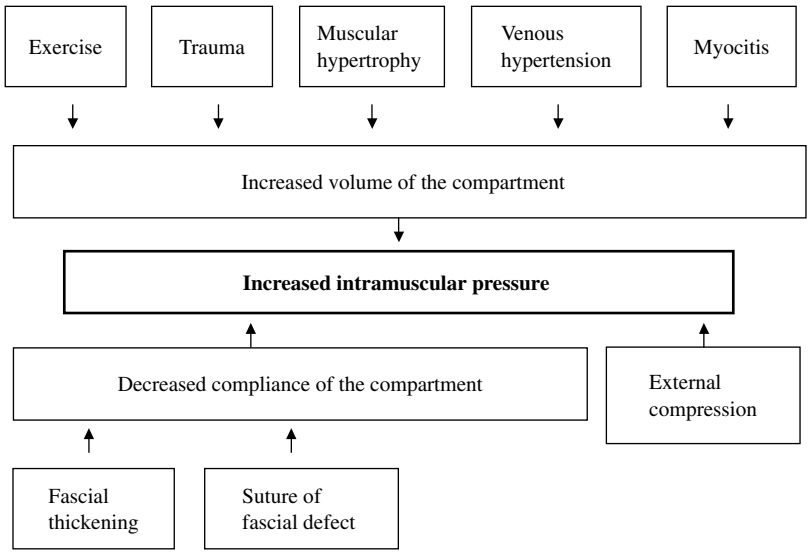

FIGURE 10.1 Possible reasons for abnormally increased intramuscular pressure in chronic compartment syndrome.

Exercise

The symptoms of patients with chronic compartment syndrome are induced by exercise. Muscle volume may increase up to 20% during maximal normal exercise. The mode of exercise may influence intramuscular pressure. Intramuscular pressure at rest is more elevated following eccentric muscular activity compared to concentric activity.[14] Seventy percent of patients with chronic anterior compartment syndrome are runners.[15]

Fluid is normally accumulated in exercising muscles.[16] The volume of the anterior compartment increases during exercise.[17] The fluid accumulation during normal exercise increases muscle relaxation pressure during exercise and intramuscular pressure in the anterior tibial muscle at rest after exercise to between 15 and 25 mmHg in normal subjects.[18–22] In a review of the literature, Bradley[8] found that 33% of all acute compartment syndromes were exercise induced. Many of the patients also had a previous history of exercise-induced leg pain for several years. In recent decades, this reason for the acute syndrome is less commonly reported, but the historical view of the problem shows that if the exercise is continued beyond pain limit in excess, it can develop into the irreversible acute syndrome.

Lawson et al. showed that pressure in the normal asymptomatic anterior compartment was not significantly different in the anterior compartment of cross-country skiers using the skating method compared to those using classic skis.[23] However, Gertsch et al. presented one patient who developed a chronic anterior compartment syndrome in the leg after adopting the new cross-country skiing technique.[24] This indicates that the etiology may be complex and probably multifactorial. Beckham et al. measured intramuscular pressure in the anterior compartment after exercise at 80% VO_2-max in symptom-free runners and cyclists. Average pressure in the anterior compartment during VO_2-max was 19 mmHg in runners and 12 mmHg in cyclists. The authors suggested that cycling as a pain-free form of exercise be tested in patients with chronic anterior compartment syndrome to determine its efficacy.[25] Maximal exercise produced a significant increase of intramuscular pressure in runners as compared to cyclists.[25] Endurance running can induce muscle fiber degeneration in humans.[26] However, increased intramuscular pressure was not responsible for fiber degeneration.[27] Hand exercise and exposure to hand vibration increases intramuscular pressure significantly more in the forearm compared to exercise without vibration in patients.

Trauma

Chronic compartment syndrome may follow major trauma to the lower leg[28] and to the forearm.[29] Ten percent of patients with chronic anterior compartment syndrome of the leg had their symptoms following major trauma.[30] Even low-velocity trauma, such as a direct blow injury during a softball game, may precipitate a chronic compartment syndrome.[31] In two out of five patients with chronic compartment syndrome of the lumbar paraspinal muscles, an L1 compression fracture preceded the symptoms of the syndrome.[32] The authors speculated that venous stasis or a postthrombotic syndrome, or both, could be contributing etiological factors.

Muscular Hypertrophy

Muscular hypertrophy as a cause of chronic compartment syndrome has been suggested, because the syndrome occurs in patients who have a high level of physical activity. The acute syndrome induced by exercise has been reported in one patient following androgen therapy.[33] Chronic compartment syndrome in the anterior compartment of the leg is not common among body builders, possibly because they do not exercise these muscles. Accessory soleus muscles increase intramuscular pressure in the deep posterior compartment but not to the level as that of chronic compartment syndrome.[34]

Venous Hypertension

Deep venous insufficiency may create a volume load of a muscle because the muscle pump is less effective. Repeated surgery for venous insufficiency has been reported as an unusual cause of chronic anterior compartment syndrome.[28] Plethysmographic examination showed subnormal values of venous emptying in a few patients with chronic leg compartment syndrome.[35] Patients with incompetent valves of the deep veins have increased muscle relaxation pressure during exercise but not to the level as that of chronic compartment syndrome.[28] Venous hypertension following thrombosis of the iliac vessels increases intramuscular pressures in both the anterior and deep posterior compartments.[36] Venous hypertension in the leg has also been suggested to follow a Baker cyst or substantial swelling around the knee joint.[15] Venous insufficiency as a cause of chronic compartment syndrome in the lower legs is probably not common. Severe venous insufficiency was documented in 3% of patients with chronic anterior compartment syndrome of the leg.[37]

The anterior tibial muscle is drained by the anterior tibial veins. The veins leave the anterior compartment through a fascial opening in the proximal part of the interosseous membrane. If this fascial opening is affected by trauma, it may constitute an anatomical predisposition for compression of the veins. It has been suggested that the vessels, mainly the veins, may be mechanically compressed between the herniating muscle tissue and the unyielding fascial edge of an enlarged opening of the interosseous membrane in chronic compartment syndrome legs (Figure 10.2). Obstruction of venous flow remains an interesting possible pathogenetic mechanism for chronic compartment syndrome in the leg.

Myositis

Microtrauma to muscle tissue and excessive stress to the capillary bed and to the interstitial space of muscle tissue may be explained by the strenuous exercise documented in most chronic compartment syndrome patients. Myositis and inflammatory states in the capillary bed may increase filtration coefficient three- to fivefold.[38] This means that increased fluid flow from the capillaries to the interstitial space may increase the volume and fluid pressure of the space.

Overload Injuries

Rupture of few muscle fibers and hemorrhage into the muscle during overexertion has been described.[5] Myofibril change and edema of the fast-twitch fibers follow

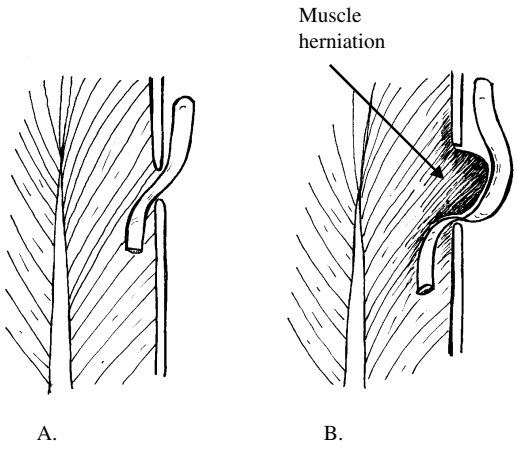

FIGURE 10.2 (A.) Normal. (B.) Muscle tissue herniation through a fascial defect. Adjacent vessels may be compressed or pinched between the fascial edge and muscle herniation.

eccentric muscular contractions.[14] Intramuscular pressure is elevated after eccentric muscular activity.[14] Other reasons for pain may make muscle relaxation more difficult. High pressure in the tibialis anterior muscle during eccentric muscular contraction is involved in a forceful heel strike.

DECREASED COMPLIANCE OF THE COMPARTMENT

The syndrome most often occurs in locations with relatively unyielding osteofascial spaces. Increased thickness of the fascia over the anterior compartment was reported in 25 out of 36 samples.[15] In another study, fascial thickness was not increased.[37] However, patients in that study had experienced pain for more than 2 years. Therefore, it was difficult to make any conclusions about the fascial thickness at the start of the syndrome. A mesh-like distension of the fascia covering the muscle is seen in many patients with chronic anterior compartment syndrome of the leg at surgery. Sometimes they have multiple small subclinical fascial hernias. Soffer et al. reported one patient with an aberrant fascial band overlying and tightly compressing the fascia over the anterior compartment of the leg.[39]

Suturing of fascial defects is never indicated. Decreased volume of the compartment following suture may increase intramuscular pressure and elicit an acute compartment syndrome.[40]

EXTERNAL COMPRESSION

External compression increases intramuscular pressure at rest and muscle relaxation pressure during exercise. Muscle relaxation pressure is the pressure in the muscle between contractions (Figure 10.3). External compression by tight stockings and braces may increase muscle relaxation pressure during exercise.[41,42] Volume of the compartment is not increased; rather, it may be decreased. The local blood perfusion

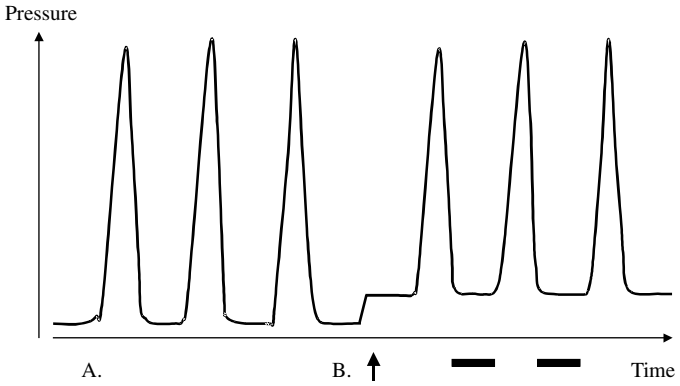

FIGURE 10.3 Redrawing from an original intramuscular pressure recording during concentric muscular activity. Pressure recording without (A) and with (B) external compression, e.g., a knee brace. During external compression of the leg (after arrow), the muscle relaxation pressure that occurs between contractions is elevated. The time period for muscle relaxation pressure is indicated by the bars.

pressure decreases by increased muscle relaxation pressure and intramuscular pressure at rest after exercise. Patients with elevated intramuscular pressure experience premature muscle fatigue. In another study, pressures were recorded in the leg and thigh without a knee brace and with a brace applied with strap tensile force of 25 N, 50 N, and a force preferred by the subject. External compression caused by the brace increased intramuscular pressure at rest and muscle relaxation pressure during exercise three- to tenfold compared to normal pressure.[43] The pressure increased to between 30 and 54 mmHg. Local blood perfusion pressure in the supine subject decreased up to 42%.[43] In other studies, these pressure levels have been shown to decrease local muscle blood flow and induce premature muscle fatigue.[44–47] Pressures increased relatively more in the braced leg compared to the braced thigh. This is, of course, not a chronic compartment syndrome, although symptoms and metabolic changes may be similar. External compression of a limb may induce relative ischemia. Following ischemia, endothelial cells may leak abnormal volumes of plasma or water into the interstitial space of the muscle and induce abnormally elevated intramuscular pressure. Eiken and Bjurstedt found that exercise performance was reduced by 40% due to muscle fatigue in subjects who were exposed to a supraatmospheric pressure of 50 mmHg.[48]

SUMMARY

Possible pathogenetic mechanisms include increased volume, decreased compliance, and external compression of the compartment. Abnormal volume increase of the compartment contents may be caused by exercise, trauma, muscular hypertrophy, venous hypertension, inflammatory changes of muscle tissue, and muscle rupture due to repetitive overloading. Suturing of fascial defects is never indicated. Decreased volume of the compartment may increase intramuscular

pressure and elicit an acute compartment syndrome. External compression caused by orthotic applications may increase intramuscular pressure at rest and muscle relaxation pressure during exercise to levels that elicit premature muscle fatigue caused by ischemia. The most common reason for chronic compartment syndrome in the leg is increased volume of the anterior compartment, which increases the muscle relaxation pressure during exercise and intramuscular pressure at rest after exercise.

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11 Pathophysiology of Chronic Compartment Syndrome

INTRODUCTION

The pathophysiology of chronic compartment syndrome describes how abnormally increased intramuscular pressure in a compartment affects muscle blood flow, tissue oxygenation, and neuromuscular function.[1–3] It also includes the study of how patients are affected by local muscular ischemia[4,5] and exercise-induced pain. Basic understanding of the pathophysiology of chronic compartment syndrome enhances the ability to diagnose this syndrome by clinical means as well as the understanding of other conditions of chronic leg pain.

Swelling of Muscle Tissue during Exercise

Muscle tissue normally swells during exercise. The swelling is most pronounced in the interstitial space. Swelling per unit area of the interstitial space is twofold higher than muscle fiber swelling per unit area.[6] In animal experiments, it has been shown that the lymph flow can probably drain the entire protein content of the capillary filtrate produced during exercise.[7] The increased volume of skeletal muscle during exercise is due to increased regional muscle blood flow and transcapillary filtration into the interstitial space. Data from human studies indicate that muscle blood flow during exercise is between 50 and 100 mL/min/100 g,[8] although values of up to 200 mL/min/100 g have been reported.[8,9] The mechanical influence of muscle contractions on flow rate is reasonable explanation for the higher muscle blood flows in conscious animals during exercise.[8] The amount of capillary filtrate accumulating in exercising muscle depends on the workload. The most important limiting factor for filtration into the interstitial space is the increased interstitial hydrostatic pressure.[10] Transcapillary fluid loss into leg muscles during heavy exercise during 6 min is 45 mL/kg.[11] After 15 min of work, the total amount of fluid accumulation averages 14.8 mL/100 g tissue.[12]

The compliance of the interstitial space in skeletal muscle is high at rest,[13] when intramuscular pressure is within the normal range. Substantial amounts of fluid can be mobilized from or added to the interstitial space of skeletal muscle with only small alterations of the tissue fluid pressure (Figure 15.9). Most authors believe that the symptoms and the dysfunction elicited by the syndrome are due to ischemia whereas others believe they are not related to ischemia.[14]

REGULATION OF LOCAL MUSCLE BLOOD FLOW

Theories on muscle blood flow at rest are discussed in Chapter 4. In the present chapter, these theories are applied for conditions with exercising muscles. The effects of increased muscle relaxation pressure during exercise, i.e., the pressure between contractions, on local perfusion pressure and muscle blood flow are discussed. Results from clinical studies are compared with theories on microvascular occlusion and critical closing pressure.

Perfusion Pressure (PP)

The arteriovenous gradient theory is based on the hydrodynamic principles described in the Law of Poiseuille:

$$Q = k(P_1 - P_2) \times r^4/L \times n \tag{11.1}$$

According to this law, fluid flow (Q) is directly proportional to the pressure difference ($P_1 - P_2$) over the tubing or vessel and to the fourth dimension of the radius (r). Fluid flow is indirectly proportional to the length of the vessel (L) and to the viscosity of the fluid (n). The constant $k = \pi/8$. In Equation 11.2, the resistance (R) is directly proportional to the length of the vessel and the fluid viscosity (n):

$$R = 8L \times n/\pi \times r^4 \tag{11.2}$$

Therefore, if Equation 11.1 is combined with Equation 11.2, muscle blood flow (MBF) by the arteriovenous gradient theory can be expressed as:

$$\text{MBF} = (Pa - Pv)/R \tag{11.3}$$

The perfusion pressure (PP) is the difference between the pressure at the arterial (Pa) and the venous (Pv) end of the capillary (Figure 11.1). Pa may be expressed as mean arterial pressure (MAP). Pressure inside patent veins (Pv) can never be less than the extramural pressure,[15] which is the total tissue pressure in the interstitial space. Therefore, muscle relaxation pressure (MRP) during exercise and intramuscular pressure at rest after exercise are equal to Pv in Equation 11.3. The local PP during muscle relaxation may therefore be expressed as:

$$\text{PP} = \text{MAP} - \text{MRP} \tag{11.4}$$

MRP in Equation 11.4 may be replaced with intramuscular pressure at rest after exercise. Increased intramuscular pressure reduces the local arteriovenous pressure gradient, and thereby reduces the local MBF.[3,16,17]

Microvascular Occlusion

Normal capillary pressure at rest is between 20 and 30 mmHg. The limiting factor in the MBF disturbances has been shown to be at the capillary level.[18,19] Increased

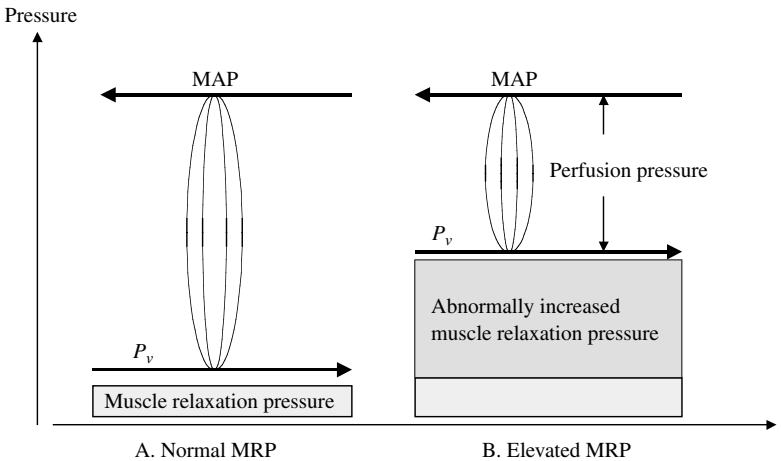

FIGURE 11.1 Perfusion pressure in a subject with normal muscle relaxation pressure (A) and a patient with chronic compartment syndrome (B). MAP is the mean arterial pressure and P_v is the intravenous pressure. Perfusion pressure is significantly lower in patients with chronic compartment syndrome due to the increasing muscle relaxation pressure during exercise.

intramuscular pressure causes capillary vessels to collapse at their distal end.[20] It has been shown that increased MRP values to between 35 and 55 mmHg in chronic compartment syndrome patients impair local MBF and elicit the clinical symptoms of the syndrome.[3] These findings may be explained by both the arteriovenous gradient theory and the theory of microvascular occlusion.

Critical Closing Pressure

The critical closing pressure theory postulates that a vascular bed may be exposed to zero blood flow in the presence of a positive PP. Perfusion pressure is defined as the difference between mean artery pressure and the intramuscular pressure. The pathogenetical mechanism for vascular collapse in the presence of a positive transmural pressure may alter with vasomotor tone. The transmural arteriolar pressure at which blood flow ceases is an index of critical closing pressure. The physiological event has also been described as flow cessation pressure.[21] Evidence for this concept was reported by Ashton,[21] who showed that the positive pressure intercept at zero flow was 33.4 (range 10 to 68) mmHg in the forearm of normal subjects at rest. This finding was explained as active closure of the small arteries in muscle tissue.[22] The work of Roddie and Shepherd also supported the concept of critical closing pressure, but they found no signs of arteriolar constriction in response to a rise in venous pressure.[23]

With this theory, it is possible to explain how patients with chronic compartment syndrome have decreased local MBF in the presence of positive PP. The critical opening transmural pressure was 5 to 10 mmHg higher than the closing pressure.[24] This implies that once the vasculature closed, it was more difficult to

open. This finding was also observed by Chang et al., who reported a significant difference of critical opening and critical closing pressure of the vasculature during external compression.[25]

Tidal Wave Theory

Dahn et al.[26] found that MBF did not cease until intramuscular pressure reached the systolic blood pressure when venous stasis was used. They also found that MBF stopped when the locally applied external pressure was equal to the diastolic blood pressure at rest. The explanation for this is given by the tidal wave theory, or Starling valve theory, which postulates that the microcirculation does not remain open long enough to allow for pulsatile muscle blood flow. They concluded that the diastolic blood pressure, rather than the MAP, determined the driving pressure over the vasculature.

The contraction frequency, level of muscle pressure during contractions, and the time period for muscle relaxation between contractions are important to control during exercise studies. If contraction frequency is high and time period for muscle relaxation short enough, the microcirculation may not stay open long enough to allow for pulsatile MBF. Rodbard and Pragay found by comparing the effects of various muscular contractions that the number of contractions increased with the muscle relaxation time.[27]

MUSCLE BLOOD FLOW IN CHRONIC COMPARTMENT SYNDROME

Skeletal muscle is not perfused during contraction,[28] and arterial inflow to the muscle vascular bed occurs only between contractions.[29,30] For these reasons, MRP during exercise, i.e., the pressure between contractions, is an important pressure parameter to study.[3,31,32] MRP exceeding 35 mmHg during exercise impedes MBF and is well correlated with the development of swelling, pain, and impaired function of the tissues within the compartment.[3,32] The symptoms of the syndrome correlate well with the decreased local PP, which is the calculated difference between mean arterial blood pressure and MRP during exercise as described in Equation 11.4.[31,32] These patients have normal MBF values in the beginning of exercise (Figure 11.2). MBF decreases significantly at the end of exercise.[3] In another study, patients with elevated intramuscular pressure during exercise had impaired MBF.[33] Other animal and human studies on the influence of increased intramuscular pressure at rest on MBF have demonstrated a significant decrease or even cessational flow when tissue pressure exceeds 30 to 60 mmHg.[18,22,34,35] These results concur with the different theories on the mechanisms of how increased intramuscular pressure affects MBF. They also concur on factual measurements in patients with chronic anterior compartment syndrome in the leg.[3]

Reneman et al. showed that a combined increase in venous and total intramuscular pressure resulted in cessation of muscle capillary blood flow in the presence of arteriolar dilatation.[19] They concluded that the arterioles were not the limiting factor in MBF disturbances in simulated compartment syndromes. They found that muscle capillary blood flow stopped while there was a positive transmural pressure of 24 mmHg over the vascular bed.

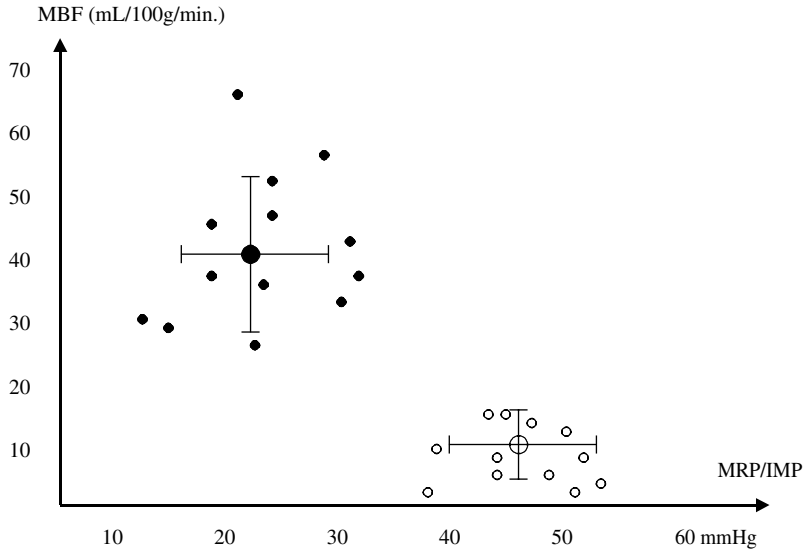

FIGURE 11.2 Correlation diagram of individual values of muscle blood flow (MBF) and muscle relaxation pressure (MRP) in patients with chronic compartment syndrome before surgery. Dots indicate results after 3 min of exercise without pain in the leg. Circles indicate results at the end of exercise when the leg is painful and muscle function is impaired. The bars indicate one standard deviation. (Redrawn from Styf, J., Suurkula, M., and Körner, L., *J. Bone Jt. Surg.,* 69-B, 301, 1987.)

NEUROMUSCULAR FUNCTION IN CHRONIC COMPARTMENT SYNDROME

Patients with chronic compartment syndrome develop muscular weakness during exercise (Figure 11.3). Pain and ischemia may impair torque generation of the muscles involved. Skeletal muscle is sensitive to pressure and direct trauma. Nerve endings are located in the walls of the blood vessels. Ischemia may induce premature muscle fatigue. Muscle fibers themselves are probably insensitive to painful stimuli. However, pain may induce muscle weakness due to inhibition of muscular function. Only in a few patients is there sensory dysfunction at clinical investigation following an exercise test.[36]

MUSCULAR OXYGENATION IN CHRONIC COMPARTMENT SYNDROME

Previous studies have demonstrated decreased MBF as well as impaired muscle oxygenation.[4] These findings suggest ischemia as etiology for chronic compartment syndrome. Changes in intramuscular oxygenation may be measured by a continuous dual wavelength near-infrared spectrometer. The device measures reflection of light transmitted at wavelengths of 760 and 850 nm. Deoxygenated hemoglobin absorbs

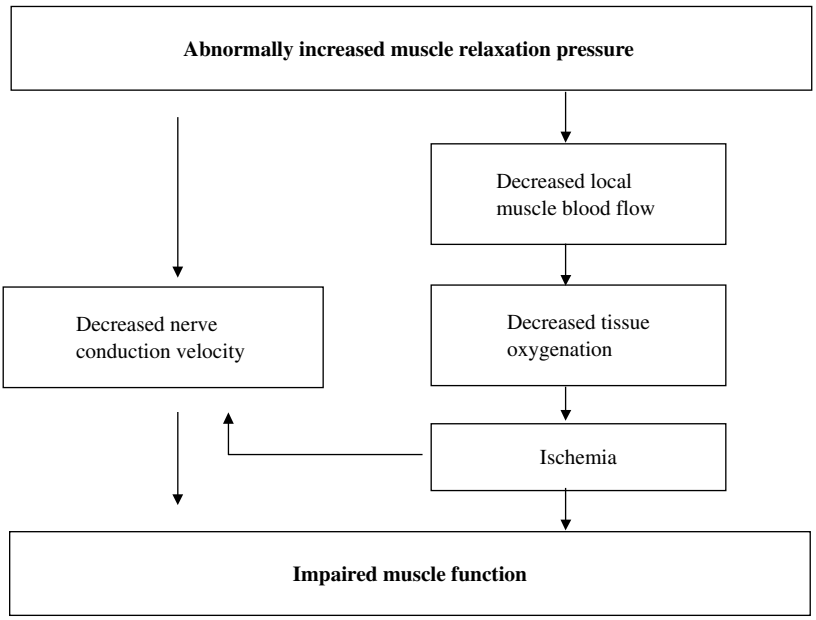

FIGURE 11.3 Pathophysiology of chronic compartment syndrome is based on abnormally elevated muscle relaxation pressure during exercise.

more light at 760 nm, whereas oxygenated hemoglobin absorbs more light at 850 nm. Myoglobin has absorption characteristics identical to those of hemoglobin.

Impaired muscle oxygenation has been demonstrated in human experimental models on chronic compartment syndrome[37,38] as well as in patients with chronic anterior compartment syndrome in the leg.[4,5] Diagnosis of chronic compartment syndrome by near-infrared spectroscopy is based on a slow reoxygenation and default reactive hyperemia (Figure 11.4).

Contractions of an ischemic skeletal muscle bed induce pain and impaired muscle function. Clinical examples of this are patients with chronic compartment syndromes and intermittent claudication. Therefore, muscle contractions appear to be necessary for development of pain in chronic compartment syndrome.

SUMMARY

All muscles swell during exercise. The increased volume of skeletal muscle during exercise is due to increased blood flow and transcapillary filtration into the interstitial space of the muscle. Arterial inflow to the working muscle occurs between contraction, i.e., during muscle relaxation. Abnormally increased MRP during exercise affects local MBF and induces ischemia and pain during exercise. Intramuscular pressure at rest after exercise does not normalize within 10 min. Changes of intramuscular oxygenation during exercise and at rest after exercise may be measured by near-infrared spectroscopy. Changes of local MBF may be measured at rest after exercise by photopletysmography.

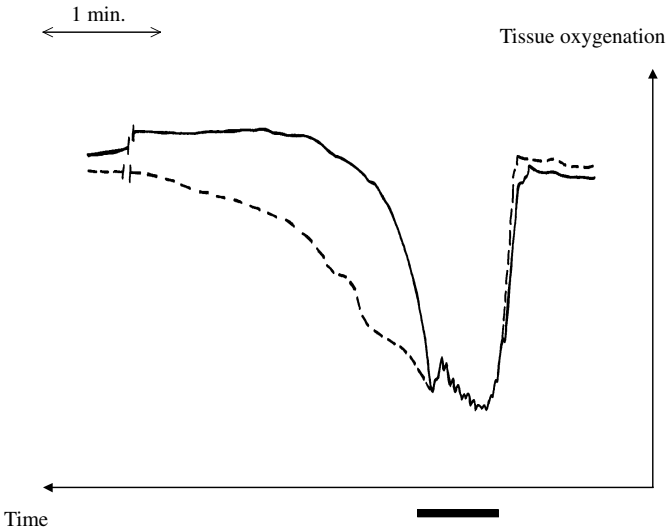

FIGURE 11.4 Tissue oxygenation measured by near-infrared spectroscopy. The bold line shows the changes in a normal subject. Patients with chronic compartment syndrome (dotted line) have delayed recovery of tissue oxygenation. The time bar indicates the time period of concentric exercise.

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12 Clinical Diagnosis of Chronic Compartment Syndromes

INTRODUCTION

Chronic compartment syndrome is defined as a painful condition in which increased intramuscular pressure during exercise impedes muscle blood flow and impairs function of the tissues within the compartment.[1,2] Diagnosing the causes of chronic pain induced by exercise in the extremities is difficult because patients have few or no specific symptoms at rest. Depending on location and severity of symptoms, exercise tests may include concentric muscular activity against resistance (e.g., body weight), indoor walking, exercise on an ergometer, running on a treadmill, or outdoor running on a track or field. Upper-extremity exercise may include whipping with a whisk, playing a musical instrument, throwing a ball repeatedly, pulling the reins tight during simulated harness racing, or any activity that elicits the symptoms. A work simulator is most helpful.[3,4] The important thing is to induce the patient's effort-related symptoms and signs by an exercise test and to perform a clinical examination when the patient is symptomatic or in pain.

SYMPTOMS AND CLINICAL FINDINGS

Symptoms and clinical findings correlate well with the pathophysiology (Figure 12.1). Patients complain of local swelling, tightness, and pain during and at rest after physical activity.[1,2,5–8] Swelling is a sign of increased volume of the compartment. Fascial defects may be related to the increased tension created by the increased volume of the compartment contents. Muscle tissue may herniate through the fascial defects. The ischemic muscle pain is intense enough to force the athlete to stop the activity. The symptoms reverse within 20 min to hours at rest. Sometimes patients describe clumsiness and weakness at the time of pain. The athletes experience ischemic pain that force them to quit exercising when blood flow decreases.[2,6,9,10]

At clinical investigation, the compartment muscles are hard on palpation following exercise. The impaired muscle function at the end of an exercise test can be evaluated clinically by estimating the strength of an active contraction against resistance. Decreased force generation in this situation is a sign of impaired muscle function induced by ischemia and pain (Figure 12.1). Nerve dysfunction impairs sensitivity and also induces muscular weakness. Dysesthesia may be recorded in a few cases.[9,11] Distal pulses are always normal.

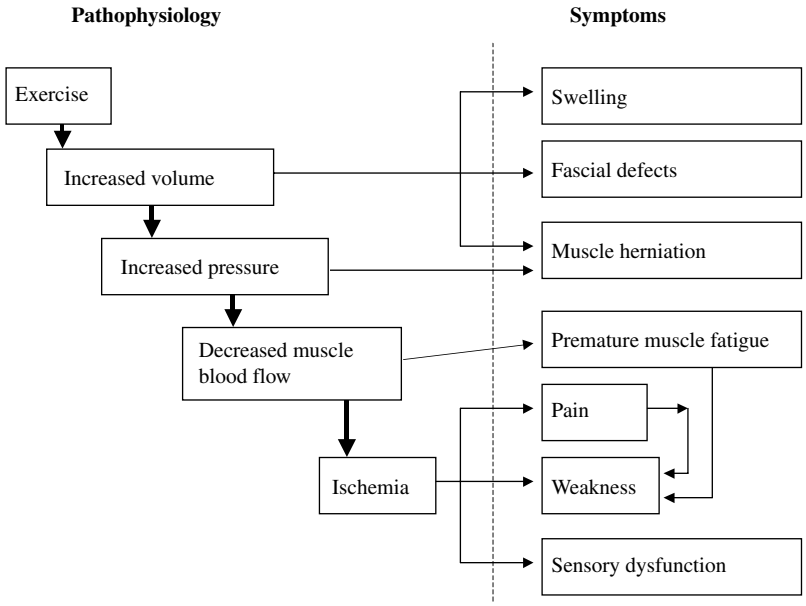


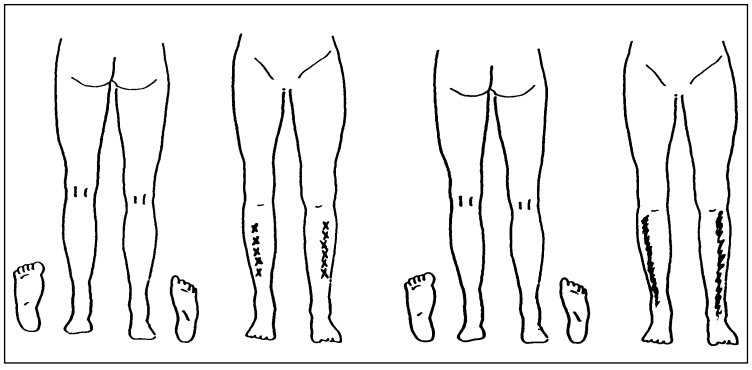
FIGURE 12.1 The connection between pathophysiology and symptoms in chronic compartment syndrome.

CHRONIC ANTERIOR COMPARTMENT SYNDROME IN THE LEG

A patient's history of pain induced only during exercise, occurring only in the anterior aspect of the leg, and requiring the patient to interrupt the physical activity suggests chronic anterior compartment syndrome. Between 70 and 80% of patients who were operated on for the syndrome in the leg were runners.[6,9,12] The syndrome is bilateral in 60 to 90% of the patients.[1,9,13] A patient's pain drawing is a helpful tool to investigate single and multiple locations, as well as impaired sensation distal to the affected compartment (Figure 12.2). Table 12.1 compares the history of a group of patients with chronic anterior compartment syndrome in the leg and patients with chronic anterior leg pain for other reasons.

SYMPTOMS

Most patients are symptom-free at rest. Pain with multiple locations in the leg induced by a short running distance, and allowing the patient to continue running despite the pain, indicates an etiology of noncompartment syndrome.[6] Clinical investigation following an exercise test that elicits the symptoms is particularly useful. Swelling is a sign of increased volume of the compartment. Following an exercise test, patients experience pain over the muscles of the anterior compartment.



I. II.

FIGURE 12.2 Two examples (I and II) of pain drawings by patients with chronic anterior leg compartment syndrome.

SIGNS

Between 30 and 50% of patients with the syndrome are tender at palpation over the anterior margin of the tibia. These patients have periostitis of the anterior margin of the tibia.[6,10] Muscle herniation is seen in 20 to 60% of patients.[14] Decreased sensitivity over the first interdigital cleft and the dorsum of the foot is a sign of impaired nerve function,[9,11] which sometimes occurs in patients with chronic anterior compartment syndrome. However, few patients report these symptoms spontaneously. Recordings of nerve conduction velocity and the EMG signal are both usually normal when recorded at rest.[1,9] The impaired muscle function at the end of an exercise test can be evaluated clinically by testing the strength of active dorsiflexion against manual resistance. MRI and phlebography are not useful in diagnosis of compartment syndrome. However, near-infrared spectroscopy,[15] laser Doppler flowmetry,[16] and thallium-201 chloride scintigraphy[17,18] have been suggested as useful tools to diagnose the syndrome.

In summary, a patient's history indicates chronic anterior compartment syndrome if pain is induced only by athletic activity, pain that occurs only in the anterior aspect of the leg, and pain that forces the athlete to interrupt running.[6] A patient's history does not indicate chronic anterior compartment syndrome if there is pain with multiple locations in the leg or if pain is induced by a short running distance, allowing the athlete to continue running despite the pain (Table 12.1).

CHRONIC LATERAL COMPARTMENT SYNDROME IN THE LEG

Only a few cases of chronic lateral compartment syndrome have been published.[1,6,10,19] It has been seen to occur in 10%[1] and in less than 5%[9] of patients with chronic anterior compartment syndrome. It has been suggested that history

TABLE 12.1
Results of History by a Questionnaire and Clinical Findings at Rest Before Physical Exercise in 80 Patients with Suspected Chronic Anterior Compartment Syndrome (CACS) who were Referred to an Orthopedic Clinic[a]

	CACS (n = 22)[b]	non-CACS (n = 58)[b]
History		
Only anterior location of leg pain	68%	29%
Pain induced only by running	55%	10%
Pain necessitating interruption of running	55%	12%
Pain only at rest after running	0%	12%
Pain during walking	27%	29%
Running distance that induced pain (km)	4.5	0.6
Range of running distance (km)	0.2–10	0.01–3
Clinical Findings at Rest Before Exercise		
No tenderness over the anterior margin	55%	24%
Tenderness only over the anterior margin	32%	19%
Tenderness over other locations in the leg	14%	76%
Tenderness over the anterior tibial muscle	18%	17%
Muscle hernia over the anterior compartment	23%	3%
Muscle hernia over the lateral compartment	0%	5%
Pes cavus or pes pronatus	14%	14%

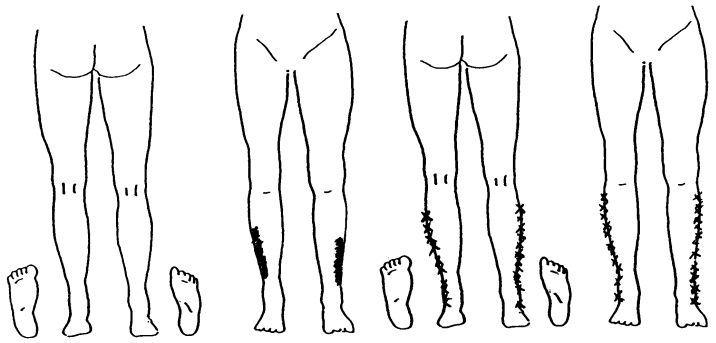
[a] Following diagnosis by intramuscular pressure recordings during exercise and at rest after exercise, 22 patients with CACS in the leg are compared with 58 patients with chronic anterior pain (non-CACS) for other reasons.
[b] Number of patients.

Source: From Styf, J.R. and Körner, L.M., *Acta Orthop. Scand.*, 58, 139, 1987.

is often sufficient to establish the diagnosis.[1] However, in a study of 22 patients with anterolateral pain, only 1 proved to have the syndrome in the lateral compartment as verified by intramuscular pressure recordings.[9,20] As reflected by the literature, the syndrome seems to be an unusual cause of pain in the lateral compartment. The peroneus tunnel syndrome has been reported to be a more common reason for anterolateral pain than chronic lateral compartment syndrome has.[9,20,21] The differential diagnosis of anterolateral leg pain is discussed in Chapter 14.

Symptoms

The pain is located over the lateral compartment. Patients may experience pricking sensation or numbness over the ankle joint and dorsum of the foot after an exercise test.



I. II.

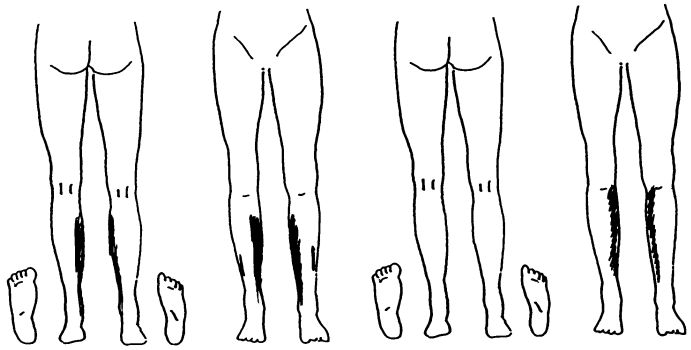
FIGURE 12.3 Pain drawings by two patients with chronic lateral compartment syndrome in the leg.

Signs

Most patients have a fascial defect and muscle herniation 10 to 12 cm proximal of the lateral malleolus. Sensitivity may be disturbed over the dorsum of the foot. The sensitivity over the first interdigital cleft is normal (Figure 12.3).

CHRONIC POSTERIOR COMPARTMENT SYNDROME IN THE LEG

The existence of a chronic compartment syndrome in the deep and superficial posterior compartments is a controversial topic. Posteromedial leg pain, which is clinically diagnosed as medial tibial syndrome, is commonly seen in runners (Figure 12.4). Pain along the posteromedial part of the leg is a common condition



I. II.

FIGURE 12.4 Two examples of pain drawings by patients with medial tibial syndrome.

and accounts for 60% of all lesions causing pain in the athlete's leg.[22] Some authors report normal intramuscular pressure measurements in the posterior compartments of these patients.[23–25] Normal intramuscular pressures during exercise and at rest after exercise were measured simultaneously in the flexor digitorum muscle and the tibialis posterior muscle.[26] D'Ambrosia et al. found no basis for increased intramuscular pressure in either the anterior or the posterior compartments as the cause for shin splints.[23] Davey et al. reported that the tibialis posterior muscle is contained in an osteofascial compartment separate from the rest of the deep posterior compartment.[27] Simultaneous pressure recordings in the flexor digitorum and tibialis posterior muscles of 28 consecutive patients with severe exercise-induced posterior leg pain could not prove that any of these patients had the syndrome.[26] They concluded that chronic compartment syndrome was not the cause of posteromedial leg pain. One possible reason for the controversy in the literature is that pressure is measured by techniques that are less, or even not at all, suitable for dynamic pressure recordings. Finally, simultaneous intramuscular pressure and EMG recordings would exclude muscular activity as a reason for elevated intramuscular pressure.

Others believe the condition may be a chronic compartment syndrome in the deep posterior compartment of the leg,[27–29] whereas still others feel it is an overuse syndrome[23–26,30] which may have multiple etiologies. It has been suggested that chronic compartment syndrome is underdiagnosed.[12] The syndrome has been defined in a different way. Patients did not always have abnormally elevated pressure on which the diagnosis of the syndrome was based. Furthermore, pressure was measured only at rest before exercise with the needle injection technique,[31] a technique that is known to give erroneously high intramuscular pressure readings.[32,33]

Symptoms

Patients experience pain along the posteromedial border of the tibia and the posteromedial soft tissues. The pain may range from a dull, aching discomfort to an intensive, persistent pain, which is aggravated by physical activity. The pain and weakness are associated with running.

Signs

Patients have well-localized area of tenderness over the middle or distal thirds, or both, of the posteromedial ridge of the tibia (Figure 12.4). They have no motor, sensory, or circulatory disturbances. Many patients have excessive pronation of their feet.

CHRONIC COMPARTMENT SYNDROME IN THE FOOT

The syndrome is most uncommon in this location. Middleton and co-workers presented a 16-year-old girl with exercise-induced acute compartment syndrome who had progressive pain and numbness in her feet after a day of high-impact aerobics.[34] Lokiec and co-workers presented an 18-year-old ballet dancer with

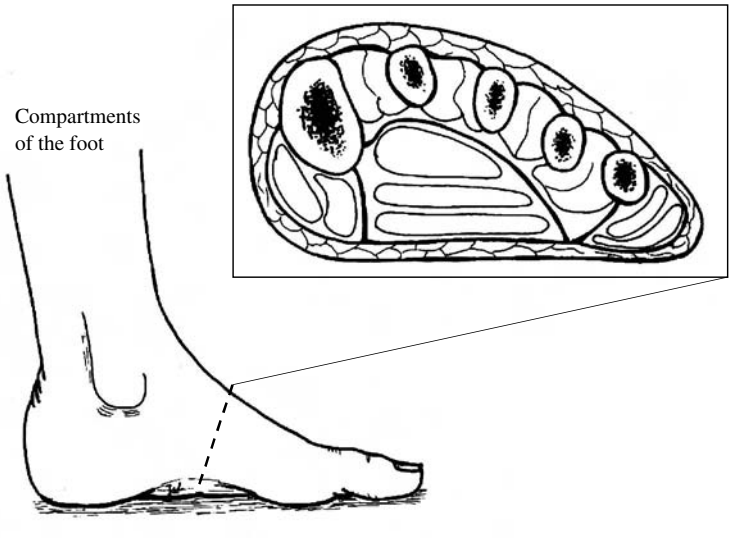


FIGURE 12.5 The foot is composed of nine compartments that may be affected by elevated muscle pressure during exercise and at rest after exercise.

pain in both his feet induced by dancing for 10 min.[35] The syndrome was described in a 19-year-old male triathlete.[36] The diagnosis is difficult because of the many compartments of the foot (Figure 12.5).

Symptoms

Patients experience pain and swelling over the longitudinal arch of the foot. They complain of exercise-induced cramps in the plantar aspect of the foot. Walking and ordinary daily activities cause no discomfort.

Signs

On examination following exercise, the midfoot is swollen over the medial aspect. The foot is tense and tender. Symptoms subside after 10 min of rest. MRI has revealed hypertrophy of all the muscles of the medial and central compartments of both feet. Pressure in the two compartments was between 35 and 80 mmHg following an exercise test that induced the clinical symptoms.

CHRONIC COMPARTMENT SYNDROME IN THE THIGH

This location of the syndrome is most uncommon. Raether et al. described a 26-year-old long-distance runner who experienced intermittent claudication, weakness, and pain in the posterior compartment of the thigh after 4 miles of running.[37] Next day, the thigh was aching. Elevated pressure in the posterior compartment and the exercise-induced pain reversed after treatment by fasciotomy. In another

study, two patients with pain and swelling over the tensor fasciae lata muscle had elevated intramuscular pressure and enlarged muscle compartment on computed tomography.[38]

Orava et al. diagnosed the chronic syndrome in nine patients over a 13-year-period.[39] They were power lifters, body builders, and endurance walkers, and one cyclist. Four of the nine patients had used steroids. The diagnosis was not confirmed by intramuscular pressure recordings.

Patients complained of gradually worsening pain in the involved muscles of the thigh. The thigh muscles were numb and weak after exercise. One patient had a small tear of the fascia. Muscle biopsy may show superficial muscle cell necrosis. Metabolic rhabdomyolysis, muscle strain, and poliomyelitis were a few differential diagnoses in these cases.[39] Exercise-induced medial thigh pain was diagnosed as entrapment of the obturator nerve in 29 patients.[40] Normal needle EMG demonstrated denervation of the adductor muscles.

CHRONIC COMPARTMENT SYNDROME IN THE FOREARM

The forearm contains at least three compartments: the extensor, the superficial, and the deep flexor compartments. Chronic compartment syndrome in the forearm seems to be a rare condition, because only a few cases have been described following trauma,[41] routine strenuous activity,[42,43] and exposure to vibrating tools.[44] Diagnosis of the syndrome demands a high level of suspicion. Competitive motor racing is a form of excessive and continuous exercise. Rydholm et al. presented 14 patients with effort-related pain in the dorsal compartment of the forearm. Six of them had abnormally elevated intramuscular pressure.

Symptoms

Patients experience swelling and pain in the forearm due to exercise, followed by weakness of the flexion grip strength of the hand. They may experience pain during passive extension of the wrist and fingers after an exercise test. Numbness and tingling in the distribution of the median nerve after exercise have been described.[41,42] Gradual decrease in muscle strength forces patients to discontinue exercise after a while. Symptoms fade away on stopping the activity, but recur with resumption of activity.

Signs

Patients have a full range of motion in the elbow, wrist, and finger joints. Swelling, firmness, and induration over the muscle bellies of the forearm are associated with weakness of the hand grip after exercise.[42,43,45] Muscular hypertrophy and postexercise edema of the abductor pollicis longus and extensor pollicis brevis muscles have been described after unaccustomed strenuous exercise.[46] The muscle bellies are tender at palpation and at passive stretch immediately after an exercise test that elicits the symptoms.

CHRONIC COMPARTMENT SYNDROME IN THE HAND

Chronic compartment syndrome is an unusual cause for hand pain. It has been described in the first web space in the first dorsal interosseous muscle. The function of this muscle is to flex the metacarpophalangeal joint and extend the proximal interphalangeal joint of the index finger. The syndrome has been described among hairdressers, guitar players, and stenographers.[7] Chronic compartment syndrome was diagnosed in 4 of the 15 patients with writers' cramp.[7]

Symptoms

Patients experience pain and swelling over the first web space. They experience weakness of the pinch grip and clinical symptoms similar to patients with writers' cramp. Patients complain of exercise-induced cramping sensation in the radial side of the dorsum of the hand.

Signs

At investigation following an exercise test, the first web space is swollen. Patients may experience pain at passive stretch of the muscle. Since no sensory nerves are found in this compartment, abnormalities related to a sensory nerve will be lacking in this localization.[7] Physical signs of hypoesthesia are not present. However, after exercise on a work simulator,[3] the peak torque for performing lateral key pinch and three-point pinch decrease, and the time for tolerated activity decreases significantly.[4] Muscle relaxation pressure during exercise[7] and intramuscular pressure at rest after exercise[4,47] increase to 30 to 50 mmHg. Hand volume increased by 20 mL as assessed by volumetry.[4]

CHRONIC COMPARTMENT SYNDROME IN THE LUMBAR SPINE

The lumbodorsal compartment of the lumbar spine is a relatively unyielding osteofascial space, which is the prerequisite for the chronic compartment syndrome in the lumbar spine.[48–50] Chronic compartment syndrome in the muscles of the lumbodorsal compartment is extremely uncommon in patients with exercise-induced low-back pain.[8] Only 1 out of 12 patients with strictly exercise-induced paravertebral pain in the lumbar spine had the syndrome. The syndrome occurs among patients who have a high physical activity level, pain only during and immediately after activity, and no neurological deficits in the legs. In a larger series of patients with low-back pain, it was diagnosed in 7 out of 102 patients.[51] Pressures during exercise and at rest after exercise in the erector spine muscles were recorded over a 15-year period in more than 30 patients who fulfilled all the clinical criteria for suspected chronic compartment syndrome.[8] Only five patients with chronic compartment syndrome in the lumbar spine were diagnosed. The syndrome seems to be a rare cause of exercise-induced low-back pain.

Patients have paravertebral pain induced by exercise in the lumbar spine. They have no low-back pain at rest. They are not on sick leave. At physical investigation, they have a normal range of motion and no neurological deficits in the lower extremities. In unilateral cases, a paravertebral swelling is visible.

ADDITIONAL DIAGNOSTIC TOOLS

Measurements of tissue oxygenation by near-infrared technique (Figure 11.4),[15] by PO_2 measurements,[52] or local muscle blood flow by laser Doppler flowmetry[16] may provide additional methods to diagnose the syndrome. Patients with chronic compartment syndrome have a slow or delayed reoxygenation and default hyperoxygenation following an exercise test. Measurements of local muscle blood flow by Xenon[133] should be used only when clinical research of the syndrome is performed.[2] Recently, measurement of muscle blood flow by a noninvasive photopletysmographic technique to diagnose chronic compartment syndrome was presented.[53]

MRI and phlebography are not useful in diagnosis of chronic compartment syndrome. However, thallium-201 chloride scintigraphy[17,18] and $^{99}Tc^{m}$ methoxyisobutylisonitrile scintigraphy have been suggested as diagnostic tools of the syndrome.[54] These techniques have been reported on 46 patients who were suspected on clinical grounds of having a chronic compartment syndrome. The sensitivity of the test and measurements was 80%.

SUMMARY

Chronic compartment syndrome in the leg occurs in a minority of patients with exercise-induced leg pain. A patient's history of pain induced only during exercise, occurring only in the anterior aspect of the leg, and requiring the patient to interrupt the physical activity suggests chronic anterior compartment syndrome. Physical examination immediately following an exercise test is most helpful. The involved muscles are swollen, weak at testing, and tense at palpation. History and clinical signs are helpful in selecting patients for pressure studies during or following exercise. Near-infrared spectroscopy is a noninvasive additional tool to diagnose the syndrome.

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13 Surgical Techniques and Results of Treatment of Chronic Compartment Syndromes

INTRODUCTION

There are three options of treatment for patients with chronic compartment syndrome. The first is to adjust the activity to a level that is pain-free. Second, patients may try other nonsurgical treatments like nonsteroid antiinflammatory drugs, diuretic treatment, massage, and physical therapy.[1] These treatments have not been successful. Third, surgical decompression by fasciotomy has been shown to give good results in most patients.[2–6]

Bear in mind that patients may have two or more leg diseases simultaneously. Successful treatment of one disease may not lead to full recovery, because a second disease is left undiagnosed and untreated. Usually, patients with multiple locations of leg pain do not have chronic compartment syndrome. Also, leg pain in patients with disseminated chronic musculoskeletal pain is not indicative of chronic compartment syndrome.

PATHOPHYSIOLOGY OF TREATMENT

The abnormally elevated intramuscular pressure in patients with chronic compartment syndrome impairs local muscle blood flow. Patients are affected by the local muscle ischemia. They experience premature muscle fatigue, swelling, and pain induced by exercise. Muscle tissue normally swells during exercise. In patients with chronic compartment syndrome, the swelling, normal or abnormal, is combined with decreased compliance of the compartment.

The goal of treatment is to restore a painless leg with normal neuromuscular function of the tissues in the compartment. This is achieved by fasciotomy, which normalizes local muscle blood flow and restores perfusion pressure by decreasing muscle relaxation pressure during exercise and intramuscular pressure at rest after exercise. The syndrome is never associated with sequel of ischemic contracture.

Treatment by fasciotomy allows the muscle to swell without increased intramuscular pressure as a result. Fasciotomy alters the compliance of the compartment as illustrated in Figure 13.1. Fasciotomy also increases the perfusion pressure by normalizing the muscle relaxation pressure during exercise and intramuscular pres-

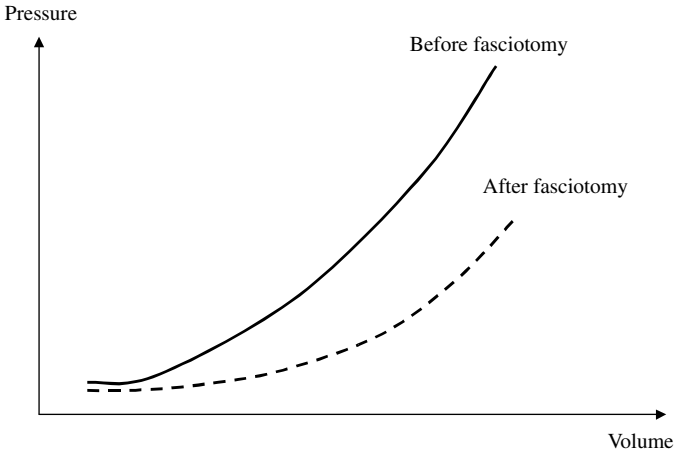

FIGURE 13.1 Compliance in patients with chronic compartment syndrome before and after fasciotomy. After fasciotomy the muscle tissue in the compartment swell but intramuscular pressure increases to normal levels.

sure at rest after exercise. Muscle relaxation pressure is the pressure in the relaxed muscle between two muscle contractions.

NONOPERATIVE TREATMENT

Nonoperative treatment of leg pain includes physiotherapy, antiinflammatory drugs, and orthotics, which may play an important role in the management of patients with exercise-induced leg pain. However, such treatment is usually not effective in the management of chronic anterior compartment syndrome. In a pilot study, treatment by massage significantly increased the work performed in dorsiflexion before pain developed.[1] No significant difference in intramuscular pressure before and after massage was found.

A trial of rest for about 1 month followed by gradual resumption of normal activities may relieve the pain, but the symptoms often return as the athlete returns to the same intensity level of exercise. Nonoperative treatment for up to 1 year was found to be unsuccessful.[7] Therefore, nonoperative treatment options include only modification of the subject's activity level.

SURGICAL TREATMENT

Surgical decompression by fasciotomy or partial fasciectomy of the symptomatic compartment is the only option available if the athlete is unable or unwilling to give up the activity that induces the symptoms. About 50% of patients with chronic anterior compartment syndrome in the leg have periostitis as a symptom.[8] The symptom may be induced by increased tension of the fascial insertion on the anterior ridge of the tibia. Fasciotomy also alleviates the pull from tensile forces acting on the anterior margin of the tibia by the fascia.

Leg Compartments

The leg is by far the most common site for the syndrome.

Surgical Technique

Several techniques have been described for decompression of leg compartments. Different techniques use variations of skin incisions. Variations of the techniques have been developed to decrease size of skin incisions. Some use two incisions,[3,7,9] whereas others include the use of one skin incision.[4–6] It is important to perform complete decompressions extending over the whole muscle compartment (Figure 13.2).

For the anterior compartment, a single skin incision of approximately 5 cm (2 in.) in length is made over the proximal part of the middle third of the leg about 4 cm lateral to the anterior margin of the tibia.[5] It may also be made more laterally

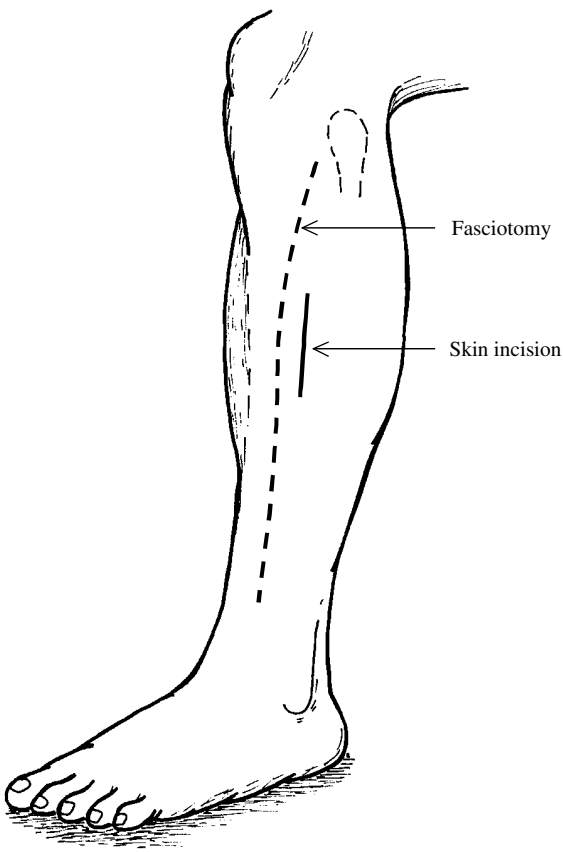

FIGURE 13.2 Decompression of the anterior compartment of the leg by a single 5- to 7-cm-long skin incision 3 cm lateral of the anterior margin of the tibia. By a tunnel technique, the fascia may be divided to the Gerdie's tubercle proximally and to the dorsal retinaculum distally.

halfway between the tibial crest and the fibular shaft.[10] The fascia over the anterior tibial muscle is dissected from the subcutaneous tissue and from the subfascial tissues all the way to the extensor retinaculum at the ankle joint and to Gerdies tubercular at the knee joint. The fascia is incised over the tibialis anterior muscle 2 to 3 cm lateral to the anterior margin of the tibia. Care must be taken to avoid the superficial peroneal nerve at its distal position.[5,11] The nerve pierces the fascia at the junction of the middle and distal thirds of the leg. It often may have an anomalous course.[12] Fasciotomy should include a fascial defect if present.

If two incisions are used, a suitable length is 4 cm, each 12 to 15 cm (5 to 6 in.) apart.[11] Complete longitudinal release of both compartments is accomplished by a fasciotome[10] or long Metzenbaum scissors. Using a meniscotome is not recommended, as the bills of the instrument are not sufficiently long to contain the fascia during the release.[5,13,14] This instrument may also damage muscle tissue. A fascial spatula inserted beneath the fascia to be divided can protect the subfascial tissues. The spatula contains a groove that guides the cutting blades of the cutting instrument.

Others have used two longitudinal incisions centered over the intermuscular septum. The advantages of the two-incision technique are that it gives easier access to the anterior and lateral compartment fascia and it is easier to confirm that the fasciotomy is complete. Fasciotomy of the lateral compartment in addition to anterior compartment is unnecessary to perform in patients with only chronic anterior compartment syndrome.[15]

Some authors recommend fasciotomy of both the anterior and lateral compartment in patients with chronic anterior compartment syndrome. Both compartments are opened by a 4- to 5-cm-(2 in.)-long skin incision halfway between the fibular shaft and the tibial crest in the mid portion of the leg. This incision gives an easy access to both compartments. The anterior intermuscular septum is identified and avoided because the superficial peroneal nerve lies in the lateral compartment near the septum.

The posterior compartments may be decompressed by a 10- to 12-cm-(4- to 5-in.)-long skin incision about 3 cm posterior to the posterior margin of the tibia (Figure 13.3). By making the incision at this location, the surgeon avoids injury to the saphenous nerve and vein. Stay close to the fascia and retract the saphenous veins and nerves anteriorly. The subcutaneous tissue is bluntly released from the fascia in distal and proximal directions. Sometimes it may be necessary to put a ligature on the distal venous perforant. The fascia over the flexor digitorum muscle is divided all the way to the retinaculum behind the medial malleolus. The soleus muscle is released from the posteromedial margin of the tibia. The fasciotomy is extended proximally and then distally till the retinaculum behind the medial malleolus is reached.

If the soleus attaches to the tibia in the distal third, it should be released from its distal insertion on the posteromedial margin of the tibia. If the operation is done under general anesthesia with a thigh tourniquet, the tourniquet must be released and the homeostasis controlled before the wound is closed. Some investigators were not able to find a distinct fascial septum separating the tibialis posterior muscle epimysium from adjacent deep posterior musculature,[16,17] whereas others have described the tibialis posterior muscle as a fifth compartment.[18,19]

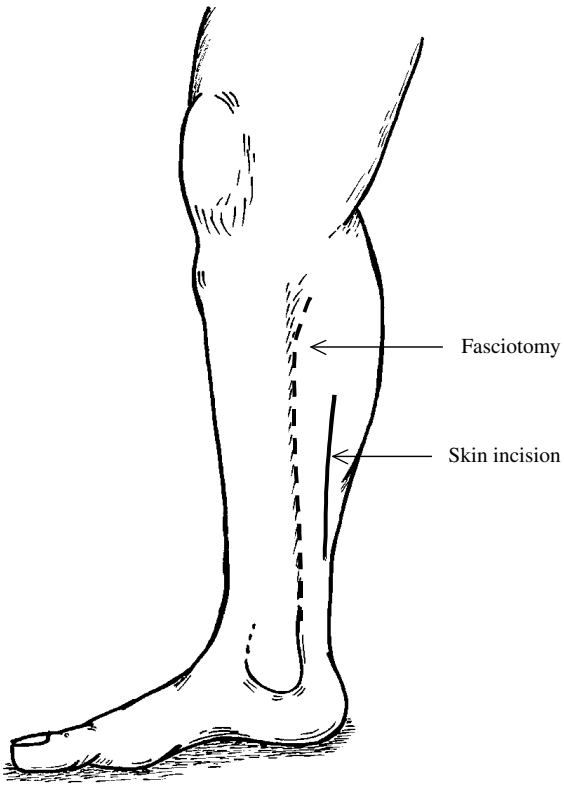

FIGURE 13.3 Fasciotomy of the posterior compartments. The skin incision should be placed at least 2.5 cm (1 in.) behind the posterior margin of the tibia, and be at least 10 to 12 cm (4 to 5 in.) long. The fascial bridge over the soleus muscle and all fascia structures attaching to the tibia should be divided. Blind fasciotomies of the posterior compartments are not recommended — surgeons must see what they do.

Endoscopic Fasciotomy

Subcutaneous endoscopic fasciotomy may be performed through three portals 2.5 cm (one inch) from the anterior margin of the tibia by a 4-mm diameter arthroscope (Figure 13.4). The fascia is cut with a retrograde blade for arthroscopic surgery. By repeating the procedure at each portal, the compartment is decompressed from the level of the fibular head to the nearest point of the extensor retinaculum at the ankle joint.[20]

Other Techniques

Decompression of the posterior compartments has been performed through a 5-cm incision approximately 2 cm behind the posteromedial margin of the tibia.[4,6] A double-incision technique 1 cm posterior of the posteromedial margin has also been described.[11] The need for a separate fasciotomy of the fifth compartment, the tibialis

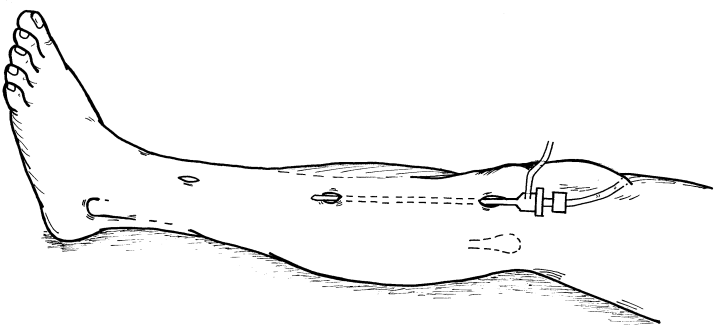

FIGURE 13.4 Endoscopic fasciotomy of the anterior compartment through three portals. A retrograde blade is used to cut the fascia.

posterior compartment, has been advocated to prevent recurrences.[3,4,19,21] Finally, diathermy has been used to cut the fascia.[22] Decompression of posterior compartments by blind techniques should be avoided.

Results

Pain relief for patients following fasciotomy or partial fasciectomy for chronic compartment syndrome is well documented in the literature.[2,4–6,11] Treatment by fasciotomy gives good results in 60 to 100% of patients. Patients' satisfaction is generally rated 85% good or excellent results. The postoperative activity level for patients with chronic anterior compartment syndrome is increased in 60 to 100% of patients. Patients normalize their muscle relaxation pressure after fasciotomy.[5] It therefore appears that fasciotomy, even though it does not treat the cause of the syndrome, is still effective in eliminating the pathological increase in the compartment pressure. After fasciotomy, the contents of anterior compartment swell but the increase of intramuscular pressure is normal. It has also been shown that fasciotomy increases the compliance of the compartment following surgery.[5,8,23,24] Fasciotomy also alleviates the pull from tensile forces acting on the anterior margin from fascia. Most patients experience pain relief and are satisfied with the results of fasciotomy. About 10% of patients may require revision surgery.[5,10,25] Muscle relaxation pressure during exercise and intramuscular pressure at rest after exercise normalize after fasciotomy.[5,8,23,24]

Complications

Complication rates of 5 to 15% have been reported.[2,5,11] Recurrences of the syndrome have been attributed to failure of decompression or incorrect diagnosis. Complications include hemorrhage, wound infection, nerve entrapment and cutaneous nerve injury, and deep vein thrombosis.[2,5,10,25] A lower success rate has been reported in women after fasciotomy.[26] Fasciotomy of the deep posterior compartment for leg symptoms may have a failure rate of 35%.[3,6,22,27,28] Outcome of treatment by fasciotomy of the anterior compartment is better than fasciotomy of the

posterior compartment. One possible explanation for this is that decompression of the posterior compartment through small incisions is not adequate. The presence of nondecompressed compartments may cause symptoms. Another possible option is that diagnosis of posterior leg pain is more difficult and may result in a higher proportion of incorrect or missing diagnoses. Complications to surgery, such as sensitivity to touch, are another explanation to suboptimal results following surgery.

Foot Compartments

Chronic compartment syndrome in the foot compartments is uncommon but is becoming more recognized. Only a few cases of chronic compartment syndrome in the feet have been reported.[29–32]

Surgical Technique

Decompressive fasciotomy of the affected compartments is recommended. The medial compartment is the most commonly involved.[31] Bilateral decompressive fasciotomy is performed through a 5-cm incision along the medial aspect of the midfoot, just beneath the proximal half of the first metatarsal (Figure 13.5). Walking with full weight bearing is encouraged after the third postoperative day and full activity is allowed 8 weeks after surgery if the patient is symptom-free.[29,30]

Results

Good results have been reported following decompression of the medial compartment of the foot.[31] The majority of patients treated by decompression experience pain relief. They are satisfied with the results of the operation because they can

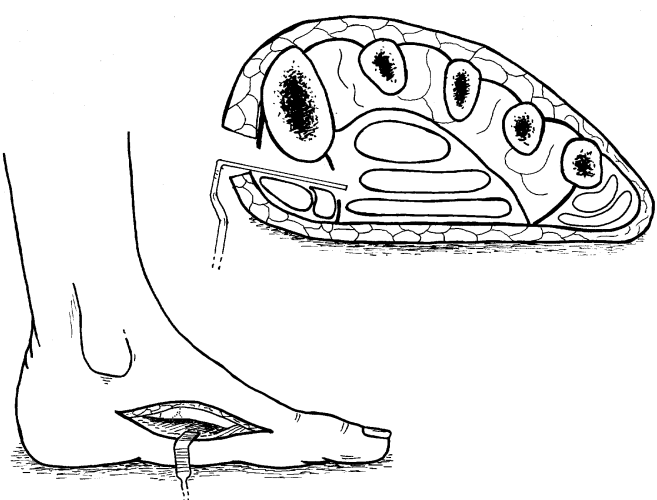

FIGURE 13.5 Medial skin incision that is used to divide the fascial structures of the medial and central compartments of the foot.

increase their level of physical activity.[25] It is unclear if treatment by fasciotomy yields satisfactory results for chronic compartment syndrome in the feet.

Thigh Compartments

Chronic compartment syndrome is an unusual cause of thigh pain. The syndrome has been reported in the anterior or quadriceps muscles of the thigh,[33,34] the posterior compartment,[35,36] and in the tensor fasciae lata muscle.[37] Modified training technique and adjusted activity level are tried first. Different techniques for fasciotomy of the thigh compartments have been described.[34–37]

Surgical Technique

The anterior compartment may be decompressed by an 8- to 12-cm-long skin incision. The fascial incision is continued 10 cm in proximal and distal directions. The fascia over the vastus lateralis muscle just anterior to the tractus ileotibialis may be opened through a 8- to 10-cm (4 in.)-long skin incision. By a tunnel technique, the fasciotomy may be extended 10 cm in a proximal and 10 cm in a distal direction.[34] A surgical drain was used for 24 h. Normal weightbearing started on the same day.

The posterior compartment is decompressed through a 6- to 12-cm-long skin incision in the middle posterior aspect of the thigh. In tall patients, two skin incisions may be necessary. The common femoral fascia over the biceps femoris, semitendinosus, and semimembranosus muscles is divided.[36] The transversal fibers of the fascia are divided and adhesions divided bluntly with fingers (Figure 13.6). The procedure may be performed with local anesthesia in about 50% of patients.[36] The tensor fasciae lata is opened through a longitudinal fasciotomy.[37]

Results

Excellent or good results following fasciotomy of the anterior compartment of the thigh were described in all patients in a series of nine,[34] and in 85% of patients with chronic compartment syndrome of the posterior compartment.[36] Biopsies from fascia were thicker than in normal cases. Complications have been reported in 15% of patients. These include hematoma, wound dehiscence, and local sensory dysfunction around the incision.

Forearm Compartments

Chronic compartment syndrome of the forearm is not very common.[38–42] It has been described in the dorsal compartment[43] and the volar compartments.[38,41,44,45] It has also been reported in association with the flexor compartment and the anconeus muscle.[46,47] The syndrome involving the anconeus muscle presents as lateral elbow pain.

Surgical Technique

Fasciotomy for chronic compartment syndrome in the forearm is carried out similarly to that described for acute compartment syndrome. The same technique as used for acute fasciotomy described by Gelberman et al. may be used to decompress the

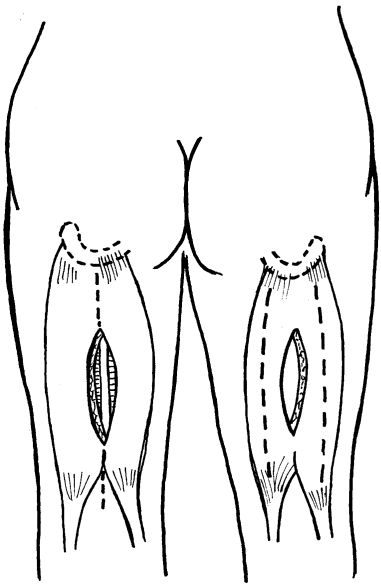

FIGURE 13.6 Skin incision (in the left thigh) and fasciotomy (dotted lines, right thigh) of the hamstring muscles.

extensor and flexor compartments of the forearm.[48] A longitudinal incision is made over the flexor muscles of the forearm (Figure 6.10 and Figure 6.11). The superficial flexor muscles are retracted laterally and the flexor carpi ulnaris muscle medially. The fascia over the deep flexor muscles must also be split longitudinally.[38,39,44] The

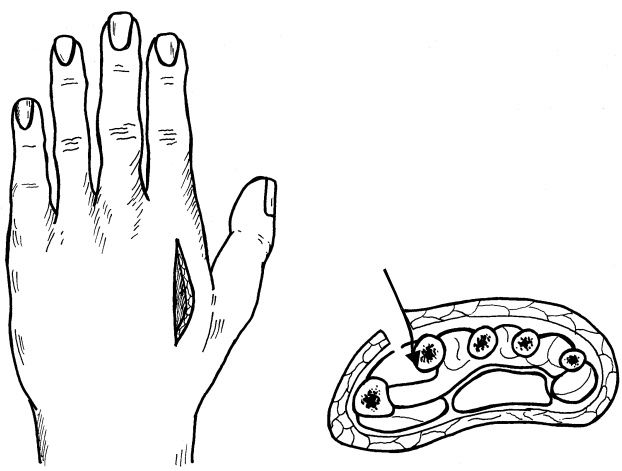

FIGURE 13.7 Fasciotomy of the first dorsal interosseus muscle of the hand.

skin is sutured primarily. It is important to decompress the carpal tunnel as well. In this way, an edge effect from the volar ligaments on the median nerve is avoided.

Chronic compartment syndrome in the flexor–pronator muscle group was treated in one patient by a 3-cm skin incision. The lacertus fibrosus was split over a length of 10 cm.[45]

Results

Decompression by fasciotomy normalizes intramuscular pressure in all patients[49] and gives satisfactory results in the presented case reports.[38,39,44,45,49]

Hand Compartments

Patients with exercise-induced pain over the first interdigital cleft, resembling writers' cramp, may have abnormally elevated intramuscular pressure recordings during exercise and at rest after exercise.[50] The syndrome is successfully treated by fasciotomy of the fist dorsal interosseous muscle with the patient under radial nerve block.[50,51] A 3- to 4-cm-long curved skin incision is used (Figure 13.7). Compression dressing is applied for 10 days. Full activity is allowed 2 weeks after the operation.[50] Fasciotomy relieves the pain and normalizes the muscle relaxation pressure during exercise and intramuscular pressure at rest after exercise.[42,50–53]

Lumbar Spine

Chronic compartment syndrome in the paravertebral muscles is a rare reason for low-back pain. During a 20-year period, only 12 patients with strictly exercise-induced low-back pain were suspected, on clinical grounds, to have chronic compartment syndrome in the erector spinae muscle. Only five of them had abnormally elevated intramuscular pressures, confirming the diagnosis.[54] Konno and co-workers diagnosed the syndrome in 7 out of 102 patients with low-back pain.[55]

A 10-cm-long skin incision is made 2 to 3 cm lateral to the spinal processes and centered at the spinous process of the first lumbar vertebrae. The fascia covering the paraspinal muscles is split to the sacrum and 10 cm proximal of the L1 level.[54] Fasciotomy of the erector spinae muscle has relieved back pain in the few reported cases.[54,55]

POSTOPERATIVE MANAGEMENT

Early mobilization is recommended by most authors to prevent scarring and adhesion.[5,6,11,56,57] The sutures are removed after about 2 weeks. This author prefers the intracutaneous suturing technique. Light running is initiated and exercise is progressed as tolerated over the next 2 to 6 weeks. Others recommend ambulating by crutches for the next 2 weeks. The first 2 days patients are treated by adjusted physical activity level, external compression of the leg, and stretching of leg muscles. Patients walk by crutches for the first 1 to 3 days. The elastic compression bandages are released after 3 days. Full weight bearing is encouraged after 12 h and patients are free to walk unlimited during the first 12 days and thereafter increase their physical activity level by jogging. Athletic activities, including competition, are

allowed after 6 weeks.[5,13,14,58–60] Return to full athletic activity usually occurs between 4 and 8 weeks.

SUMMARY

Treatment of the syndrome by fasciotomy gives good results in 60 to 100% of patients. Patients experience a high level of pain relief, and are satisfied with the outcome because they are able to increase their level of physical activity. The surgeon must use an atraumatic technique. Surgeons must be able to see what they are doing; there should be no blind fasciotomies in the posterior or lateral compartments. The anterior compartment may be decompressed by limited or by multiple skin incisions. Patients should be informed of the less favorable outcomes following fasciotomy of the posterior compartments and complication rates of surgery. Patient selection is important. Patients who experience leg pain as a part of disseminated chronic musculoskeletal pain do not benefit from surgical treatment.

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14 Differential Diagnosis of Chronic Leg Pain Induced by Exercise

INTRODUCTION

Patients with chronic compartment syndrome may have other painful leg conditions simultaneously. Therefore, discussion of differential diagnosis of patients with chronic leg pain is important. Leg pain of patients has many etiologies. Each of the leg tissues may be a source of pain. Pain may originate from bone, periost, muscle, fascia, tendons, arteries, veins, and nerves. The tissues are closely gathered in the leg and overlap each other. Diagnosing the causes of chronic pain in the leg may be difficult when patients have few specific symptoms and signs at rest. Therefore, clinical investigation following an exercise test is helpful. The test may include work on an ergometer, running, or any specific activity that elicits the typical pain or dysfunction in the patient.

In diagnosing the conditions, it is helpful to determine from the patient's history whether the pain is strictly exercise-induced or not. Some diseases are induced strictly by exercise, such as the chronic compartment syndromes and entrapment of the popliteal artery. Other diseases may give more symptoms at rest after the exercise, such as the medial tibial syndrome and venous insufficiency. Pain may occur more randomly both at rest and during exercise, such as in nerve entrapments.

Understanding of leg anatomy, pathogenesis, and pathophysiology of leg diseases is most helpful in clinical diagnosis of the causes of chronic leg pain. History and clinical findings are helpful in selecting patients for pressure recording studies. Intramuscular pressure recording and measurement of tissue oxygenation during exercise with near-infrared spectroscopy are helpful to establish or exclude the diagnosis of chronic leg compartment syndrome. Findings can be compared with the symptom-free limb. Range of motion of all joints from the hip down to the foot joints must be examined. Peripheral nerves muscles and bony prominences must be thoroughly palpated. Pseudoradicular pain, sensory disturbances, and muscular dysfunction masquerading lumbar radiculopathy may be caused by lower-extremity peripheral nerve entrapment.[1] In a study of 150 patients with leg pain caused by exercise, 33% had chronic compartment syndrome, 25% had stress fractures, 14% had muscle strains, 13% had medial tibial stress syndrome, 10% had neuropathies, and 4% had venous diseases.[2] One patient had spinal stenosis. In another study on 98 patients, all of whom were suspected on clinical grounds of having a chronic leg compartment syndrome, only 25% had the syndrome.[3] In this chapter, possible

reasons for exercise-related leg pain will be discussed. The aim is to give a brief overview of possible causes for chronic leg pain.

Overuse Injuries in the Leg

Injuries resulting from overload pose major diagnostic and therapeutic problems. The leg is, second to the knee, the most common site of pain in runners, accounting for about 20% of all running injuries.[4] Figure 14.1 illustrates the relationship between load and tissue growth or strength. Overuse injuries by extrinsic factors such as excessive load and training errors may induce chronic leg pain. Extrinsic factors such as training, poor techniques, poor shoes, and inappropriate surfaces, as well as intrinsic factors such as malalignment and muscle imbalance, may contribute to chronic overuse injury.[5] Intrinsic factors include leg length discrepancies and malalignment. The origin of plantar flexor muscles on tibia may vary considerably between individuals.[6,7] Increased pronation of the feet may be associated with injuries such as the medial tibial syndrome and stress fractures. Many patients improve or become symptom-free following thorough education about the importance of these factors and implementation of appropriate changes.

Anterior Leg Pain

The ranges of diagnoses differ between locations. Therefore, classifying symptoms by location, that is anterior (or anterolateral) and posterior (or posteromedial) is helpful in diagnosing the causes of exercise-induced pain in the leg.[3,8] Anterior

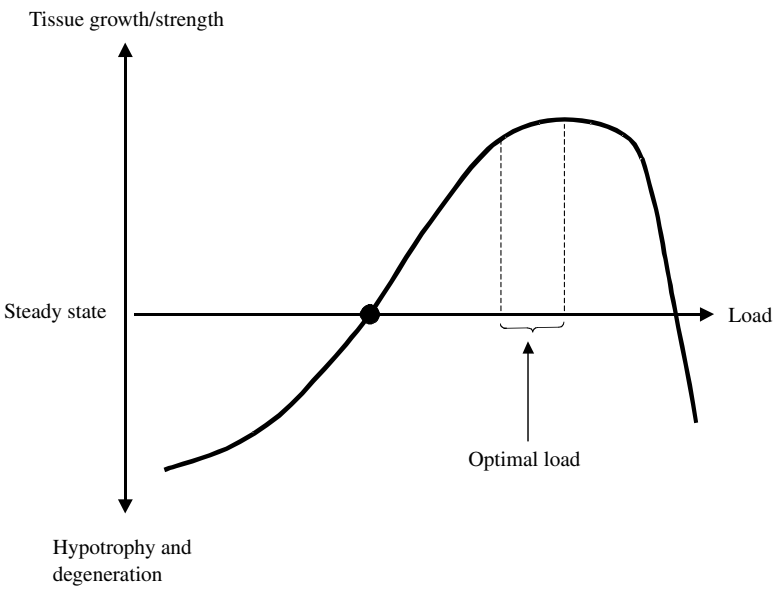

FIGURE 14.1 Relationship between load and tissue growth or strength. Steady-state load (black dot) and optimal load level are indicated on the *x*-axis.

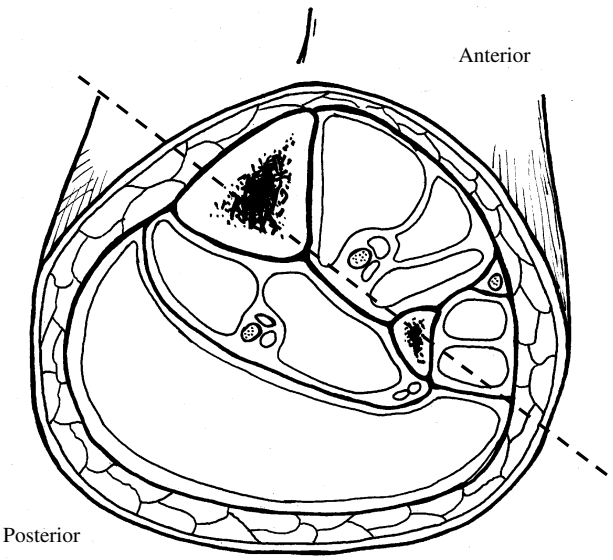

FIGURE 14.2 Cross section of the leg. The dotted line defines anterior and posterior pain locations in the leg. Patient's history and clinical investigation are therefore valuable tools in the diagnosis, because the range of diagnoses differs between locations.

location is defined as all the tissues located anterior to the interosseus membrane and the posterior intermuscular septum (Figure 14.2). Painful conditions in the anterior or anterolateral location are chronic anterior and lateral compartment syndromes, periostitis over the anterior margin of the tibia (periostalgia), peroneal tunnel syndrome, and entrapment of the common peroneal nerve at the fibular head, fascial defects as well as stress fractures of the tibia and fibula. An uncommon reason for anterolateral pain is instability of the proximal syndesmosis.

Posterior Leg Pain

Painful conditions in the posterior or posteromedial location are the medial tibial syndrome, chronic muscle strain injuries, accessory soleus muscle, muscular hypertension syndrome, and stress fractures of the posteromedial margin of the tibia. Entrapment of the popliteal artery and entrapment of the tibial, sural, and saphenous nerves are less common conditions. Pain due to venous diseases and conditions of referred leg pain must be excluded.

Skeletal Muscle Pain

Myelinated or unmyelinated nerve fibers may mediate pain in the skeletal muscle. Muscle pain induced by exercise may be due to muscle fatigue in normal persons, vascular insufficiency, or metabolic myopathies. Other reasons include ischemic mononeuropathy, venous dysfunction, and muscular cramps. The muscle pain may be mechanical, inflammatory, ischemic, or unknown in origin.

Mechanical Pain

Mechanical distension of the muscle fascial envelops and other fascial structures may be painful. One example of this is traction periostitis (periostalgia) seen in some of the patients with chronic compartment syndrome and in patients with medial tibial syndrome.

Inflammatory Pain

Patients with myopathies and rhabdomyolysis have constant pain and clinical findings of tenderness, swelling, and elevated creatinine phosphokinase. Eccentric muscle activity may also induce acute muscle pain. If this is associated with chronic pain due to partial muscle tears, the diagnosis may be very difficult.

Ischemic Pain

Ischemic pain subsides when circulation is restored, such as in chronic compartment syndrome, intermittent claudication, and entrapment of the popliteal artery.

Miscellaneous

Calf muscle cramps induced by exercise are a common complaint in athletes. They are triggered by contraction of a susceptible muscle. They are terminated by passive stretching (or elongation) of the cramping muscle. The cramps are painful and may result in muscle soreness, swelling, and impaired muscle function. The risks for muscle cramping are increased in patients with partial and total muscle rupture.

Growing pain is a deep aching that may affect legs and thighs in children. It may occur in up to 30% of all children.[9] It may be associated with recurrent headaches and abdominal pain. Restless legs are symptomatic in the evening and during the early hours of the night. The symptoms are relieved by muscular activity. Patients with venous dysfunction may experience symptoms of restless or "heavy" legs. The cause of referred pain is not in the muscle.

Patients with primary fibromyalgia have tender spots (or trigger points) in muscles, ligaments, and tendon insertions. Leg pain is seldom the main complaint in these patients.

ANTEROLATERAL LEG PAIN

Anterior Periostitis (Periostalgia)

Periostitis over the anterior margin of the tibia is not a disease but a common clinical finding of soreness possibly due to an inflammatory reaction. This sign is also used to describe those conditions that are not obvious muscle strains, myositis, tendinitis, compartment syndromes, or stress fractures.[10–12] Patients with stress fractures over the anterior margin of the tibia or chronic anterior compartment syndrome are also painful at palpation.

Etiology

Pain over the anterior margin of the tibia may occur in patients with stress fractures, myocitis, and tendinitis. Athletes are prone to get this condition when they change the running surface and when they increase the intensity level of their exercise. Periostitis is considered as an overuse injury. It has also been described as a traction periostitis.

Symptoms and Signs

The pain and tenderness at palpation is located over the anterior margin of the tibia (Figure 14.3). Periostitis occurs in about 40% of patients with anterior pain in their legs, and from 30 to 50% of patients with chronic anterior compartment syndrome.[13] This sign alone is therefore of limited value in diagnosing the causes of leg pain.

Treatment

Antiinflammatory drugs and different methods of physiotherapy have been described. Periostitis not responding to nonsurgical treatment may be treated by fasciotomy of the anterior compartment in a similar way as chronic anterior com-

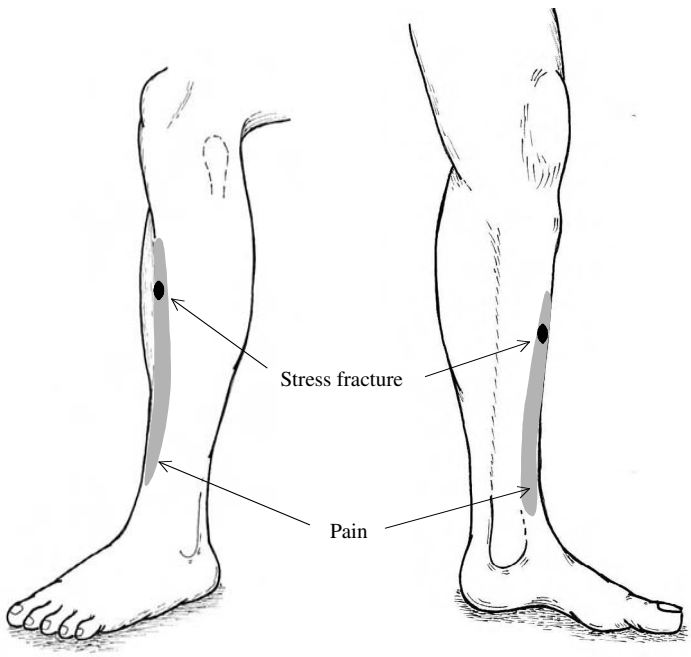

FIGURE 14.3 Location of pain (light grey area) in patients with periostitis of the anterior margin of the tibia. Patients with stress fracture (black dot) over the anterior margin have more localized pain.

partment syndrome. The results of surgical treatment are inferior to those of chronic anterior compartment syndrome.

Entrapment of the Common Peroneal Nerve

The common peroneal nerve may be entrapped at the neck of the fibula due to trauma or following surgical procedures around the knee as well as the proximal part of the lower limb.[14] Entrapment of the nerve may be a more common entity than previously recognized. It should be considered in the differential diagnosis of exertional lateral leg pain.[15] Etiology is idiopathic in most cases.[16] Saal et al. described 20 patients with entrapment of the peroneal nerve proximal to the knee joint.[1] Patients with increased sagital motion of the fibular head due to rupture of the proximal syndesmosis may experience pain and sensory dysfunction elicited by the nerve (personal observation).

Symptoms and Signs

Leg symptoms from entrapment of the common peroneal nerve and chronic anterior compartment syndrome can be quite similar. Patients complain of pain over the anterolateral part of the leg and of dysesthesia, hypoesthesia, or anesthesia. They experience foot drop or extensor weakness of the foot. The pain may radiate up into the thigh. Reduced motor conduction velocity of the common peroneal nerve caused by inversion damage was shown in 118 soldiers.[17] At clinical investigation, patients have a positive Tinel's sign at the fibular neck. The diagnosis is made on the basis of a combination of clinical and electrophysiological findings.

Treatment

If conservative treatment fails, the nerve may be decompressed by division of the tendinous arch of the peroneus longus muscle.[14,15] Surgical treatment should be considered after 2 months if the patient does not recover or after 4 months if recovery is slow.[14] A thick fibrous arch and narrowing of the tunnel through which the nerve passes is seen during surgery. Postoperative recovery of motor function is good in 87% of patients who had sensory and motor dysfunction before treatment. Poor results are seen in patients who were operated on with sensory dysfunction only.[16] This is also true for patients with polyneuropathy.

Peroneal Tunnel Syndrome

Entrapment of the superficial peroneal nerve is a difficult diagnosis to establish because patients often have no abnormalities on testing the neurological status at rest. Half of the patients with peroneal nerve entrapment have a peroneal tunnel syndrome.[18] A recent report suggests that the condition is a more common cause of anterolateral pain than reflected by the literature.[19] Clinical investigation following an exercise test that elicits the pain and the clinical signs is most useful. The fourth toe flexion sign can be used to identify the subcutaneous course of the nerve at the ankle joint.[20]

Etiology

Several reasons for entrapment of the superficial peroneal nerve have been reported. These include fascial defect and muscle herniation,[21,22] lipoma,[23] prolonged peroneal tunnel,[19,24] ankle sprain,[25] following fasciotomy of the anterior compartment in patients with chronic anterior compartment syndrome,[3,8,13] and an anomalous course of the nerve.[19] All these etiologies can cause compression of the nerve at the site where it emerges from the lateral compartment.

Symptoms and Signs

Patients have pain over the anterolateral part of the leg (Figure 14.4). The pain may radiate up to the knee. Lost or disturbed sensitivity over the dorsum of the foot, including the second to fourth toes, is a common symptom and sign of entrapment of the nerve. Patients may also experience pain and tingling at rest when the foot and ankle are held in specific positions, such as keeping the foot on a pedal while driving a car. If this is the case, this position should be used when nerve conduction studies are performed. Pain at rest is a useful symptom in diagnosing the causes, because pain never starts at rest in patients with chronic anterior or lateral compartment syndrome.

About 50% of patients with nerve compression have a fascial defect at the spot where the nerve emerges from the lateral compartment. This is seldom a sign of

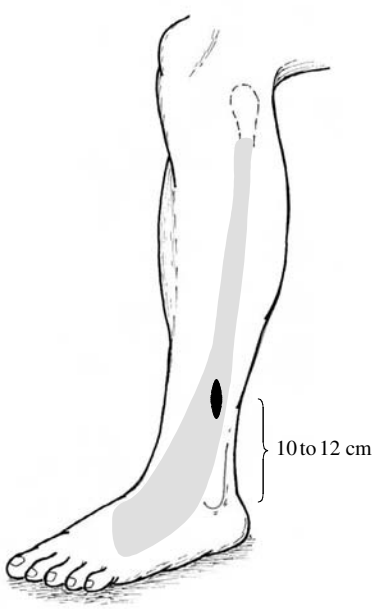

FIGURE 14.4 Location of pain and dysesthesia in patients with entrapment of the superficial peroneal nerve. The pain may radiate up in the thigh and down to the dorsum of the foot. The common site for entrapment is 10 to 12 cm proximal of the lateral malleolus.

compartment syndrome but more often a sign of nerve compression.[3] Findings from three tests are helpful in diagnosis: (1) Pain at palpation over the anterior intermuscular septum 8 to 15 cm proximal of the lateral malleolus while the patient is performing an active dorsiflexion and eversion of the ankle joint is a sign of nerve compression.[19] (2) A positive Tinel's test may also be elicited when the ankle joint is passively flexed and supinated. (3) Tinel's sign rises radiating pain by local percussion over the compression site.[26] Palpation over the nerve must be performed gently, and results of palpation must be compared to the contralateral side. As all peripheral nerves, the superficial peroneal nerve is sensitive to pressure. Other findings include swelling over the anterolateral distal part of the lower leg and decreased or even lost sensitivity over the dorsum of the foot.[19,25] Fifty percent of the patients have been reported to have normal neurographic findings when investigated at rest.[19]

Treatment

Decompression by local fasciotomy[22,24,25] and fasciotomy of the lateral compartment in patients with a simultaneous chronic lateral compartment syndrome[19] have been reported to give good results in 50 to 75% of the patients. Treatment by local fasciectomy or decompression of the peroneus tunnel without complete fasciotomy of the lateral compartment (Figure 14.5A and B) is an alternative method of treatment in patients who do not have a chronic lateral compartment syndrome.[18]

Fascial Defects

Fascial defects occur in 20 to 60% of patients with chronic anterior compartment syndrome[8,27] and in about 5% of patients with anterior pain for other reasons.[8,28]

Etiology

Fascial defects may follow soft tissue trauma to the legs and after tibial and fibular fracture.[8] The pain generating mechanism may be an edge effect from the firm fascial structure on the herniating muscle tissue.

Symptoms and Signs

Patients experience pain over the fascial defect. In the standing patient, a well-localized bulging of subcutaneous tissues is seen, which is similar to that in patients with superficial venous insufficiency.

Treatment

Symptomatic fascial defects should be treated with fasciotomy of the compartment. The fascial incision is performed through the fascial defect. Closure of the defect is never indicated, because it decreases the compartment size and may precipitate an acute compartment syndrome.[27,29,30]

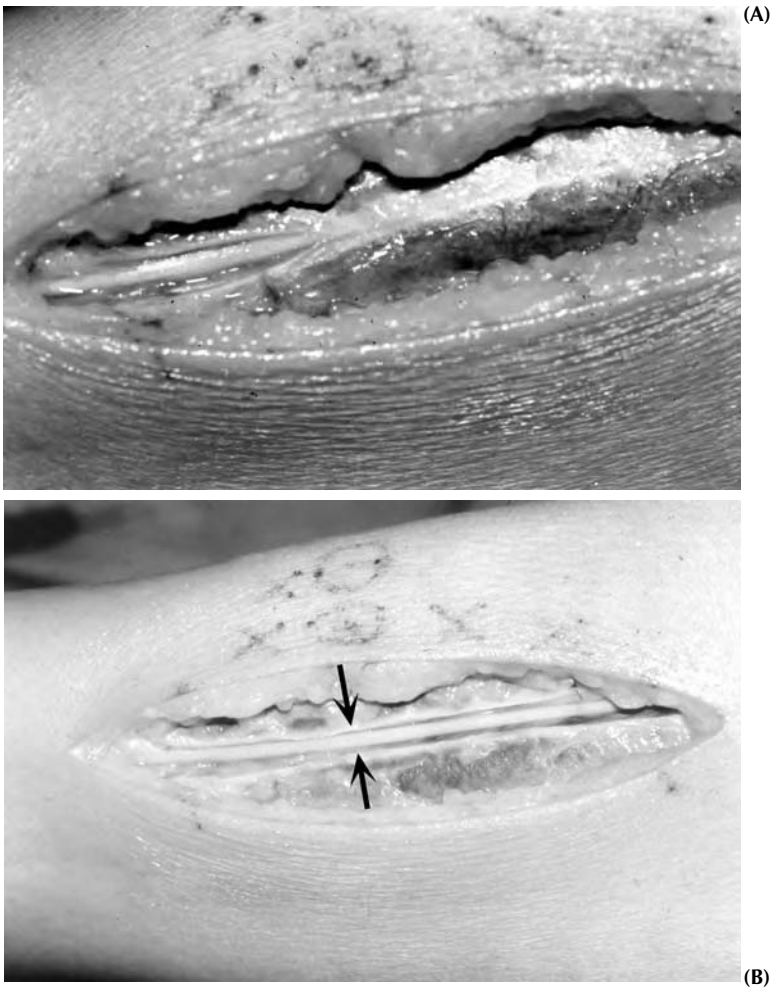

FIGURE 14.5 (A) Intraoperative picture of partly decompressed peroneal nerve in a tunnel. (B) Complete decompression of the superficial peroneal nerve. Arrows indicate local narrowing of the nerve.

Stress Fracture of the Anterior Margin of the Tibia

Stress fractures in the leg comprise about 10% of all overload injuries in athletes. Between 30% and 50% of all stress fractures in runners are in the tibia. Stress fractures are 3 to 12 times more common in women than men. The increased risk for females is explained by greater tension strain and strain rates in response to muscle fatigue.[31] Stress fractures have been reported in about 30% of U.S. military recruits in a 14-week period.[32] The vertical ground reaction force during running may be 3 to 5 times higher, and up to 10 times higher during jumping.[33,34] These forces are absorbed 50 to 70 times/min in the running athlete. Anterior stress fractures of the tibia were first described in ballet dancers in 1956.[35] These fractures are prone to delayed union and nonunion.

Stress fracture of the tibia is defined as a fracture of the bone due to its inability to withstand nonviolent stress that is applied in a repeated subthreshold manner. Fatigue fractures occur when abnormal stress is applied to normal bone. Insufficiency fractures occur when normal stress is applied to a weak bone.

Etiology

The causes of stress fracture may be divided into extrinsic and intrinsic factors. Extrinsic factors are excessive body weight, poor equipment, and training errors. Training errors may include rapid mileage increase, excessive training, and changes of surface and equipment. Inexperienced runners sustain more injuries than experienced runners do. Ekenman et al. found no intrinsic factor to be associated with stress fracture of the tibia.[36]

The etiology of stress fracture is an overload. Predictive factors are long running distances of more than 60 to 70 km per week.[37] Fifty percent of these patients run on a hard surface. Differences in muscular anatomy may be important. Athletes with a Type A behavior and exercise dependency have been related to tibial fractures.[38] Athletes with a Type A personality often underestimate an experienced effort and they may deny muscle fatigue. These persons are overrepresented among athletes with stress fractures.[38]

Motivation, ambitiousness, and competitiveness are parts of most Type A inventories. Athletes who are scoring high on these inventories must be considered to belong to a high-risk population for stress fractures. Leg-length discrepancy, muscular imbalance, joint laxity, presence of a cavus foot, overpronation, and Type A personality have been suggested to be associated with overuse injuries.

Symptoms and Signs

Patients with anterior stress fracture show a slow onset of symptoms, which are exercise related. They experience a diffuse dull pain in the leg, increasing with physical activity. They have local pain over the anterior margin of the tibia at palpation (Figure 14.3). On the lateral view of a radiograph, a V-shaped cut into the cortical bone and cortical hypertrophy is visible. Bone scintigraphy is more sensitive than radiography for diagnosis of stress fracture. In some cases, lesions are asymptomatic.[39]

Treatment

Nonsurgical treatment includes restricted running activities. Athletes with stress fractures over the anterior margin of the tibia heal slowly despite long immobilization periods.

In a study, surgical treatment was required in 4 out of 11 patients.[40] Various operative techniques include plating, drilling,[41] and intramedullary nailing.[42] Surgical treatment by excision and bone grafting is an alternative treatment after 4 to 6 months of failed closed treatment. Intraoperative biopsy is recommended.

Stress Fractures of the Fibula

Stress fractures of the fibula occur in the distal third of the bone. Proximal fibular fractures are uncommon. The fracture is seen in runners, football players, and long jumpers. Running on hard surfaces is more risky than running on soft surfaces.

Symptoms and Signs

Many patients do not experience a particular moment of onset. Symptoms come gradually. Patients report pain to palpation over fibula, local swelling, and percussion tenderness. Most fractures are located a few centimeters proximal to the distal syndesmosis. Radiographic findings are positive in most patients after 4 weeks of symptoms.[43] Scintigraphy establishes the diagnosis earlier.

Treatment

Athletes heal within 6 weeks if the activity level is adjusted. Elastic support or orthotic application may be used. Below-knee plaster is not recommended.[43] Athletes are allowed to put full weight on the leg. Activity is increased gradually as long as pain does not recur. Stretching exercises and muscular strengthening should be encouraged during the healing period.

Tendinosis of Extensor Tendons

Painful areas of tendons are usually diagnosed as tendinitis, although histological studies have not shown characteristic signs of inflammatory response. The histological pattern is more characteristic of a degenerative condition. Therefore, tendinosis may be a better and more commonly used word. In the acute phase, an inflammatory reaction may give crepitations of the anterior tibialis tendon. Pain is elicited during passive and active movement of the ankle joint. Treatment includes adjusted physical activity level.

Instability of the Proximal Syndesmosis

The condition is unusual. All patients have trauma to the leg or knee. They experience anterolateral pain in the leg that may radiate up into the thigh (Figure 14.6). Clinical investigation reveals that the proximal syndesmosis is unstable. Patients may have neurogenic pain due to irritation to the common peroneal nerve, which passes around the neck of the fibula.

If physical therapy is unsuccessful, surgical stabilization of the syndesmosis is indicated. Surgery is also indicated if patients with large sagital instability evolve dysfunction of the common peroneal nerve with weakness of the foot extensors.

POSTERIOR LEG PAIN

Painful conditions in the posterior or posteromedial location of the leg include medial tibial syndrome, chronic muscle strain injuries, accessory soleus muscle, muscular hypertension syndrome, and stress fracture of the posteromedial margin of the tibia. Entrapment of the popliteal artery and compression of the tibial, saphenous, and sural nerves are less common conditions. Venous diseases of the leg are very common and occur simultaneously with other painful conditions in the leg.

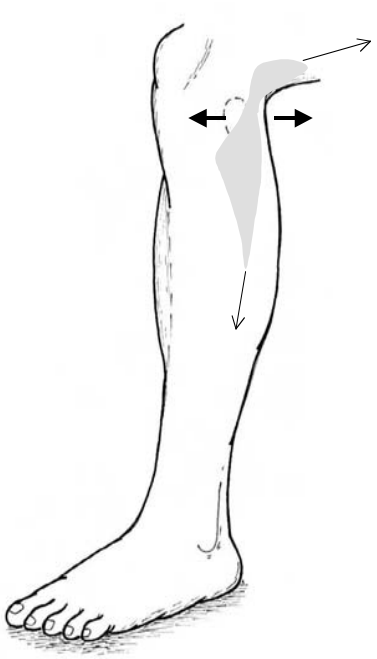

FIGURE 14.6 Pain location of patients with instability of the proximal syndesmosis. Arrows indicate the abnormal sagital motion of the syndesmosis. The pain may radiate proximally and distally.

Medial Tibial Syndrome

The medial tibial stress syndrome is defined as an exercise-induced pain over the middle or distal thirds, or both, of the posteromedial margin of the tibia. It is the most common reason for posteromedial pain in the leg. It occurs simultaneously in about 15% of patients with chronic anterior compartment syndrome and in 25% of the patients with chronic anterior leg pain for other reasons.[8,13] The condition may cover several possible pathological conditions, which all affect the posteromedial margin of the tibia.

Painful conditions that affect the posteromedial tissues of the leg may include (1) tibial microstress fracture, (2) chronic periostalgia due to tension at the junction between fascia and periost, (3) chronic compartment syndrome of the deep posterior compartment,[44,45] and (4) tendinitis and tendovaginitis.

Etiology

The syndrome is not a disease but rather a painful clinical condition without either distinct tissue pathology or pathogenesis. It has been regarded as a periostitis in clinical studies. There are several theories about etiology of the syndrome. Histological studies have indicated that the tissues have an inflammatory reaction at the periosteal attachment and that the bone changes are consistent with microstress

fractures.[46] Increased bone activity with periosteal new bone formation and osteoblastic activity has been shown.[46]

Reneman considered that the symptoms of shin splints could constitute a mild form of chronic compartment syndrome.[47] Some have suggested that the syndrome is caused by abnormally increased intramuscular pressure in any of the posterior compartments of the leg,[48–51] whereas other studies have reported normal intramuscular pressure increase during exercise and at rest after exercise in these patients.[8,13,52–54] Melberg and Styf measured intramuscular pressure simultaneously both in the flexor digitorum muscle and the tibialis posterior muscles in 28 patients with strictly exercise-induced medial tibial syndrome.[53] None of these patients had abnormally elevated intramuscular pressure during exercise or at rest after exercise, indicating a compartment syndrome.

Other etiologies include a short tibialis posterior tendon.[55] Tight muscle tendons are more susceptible to strain.[56] Saxena et al. showed that the tibialis posterior muscle extended much more distally than previously described.[55] It is therefore likely that contracture of the tibialis posterior muscle may cause and maintain a secondary contracture of the flexor digitorum longus muscle by virtue of their crossing. On the other hand, anatomical dissections demonstrate that the sites of tenderness to direct palpation over the posteromedial border of the tibia and increased activity on bone scan correlate better with the soleus bridge overlying the deep posterior compartment than the posterior tibialis muscle.[57]

The soleus muscle is probably the major contributor to possible traction, which may induce medial tibial syndrome.[58] It has been found that both the soleus and the flexor digitorum longus muscles arise on the medial border of the tibia at the site of symptoms of the media tibial syndrome. Excessive foot pronation is seen in many patients with medial tibial syndrome.[57,59] This has been correlated with forefoot pronation and overuse syndromes in the lower extremity.[60]

Symptoms

Symptoms and signs of medial tibial syndrome are so typical that the diagnosis can most of the time be made by clinical investigation. Patients complain of pain along the middle or distal thirds, or both, of the posteromedial ridge of the tibia (Figure 14.7). The pain may occur soon after the start of exercise. On average, patients with medial tibial syndrome experience leg pain after 200 m of running, compared to 3.5 km (ca. 2 mi) in patients with chronic anterior compartment syndrome. Leg pain in patients with medial tibial syndrome may even be relieved by continued running, whereas it forces the athlete with chronic anterior compartment syndrome to stop running. In some patients, pain starts at rest after exercise and some may have more pain the following day. The pain is less focal than in a patient with a stress fracture. It may vary from dull ache to intense pain.

Clinical Examination

At investigation, the tenderness is located in the middle third or the middle and distal thirds of the posteromedial margin. Often the most painful area is centered on the

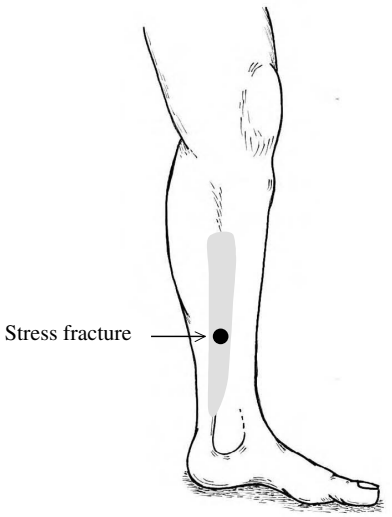

FIGURE 14.7 Pain location of patients with medial tibial syndrome. The dot indicates the location of the stress fracture. The light gray area shows localized pain and swelling of patients with a stress fracture of the postromedial margin of the tibia.

junction of the middle and distal thirds. The pain is aggravated on active plantarflexion and supination of the ankle joint against resistance. No sensory, motor, or vascular abnormalities have been reported. Many of these patients have excessively pronated feet.

Plain x-ray films are normal or may show diaphyseal cortical hypertrophy. Scintigraphy may show a diffuse and varied intensity uptake along the length of the posteromedial margin of the tibia.[57] The uptake on the positive bone scan is quite diffuse and different from that of a stress fracture.[61,62] There is a moderate increase of radionuclide along about one third of the posteromedial border of the tibia on delayed images. In stress fractures, the length of the increased radionuclide activity along the tibia is much shorter. The scintigraphic pattern can be used to differentiate medial tibial syndrome from stress fractures.[62] Allen and co-workers showed that 75% of patients with medial tibial syndrome and 20% of patients with chronic anterior compartment syndrome had a positive $^{99}Tc^{m}$-MDP bone scan.[61] They concluded that bone scans were a useful diagnostic tool in the differential diagnosis of exercise-induced leg pain.

Treatment

Nonsurgical treatment includes adjusted activity level and alternative activities such as bicycling, swimming, as well as even rest combined with NSAID initially. In severe cases, rest from running is mandatory. Physiotherapy, orthotic applications, and NSAID must be properly prescribed before surgery is considered. Patients who have short muscles are referred to physiotherapy. Short muscles are more susceptible

to strain.[56] This is especially important in patients with pronated feet. Up to 30% of patients may improve by static or dynamic orthotics. If the runner's limb alignment indicates a varus heel, treatment by a medial heel wedge may be indicated.[5] A great deal of art is involved in the management of an athlete with medial tibial syndrome. It is important to teach patients to listen to their legs and adopt realistic goals and timetables. Proper advice regarding running technique, shoes, and surfaces is a challenging pedagogic duty.

Surgical treatment is not the mainstream treatment. Nonsurgical treatment is recommended for at least 6 months because the prognosis by conservative treatment is good. At surgery, the author follows the principles outlined by Michael and Holder.[57] The operation is performed in a bloodless field with a thigh tourniquet. The risk for damaging nerves and veins is much more pronounced than in surgery on the anterior compartment. Therefore, the author recommends the use of an at least 10-cm-(4 in.)-long skin incision, about 3 cm (1 to 1.5 in.) behind the posteromedial margin of the tibia. The veins and the saphenous nerve branches are elevated anteriorly. In many patients, the attachment of the soleus muscle to the tibia (or soleus bridge) must be detached to allow for fasciotomy of the deep posterior compartment. The tourniquet is released and the homeostasis is controlled carefully before the wound is closed by intracutaneous sutures. Surgery relieves the tension of the fascial insertion on the tibial bone and increases compartment volume. Results of surgery are reported as good or excellent, between 30 and 80%.[44,63,64]

Muscle Strain Injury (Tennis Leg)

Strain injuries to muscle are common. They may occur during ordinary running, but more often during a powerful eccentric muscular contraction.[56] The condition is known as tennis leg and may cause a bothering disability.

Etiology

The painful condition is caused by a strain injury to muscle. It may include rupture of fibers of the medial head of the gastrocnemius muscle. These muscle fibers are fast twitch as opposed to the soleus muscle fibers, which are primarily slow twitch. Muscle strain injury often occurs at the myotendinous junction unless the muscle has suffered previous compression injury leading to failure within the muscle.[65]

The gastrocnemius muscle is more vulnerable because it passes by both the knee and ankle joint. The mechanism of injury includes overstretching of this muscle by a combination of forced ankle dorsiflexion and knee extension. The same injury mechanism may also create an Achilles tendon rupture. In some patients who have been exposed to repeat muscle strains, the clinical picture may become more complex.

Symptoms and Signs

Patients may report sudden acute calf pain during running. Some of them experience a sudden sound as if someone had kicked them in the calf. Pain and calf swelling develops during the following 24 to 48 h. This history is important to document initially. Repeated sudden onset of acute pain during running is a key

finding in the history that speaks against chronic compartment syndrome in the leg. During this time period, patients may have had repeated strain injuries to the calf muscles. Patients with chronic pain following partial calf muscle ruptures complain of local deep muscular tenderness. They may experience muscle cramps during and after exercise.

At investigation, patients may have a positive Hohman's sign, which is calf pain during passive dorsiflexion of the ankle joint. The pain may also occur during active dorsiflexion. Flexor tendons may be short. Point tenderness is found over the musculotendinous portion of the gastrocnemius muscle bellies. Ultrasonography or MRI can be used to evaluate the extent of muscle injury. Athletes with leg weakness can be tested with a kinetic computerized ergometer.

Treatment

In the acute phase, patients should be treated by a supportive wrap and ice application. Crutches are helpful during the first 3 days, if necessary. A heel lift for 2 to 4 weeks before patients return to full activity is recommended. Weight bearing is slowly increased. Calf muscle stretching and strengthening exercises are important. When muscle strength is 90% of the noninjured side, patients may resume sports activities. Nonsurgical treatment, including stretching and relief of the posterior structures by foot orthotics, is helpful in most patients. Surgery for chronic cases is not in the mainstream of treatment and should be avoided.

Accessory Soleus Muscle

Symptomatic accessory soleus muscle is one of the supranumerous muscle belly syndromes. Patients have an extra soleus muscle belly and a tendon that inserts on the calcaneus or to the Achilles tendon.[66–68] The incidence of an accessory soleus muscle ranges between 0.7 and 5.5%.[69] The accessory soleus muscle can vary in its anatomic configuration. The flexor digitorum longus muscle may also have an accessory muscle belly.[70] In these patients, the tibial nerves were compressed by the muscle.

Pathophysiology may be diminished blood flow leading to ischemic pain.[67,69] Exercise-induced compartment syndrome and compressive neuropathy of the tibial nerve have been suggested.[71] Others have suggested that the pain comes from fascial traction on the periosteum.[68]

Symptoms and Signs

Patients experience burning pain and persistent swelling, which is accentuated during and after exercise over the distal third of the posteromedial part of the leg, including the tarsal tunnel. The pain may radiate distally into the longitudinal arch of the foot. Asymptomatic swelling occurs in about 25% of patients. Intramuscular pressures during exercise and at rest after exercise are increased in these patients, but not to the level seen in chronic compartment syndrome. The condition may be evaluated by ultrasound, CT, or MRI. On MRI imaging, the accessory soleus muscle may insert into the calcaneus in five different ways.[72,73]

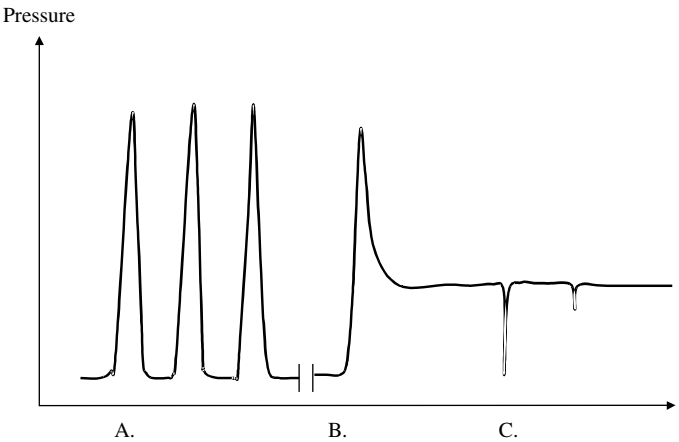

FIGURE 14.8 (A) Patients with muscular hypertension syndrome have normal muscle contraction pressure and muscle relaxation pressure during exercise. (B) At rest after exercise, patients are unable to relax their leg muscles. Therefore, the pressure is abnormally elevated and can be interpreted as chronic compartment syndrome. (C) Short, sometimes repeated, periods of low pressure indicate that the patients are unable to relax their muscles. This condition is not based on a real volume load of the muscle.

Treatment

Fasciotomy has been reported to be successful.[69] Treatment may also include excision of the supranumerous muscle or muscle belly.[66,67] Excision should be reserved for those patients who do not respond to fasciotomy.[69]

Muscular Hypertension Syndrome

This syndrome may easily be judged as a chronic compartment syndrome because intramuscular pressure is abnormally elevated at rest after exercise. However, intramuscular pressures during muscle contraction and muscle relaxation during exercise are normal (Figure 14.8). The patient is unable to relax the leg muscles completely initially at rest after exercise.[13] The condition has also been described as tension myalgia.[74] For example, patients with low-back pain were found to have EMG activity at rest.[75] A rare condition of muscle stiffness and spasm was described in 14 patients over a time period of 32 years.[76]

Symptoms and Signs

Patients with muscular hypertension syndrome have pain during exercise and at rest after exercise. They are not able to relax their calf muscles at rest after exercise. Intramuscular pressure at rest after exercise is elevated because of an active remaining muscle contraction.[13] The EMG signal is positive. When the signal turns silent, the intramuscular pressure also normalizes. Some patients may have muscle pain in other locations, although leg pain is dominating.

Treatment

Treatment by fasciotomy is not helpful. However, stretching and muscle relaxation by biofeedback have been encouraging. Treatment by stockings, which give external compression of 15 to 22 mmHg, is helpful in patients with simultaneous venous insufficiency.

Stress Fractures of the Posteromedial Margin of the Tibia

In athletes, the tibia is the most common site for stress fracture. Tibial fractures are involved in up to 50% of all stress fractures. The overall incidence of stress fractures in an athletic population is 0.12%.[77] In a population of runners, the incidence has been reported to range from 4 to 16%.[41,77] The risk for a tibial stress fracture in women is greater. Stress fractures may be multiple. They include all areas of the tibia. When located in the proximal or distal third of the tibia, they are found on the posteromedial ridge of the bone. These fractures develop slowly, which allows for adequate bone remodeling and hypertrophy.

Etiology

The development of a stress fracture is multifactorial. It is related to the level and intensity, duration, and type of physiological activity, sex, bone size and mineralization, and the athlete's personality. Risk factors include running on hard surfaces, inadequate shoes with poor impact-absorbing quality, and, finally, lower-limb malalignment. Intrinsic factors are forefoot varus, subthalar varus, tibia vara, leg-length inequalities, and pes cavus.[41,77]

Pathogenesis

According to one theory, muscle fatigue and weakness may reduce the relative shock-absorbing capabilities of muscles in the lower extremities. The reason for this is the higher tensile forces in the bone when muscle fatigue occurs.[31] According to another theory, the tight forces across bones generated by rhythmic muscular activity may cause mechanical insult, resulting in fatigue fracture. If repetitive overload continues, the weakened cortex may fracture completely. In early stages of stress fractures, an increased number of erythrocytes are found within bone, resulting in vascular congestion and thrombosis. Subsequently, osteoclastic reposition and periosteal new bone formation by osteoblasts take place.

Symptoms and Signs

Stress fractures of the tibia cause local tenderness and subcutaneous swelling (Figure 14.7). Radiographic examination is positive in 90% of the patients within 1 month. Radiographic examination and bone scans are normal in patients with chronic anterior compartment syndrome.[54]

Treatment

Most tibial stress fractures heal without surgical treatment. Nonsurgical treatment includes restricted running activities.

Entrapment of Popliteal Artery

Entrapment of the popliteal artery caused by anatomic variation of blood vessels and muscles in the popliteal fossa was described by Stuart in 1879. The prevalence of the syndrome has been reported to be 0.17 to 3.5% in cadaver studies. The incidence of popliteal artery entrapment is up to 3.5% in cadaver dissection.[78] Darling et al. believed that the syndrome was commonly overlooked although it is unusual.[79] Absence of the posterior tibial artery has been reported in 0.5 to 2.5% of extremities.[80]

Etiology

Patients with entrapment of the popliteal artery have an abnormal anatomical relationship between the artery and surrounding soft tissues. The artery may be entrapped under the medial gastrocnemius head or have an anomalous course (Figure 14.9). The popliteal vein may follow the anomalous course of the artery.[78] Intimal hyperplasia may occur as a result of endothelial injury. Intramuscular pressure during exercise is not elevated in patients with entrapment of the popliteal artery.[81] The disease is possibly more common than reflected in the literature. However, the patient's symptoms resemble those of chronic compartment syndrome and may be misdiagnosed.[70,82,83]

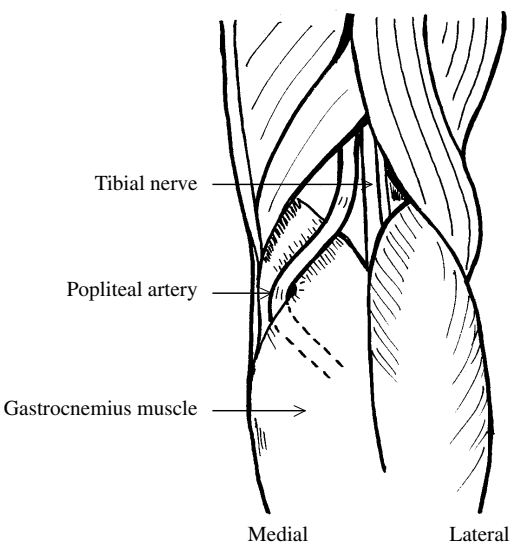

FIGURE 14.9 Entrapment of the popliteal artery. The artery has an anomalous course medial to the medial head of the gastrocnemius muscle. The artery may also run through the medial head of the muscle.

Classification

There are many variations of how the medial head of the gastrocnemius muscle entraps the popliteal artery.[84] Represa et al. classified entrapment of the popliteal artery into three types:[85] (1) *Type 1*. The popliteal artery passes medial to the medial head of the gastrocnemius muscle. This is the most common type (63%). (2) *Type 2*. The popliteal artery cuts across the medial head of gastrocnemius (23%). *Type 3*. The artery passes deep to the popliteal muscle (7%). In the remaining cases, the abnormality was not categorized. Structures compressing the popliteal artery in 296 limbs were described by Stipa et al.[84] Both legs were involved in 20% of cases.[86]

Symptoms and Signs

Patients are symptom-free at rest but the knee may feel warmer. The foot may be cold. Young athletes may have calf cramping after exercise. Many nonathletes may not experience symptoms, possibly because of the low activity level. Others experience progressive calf pain on walking. The typical history is intermittent claudication in the leg and foot. Pain is typically located to the level of the musculotendinous junction in the calf and may extend into the foot.

Results of physical examination with the patient at rest are usually normal. Maximal active dorsiflexion or plantarflexion of the ankle joint decreases muscle blood flow in the leg and diminishes or obliterates pulses in the leg. Measurement of local blood flow by Xenon[133] shows decreased local blood flow. Measurement of muscular oxygenation by near-infrared spectroscopy shows decreased oxygen tension.

Examination with Doppler sonography reveals decreased pulses in the tibialis posterior and dorsal foot arteries in their resting state but more often with passive, or active, dorsiflexion of the ankle joint with the knee hyperextended. Active plantarflexion of the ankle joint and ipsilateral knee hyperextension is another provocative test of the popliteal artery function. Evaluation includes Doppler ultrasonography, either with the continuous wave technique or a color Doppler, treadmill exercise testing, and pulse volume recording. Intramuscular oxygenation decreases. Intramuscular pressures during exercise and at rest after exercise are normal.[81,82] Angiography should be done only prior to surgery. It should not be the first choice. MRI may be an alternative possibility.

Except chronic compartment syndrome, differential diagnosis should include Burger's disease and arteriosclerosis. Other etiologies may be external compression from stockings or knee braces. Also, adductor canal compression may induce leg and foot ischemia. These patients have a history of burning pain and loss or diminished pulses of the dorsalis pedis and posterior tibial artery. [87] Young athletic patients with exercise-induced leg pain, cramping, and neuritic symptoms may thus have a more proximal lesion.

Treatment

The syndrome must be treated by surgery. Results are better if patients were treated early by division of musculotendinous tissues (90% good results). When arterial grafting is required, the long-term patency rate is about 60%.[88] A posterior Z-incision

is made, which allows for a complete exposure of the artery and its surrounding structures. Treatment in the early stages can often be performed by release of the gastrocnemius anomaly. It has been demonstrated during surgery that by plantarflexing the foot, the distal pulses disappear and a fibrous band visibly occludes the popliteal artery. Removal of the band restores normal blood flow to the limb. Other intraoperative findings include the vessels through a common ligamentous and muscular tunnel, which is formed by a wide head of the gastrocnemius muscle.[79] In late cases, the artery may undergo thrombosis or wall deterioration. In these cases, excision of the damaged section and grafting with a saphenous vein graft are indicated.

Entrapment of the Tibialis Nerve

Entrapment of the tibial nerve in the tarsal tunnel is well recognized whereas other compression sites of the nerve are rare. Patients with pseudoradicular pain and sensory disturbances in the foot may have lower extremity peripheral nerve entrapment.[1] The popliteal artery, veins, and the tibial nerve pass through the tendinous arch, which is formed by the soleus and popliteus muscles. Symptoms from nerve compression at this site are very uncommon. The numbness, tingling, pain, and paresthesia seen in patients with entrapment of the popliteal artery have been ascribed to ischemia from arterial compression. However, Saal et al. described nine patients with tibial nerve entrapment in the popliteal space.[1]

Etiology

The nerve may be entrapped at the tendinous arch of the soleus muscle[89] or occur simultaneously in patients with entrapment of the popliteal artery (Figure 14.9).[84] It may also be compressed by the medial head of the gastrocnemius muscle.[84,90,91] The most common site of entrapment at surgery is the hypertrophied medial gastrocnemius muscle. Fibrosis of the posterior knee joint capsule has also been described.[1] The tibial nerve may also be compressed by an accessory flexor digitorum muscle at the tarsal tunnel.[70]

Symptoms and Signs

Patients may experience burning pain that radiates up in the thigh and down into the longitudinal arch of the foot. They have severe calf pain similar to sciatica. They experience decreased sensitivity over the sole of the foot, weakness of the flexor hallucis longus and flexor digitorum muscles, while the Achilles tendon and plantarflexion force is normal. Calf pain is aggravated by active plantarflexion of the ankle joint or passive ankle joint extension. This suggests that the lesion is located in the leg rather than the foot.

At the entrapment site, Tinel's sign and tenderness at palpation are positive in 90% of the patients, whereas other neurologic signs and deficits such as a positive straight leg raising test are found in about 40% of the patients.[1,90] EMG of the flexor muscles show denervation potentials.

Additional diagnostic methods including CT, MRI, and electromagnetic studies are helpful in diagnosing this condition.[89] Nerve conduction velocity is decreased in about 80% of the patients.[1]

Treatment

The nerve is decompressed through a complete exposure.[1,84,92]

Entrapment of the Saphenous Nerve

The saphenous nerve is a sensory branch of the femoral nerve. It passes through the adductor canal in the thigh. The canal is surrounded by the sartorius, vastus medialis, and adductor Magnus muscles. In the leg, the nerve is accompanied by the saphenous vein. Entrapment of saphenous nerve may simulate vascular disease[93] and radiculopathy.[1,94] Furthermore, lower extremity nerve entrapment may mimic lumbar pain syndromes in 50% of the cases. Seventy percent of patients with entrapment of the saphenous nerve at the adductor canal of the thigh have leg pain.[1] The condition may mimic other diseases, which affect the posteromedial part of the leg.

Etiology

Trauma to the adductor canal and vascular surgery are the most common causes for saphenous nerve dysfunction.[95,96] Entrapment under the sartorius muscles in a body-builder and compression by a meniscal cyst have been reported.[95] Traction on the nerve at the site where it exits the adductor canal may cause local inflammation and edema. The nerve may also be compressed by a pes Anserinus bursitis.[97]

Symptoms and Signs

Most patients experience anteromedial knee pain, which may radiate into the medial aspect of the leg following the saphenous nerve distribution (Figure 14.10).[95] They may experience medial leg pain, which is similar to the pain patients with medial tibial syndrome experience. The patient experiences burning pain, which may be aggravated by exercise. The pain may awaken the patient at night. It may simulate intermittent claudication, sensation of fatigue, and heaviness of the leg.[93] The pain may even mimic tears of the medial meniscus and a stress fracture of the tibia.[97]

At clinical examination, Tinel's sign and tenderness at the lesion site are positive in all patients, whereas other neurologic deficits distal to the compression site are found in about 50% of the patients.[1,95] The patient experiences point tenderness at palpation over the site of entrapment or compression. This may include pressure over the adductor canal, which induces radiating pain down to the medial malleolus of the ankle joint. Peripheral nerve block at the adductor canal is useful in differentiating peripheral nerve pain from a spinal source.[1] Electrophysiologic data indicate a side-to-side decrement of SNAP amplitude in 60% of patients.[93]

Treatment

Adjusted physical activity level is often sufficient. It has been suggested that abnormal retrograde nerve impulses may be inhibited by local anesthesia.[98] Saphenous nerve block at the adductor canal by a 10 mL (cc) solution of bupivacaine

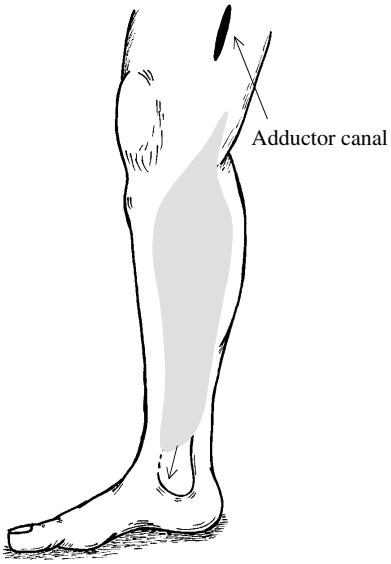

FIGURE 14.10 Location of pain and sensory dysfunction of patients with entrapment of the saphenous nerve.

(0.25%) and 25 mg of triamcinolone diacetate in a 9:1 ratio had a favorable outcome in 80% of patients. Seven percent of patients had increased pain after this treatment.[95] In another study, only 38% of patients reported favorably by corticosteroid treatment.[93] The length of symptoms before treatment increased the risks for increased pain intensity at follow up. Treatment by partial release of the sartorius muscle has been suggested. Saal and co-workers recommended treatment by neurolysis at the site of compression between the vastus medialis and adductor Magnus muscles.[1]

Entrapment of the Common Suralis Nerves

The anatomical course of the sural nerve may vary. The lateral sural nerve pierces the fascia in the popliteal fossa dorsal to the fibular head. It may be entrapped anywhere along their course. The common sural nerve runs between the heads of the gastrocnemius muscles and pierces the fascia in the middle third of the leg. It runs close to the Achilles tendon and within a couple of centimeters posterior and inferior to the lateral malleolus.

Etiology

It may follow recurrent ankle sprains and calcaneus fractures,[99] ganglion from the peroneal tendons in the distal part of the leg, as well as other types of trauma to the nerve along its course. Dorsal extension and inversion of the ankle joint may lead to a traction injury to the nerve. Other etiologies include Baker's cysts,[100] external compression,[101] and trauma.[102]

Symptoms and Signs

Patients have a subacute onset of a neuritic type of pain, including burning, tingling, or radiation. They experience diminished sensation with or without paresthesia along the distribution of the sural nerve in the leg and the foot.[103] Pain may radiate proximally into the calf.

Local tenderness and a positive Tinel's sign along the entrapment site of the nerve are found at clinical investigation. Patients with entrapment of the sural nerve may have pain, tenderness, and swelling behind the lateral malleolus. Pressure over the entrapped nerve may cause discomfort along the lateral border of the foot.[103]

Treatment

Decompression by removal of Baker's cysts may help recovery. Local fasciectomy or fasciotomy at the site where the nerve emerges the fascia may help. Alternative exercises, injections of local anesthetics, massage, nonsteroid antiinflammatory drugs, and shoe modifications have been tried.

VENOUS DISEASES OF THE LEG

Return of venous blood from the lower extremity to the heart is maintained by a series of interconnected venous muscle pumps from the foot to the thigh.[104,105] During weight bearing and muscular action, blood is expelled from the calf veins into the thigh veins. This action depends on competent valves in the veins. More than 90% of swollen legs seen in general practice are due to incompetence of the venous system.[106] Most people have only mild or no symptoms. Deep venous insufficiency may follow deep venous thrombosis in 70% of the patients.

Venous Insufficiency

Many patients with calf pain have venous insufficiency. Advanced cases of deep venous insufficiency are often referred to as a postthrombotic syndrome. The main pathophysiological factor in these patients is valvular dysfunction or venous dilatation in the deep veins. Intravenous pressure in the foot may be up to 100 mmHg. It decreases to below 30 mmHg following activation of leg muscles. Competent venous valves are a prerequisite for the decrease of intravenous and intramuscular pressures.[105,107] The total leg volume is increased in limbs with venous insufficiency. The filling speed increases significantly. Doppler sonography and plethysmography are helpful investigations to reach diagnosis.

Symptoms and Signs

Patients complain of pain and ache. The legs may feel heavy and they may be restless at night. At physical examination the legs may be swollen and show marked skin changes such as hyperpigmentation. Elderly patients may develop ulcers. At physical examination, patients experience a calf tenderness and a positive Hohman's sign. Patients experience diffuse aching in the legs, which may be worse after standing.

The legs hurt at night. Patients may experience calf pain and muscular cramps. They have more pain at rest after exercise than they experience during exercise. Intramuscular pressures during exercise and at rest after exercise are elevated, but not to the level seen in patients with chronic compartment syndrome.

Treatment

Compressive stockings are the method of choice initially. Surgery for patients who suffer from both medial tibial syndrome and venous insufficiency should not be the mainstream, because venous rheology may become considerably impaired following fasciotomy, and the risks for an inferior surgical outcome are increased. Low-grade compression stockings, which generate up to 15 to 22 mmHg of pressure are sufficient for most patients. This treatment enhances the calf muscle pump leading to decreased venous pressure. Local excision of varicose veins combined with postoperative compression stockings for extensive time periods is helpful.

Varicose Veins

Patients have diffuse leg aching, which may worsen after standing. They complain that their legs hurt or are restless when they go to bed at night. Pain is not localized to any specific muscular compartment. Patients repeatedly treated by local extirpations and vein stripping may develop elevated intramuscular pressure during exercise and at rest after exercise. Only rarely are chronic compartment syndromes seen in these patients. The condition is treated with use of compressive stockings.

Venous Claudication

Patients experience calf pain while walking. The condition is sometimes difficult to differentiate from intermittent claudication due to arterial insufficiency. Venous claudication may follow thrombosis or incompetent valves of the deep veins.

Thrombophlebitis

Thrombophlebitis is a common cause of leg pain due to low-intensity exercise. The leg may be edematous. The diagnosis is documented by venography. Patients experience leg tension at speedy walk, due to high intravenous pressure. Treatment includes anticoagulants for 3 months. The patient should be referred to a specialist for additional investigation of risk factors.

Effort-Induced Venous Thrombosis

The condition is uncommon. It has been associated with extensive muscular activity in runners,[108] kick boxers,[109] and in football players.[110] It has been seen in patients with underlying anatomical abnormality.[111] The patient experiences sudden onset of leg pain and swelling, similar to the symptoms of patients with partial rupture of the gastrocnemius muscle. Clinical findings include a positive Hohman's sign and point tenderness over the calf. The diagnosis is confirmed by ultrasonography or by phlebography. The condition is treated by anticoagulation therapy.

MISCELLANEOUS

Several conditions such as inflammatory reactions, lumbar disc herniation, pain after fractures of the tibia, intramuscular lipoma in the lateral compartment, and supranumerous muscle belly syndromes may mimic the symptoms of chronic anterior compartment syndrome in the leg. Exercise including eccentric muscle contractions, e.g., downhill running, causes the greatest degree of delayed muscle soreness with pain, swelling, and tenderness (Armstrong, 1984; Fridén, 1984). Runners in this situation may even have increased intramuscular pressure at rest up to 24 h following exercise. Eccentric exercise increases water content by 3% more than concentric exercise and results in a higher intramuscular pressure at rest after exercise (Fridén et al., 1988). The contractile apparatus can be damaged by eccentric exercise (Armstrong, 1984; Fridén, 1984). Repeated episodes of muscle soreness, as a reason for chronic pain in the leg, have not been established. Increased local muscle spasms, as verified by quantitative EMG recordings, may be one reason for chronic muscle pain (DeVries, 1966).

Combined Symptoms

Each tissue of the leg, including bone, periost, muscle, fascia, tendon, ligament, arteries, veins, and nerves, may be a source of pain. All the tissues may participate in the generation of symptoms of the painful leg simultaneously in patients with overuse injury. In some patients, several tissues may simultaneously contribute to pain generation. The medial tibial stress syndrome may sometimes occur with deep venous insufficiency or with chronic anterior compartment syndrome. Chronic anterior compartment syndrome may occur with the peroneus tunnel syndrome. Achilles tendinitis is a common overuse injury causing posterior leg pain. It may occur simultaneously with other conditions giving posteromedial leg pain.

SUMMARY

Leg pain in athletes has many etiologies. Each of the leg tissues may be a source of pain. The ranges of diagnoses differ between location. Therefore, classifying symptoms according to pain location, anterior and posterior, is helpful. A careful history including pain analysis is important. Pain induced strictly by exercise is indicative of chronic compartment syndrome. Pain with multiple locations in the leg that starts soon after exercise and allows the athlete to continue running despite pain does not indicate the syndrome. Painful conditions that cause anterior leg pain include chronic anterior compartment syndrome, anterior periostitis, entrapment of the peroneal nerves, fascial defects, stress fractures of the anterior margin of the tibia, extensor tendinosis, and instability of the proximal syndesmosis. Painful conditions that cause posterior leg pain include medial tibial syndrome, tennis leg, accessory soleus muscle, muscular hypertension syndrome, entrapment of the popliteal artery, tibialis, saphenous and suralis nerves, as well as venous diseases.

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15 Composition and Function of the Interstitial Space

INTRODUCTION

The interstitial space includes the connective tissues that intervene among cells, blood capillaries, and lymph vessels. The volume of the space is about 15% of body weight.[1] It is also an extravascular fluid system that is generated by capillary filtration (Figure 15.1). The extravascular flow system functions to support the tissue's metabolic demands and to maintain an optimal and stable environment for the cells. This is the space in which hydrostatic pressures are measured for clinical purposes and for research to determine limits for tissue nutrition and viability.

Muscle and Tendon

Skeletal muscles comprise approximately 40% of the body weight. The volume of interstitial space varies among tissues. It is 5% of the muscle tissue volume whereas it is 95% of the volume in the tendon. Muscles may increase metabolism 50-fold. Muscle tissue may increase blood flow 100-fold during activity compared to at rest. Skeletal muscle is the engine of locomotion, posture, and respiration. Muscular tension increases the strength of the long bones of our limbs by pretentioning. Skeletal muscle protects the viscera, participates in heat production, and is storage for many important nutrients, electrolytes, and divalent ions.

Normal function of muscle tissue requires normal function of muscle fibers, nerve, vasculature, and tendon. The study of these functions is labeled muscle physiology. Normal function of muscle tissue also requires a mechanical load. Muscular biomechanics is the study of the response of the muscle to mechanical loading. Therefore, muscular biomechanics is the study of how force is transferred from muscle fiber through the interstitial space to the tendon and its insertion. It also includes studies on muscular geometry, muscular stress, and intramuscular pressure generation.

COMPOSITION OF THE INTERSTITIAL SPACE

The interstitial space is composed of a framework of fibers (matrix) and an amorphous mucopolysaccharide gel (Figure 15.2). The fibers may extend long distances through the interstitium. They are strong and carry most of the compressive and tensile forces of the interstitial space of all tissues. The fibers may be collagenous,

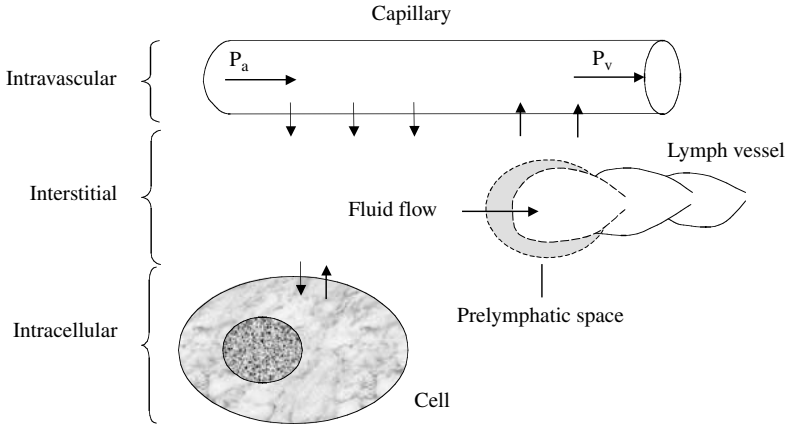

FIGURE 15.1 Schematic illustration of tissue spaces. The fluid flow to and through the interstitial space is indicated by solid arrows.

reticular, or elastic. They are synthesized by mesenchymal cells and fibroblasts. Molecular collagen is secreted from the cells into the interstitial ground substance, where it is polymerized to collagenous and reticular fibers.[2] The gel consists of two interpenetrated phases, one a solid-like phase arranged in the form of a network whose strands are linked together. The solid-like phase consists of thin, coiled proteoglycan filaments. They look like brushes and are composed of 98% hyaluronic acid and 2% protein. The second phase is the space between the solid filaments, which is filled with entrapped fluid. The gel is thus a conglomerate of small filaments of small proteoglycan and hyaluronic acid fibers in which most of the water is entrapped (Figure 15.3). This prevents, or at least resists, fluid shift from upper parts to dependent parts of the body. An additional function of the gel is to keep adjacent cells partially separated from each other.[3] The interstitial space also contains non-

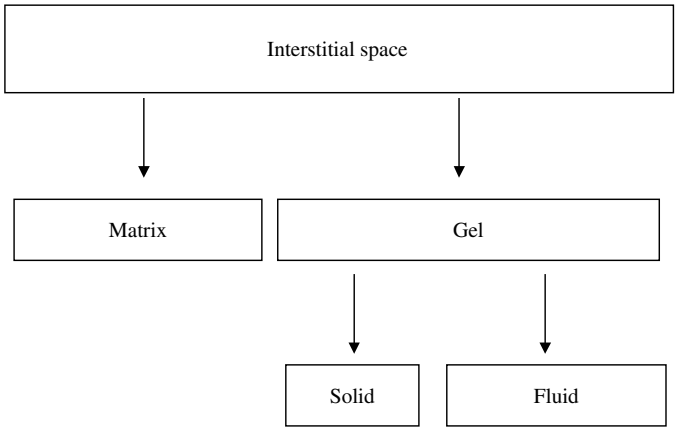

FIGURE 15.2 The interstitial gel is composed of solid components with entrapped fluid.

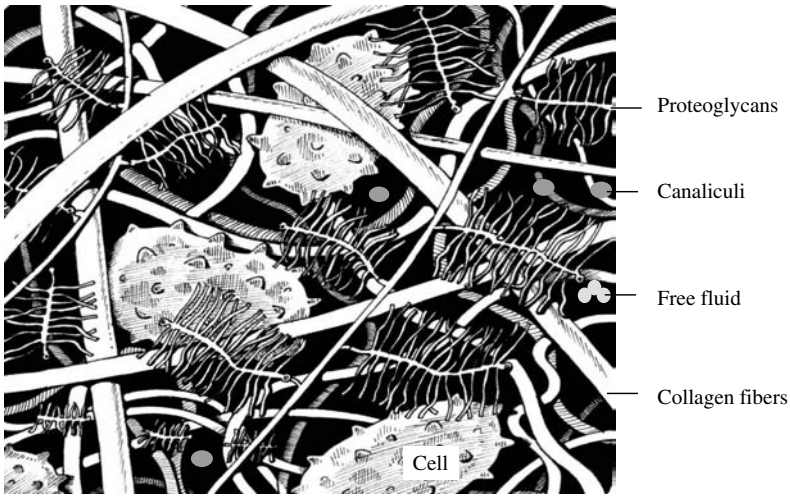

FIGURE 15.3 Components of the interstitial space. The background represents the gel and interstitial fluid.

endothelialized microscopic channels ending in the prelymphatic space.[4] They are particularly important in regions where there are no lymphatic vessels.

The extracellular matrix of muscle consists of laminid, fibronectin, entactin, and collagen. It is organized into three interconnected sheaths. A collagenous epimysium surrounds the whole muscle. Collagen fibers extend inward from the epimysium to form the perimysium. Nerve branches, blood vessels, muscle spindles, and fat cells lie within the perimysium. Individual muscle fibers are covered with a sheath of endomysium. The endomysium contains capillaries, fine nerve branches, fibroblasts, macrophages, and a network of extracellular fibrils. The basal lamina is the endomysial component that lies closest to the muscle fiber surface. Satellite cells are wedged between muscle fiber and basal lamina.

FUNCTION OF THE INTERSTITIAL SPACE

The location of the interstitial space reflects the different functions it serves. The interstitial space is an extravascular flow system and a fluid reservoir. It has a storage function and gives immunological support. The solid structures of matrix give elastic support and participate in force transmission.

Extravascular Flow System

As a gel interposed between the capillary and lymphatic barriers, the interstitial space exerts influence on transcapillary partition and exchange of water and large solutes between blood and lymph. The interstitial fluid hydrostatic pressure determines the magnitude and direction of the fluid flow. The fluid flow system is a transport medium for nutrients and waste products.

Large molecules are transported through interstitial channels that end in endothelial-lined sacs that convert to collecting lymph channels.[5] The channels, which are widened by edema, facilitate convective flow.

In normally hydrated tissue, the interstitial space is a barrier against macromolecular transport. The structure of the interstitial space prevents flow of interstitial fluid from one level of the body to another. This preventive mechanism is disregarded in clinical conditions with edema. The change of hydraulic conductivity through the interstitial space in edematous tissue results in fluid flow to dependent parts of the extremity. On the other hand, fluid flow by convection is superimposed on many diffusion processes. This kind of flow is a prerequisite for transportation of large molecules such as hormones, enzymes, inhibitors, and activators.

Fluid Reservoir

The interstitial fluid space, which is three times larger than the intravascular space, also serves as a fluid reservoir for the cardiovascular system. An alteration in circulating plasma volume elicits a fluid shift across the capillary wall to maintain normal vascular filling.[1] The interstitial space consumes large volumes of fluid from the intravascular space during limb swelling. This may occur during a reperfusion syndrome[6–9] or following longstanding external compression, resulting in a crush syndrome.[10–13] Large volumes from the intravascular space may be withdrawn, leading to hypovolemic shock. The space is a continuous fluid reservoir that provides a pathway for diffusion and convection.

Storage Functions

As an ion-exchange resin, the matrix serves as a site for storage of large quantities of electrolytes, especially Na^+ and Ca^{2+}. It is an important reservoir of water and lipids.

Immunological Support

Many immunological reactions occur in this space. The channels of the space facilitate the communication and control of the defense mechanisms. The space acts as a barrier against infections and transportation of large molecules.

Elastic Support

The solid structures of the matrix provide elastic support and shock absorption for all cells. They can withstand longstanding and high pressures to resist tensile, compressive, and shear mechanical stresses, especially in bone and cartilage tissue. In bone tissue, most of the interstitial tissue is mineralized. In myocitis ossificans, the muscle tissue is calcified.

Regeneration of Muscle Tissue

The interstitial space serves as a scaffold for muscular generation following muscular damage due to ischemic, thermal, and mechanical insult. New muscle fibers regenerate from a resident population of satellite cells, which are wedged under the

endomysium of muscle fibers.[14–16] The numbers of nuclei of the satellite cells are about 5% of the muscle nuclei. The basal lamina of nerves and blood vessels also acts as a scaffold for generation. If the basal lamina is preserved, myotubes form within the original sheaths. If the myotubes are not oriented in parallel, the regenerated muscle generates less tensile force.

Force Transmission

Components of the extracellular matrix contribute to the mechanical properties of muscle. They serve to bind the contractile units together to provide for integrated motion among the muscle fibers during contraction. Longitudinal tension is required to restore the normal anatomy of the internal fibrous tissue of regenerating muscle tissue.[14] It is a prerequisite to regain normal muscle function.

The interstitial space accounts for the tensile strength of muscle, the distribution of force within muscle and the transmission of force from muscle to tendon. The contractile entity of muscle fibers is the sarcomere. The basal lamina takes part in the distribution of force along the muscle fiber as well as in the transmission of force from muscle to tendon. Molecules that connect basal lamina to muscle plasma membrane along its length and at the myotendinous junction provide possible basis for this mechanical linkage.[17] Muscle fibers are bound together by the matrix of the interstitial space into contractile units, which provide integrated motion stimulation. During muscle contraction, these filaments transfer tensile force generated by the muscle fiber to the interstitial space and further to the tendon. In this respect, the tendon can be regarded as a continuous extension of the interstitial space of the muscle itself (Figure 15.4).

Tendons transmit the forces generated by muscles to bone. The primary function is to achieve movement and stability of the body. The digital flexor and extensor tendons are stiff, whereas the Achilles tendon is more elastic and may be stretched in late stance phase. Tendons store energy on stretching and release it when the muscle contracts again.

Neuromuscular Transmission

The structure and function of the extracellular matrix are specialized at site for neuromuscular transmission. Acetylcholine transmits the nerve signal to the muscle. It is hydrolyzed by acetylcholinesterase, which is the only enzyme known to be associated in the extracellular space.

BIOMECHANICS OF THE INTERSTITIAL SPACE

Mechanical forces alter tissue growth and remodeling. The relationship is well documented and described in Wolf's law.[18] One mechanism for this may be convection, which alters the mRNA synthesis. The solid network can carry shear stresses for an indefinite period of time. A force applied to one face of the matrix exerts compressive stresses or tensile stresses in the collagen fibers throughout the matrix. The matrix allows the interstitium to become mechanically deformed and to return to the initial state (Figure 15.5).

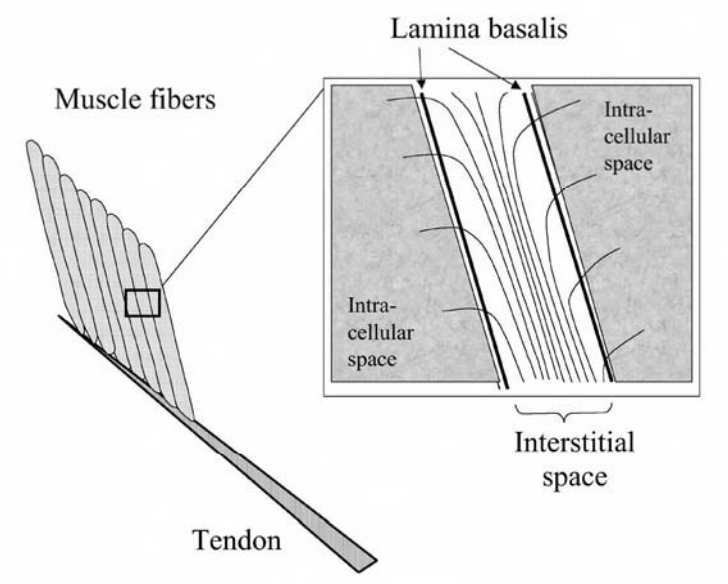

FIGURE 15.4 Relation among muscle fibers, the interstitial space, and the tendon. Tensile forces are transmitted along the whole muscle fiber by filaments into the interstitial space, which extends into the tendon.

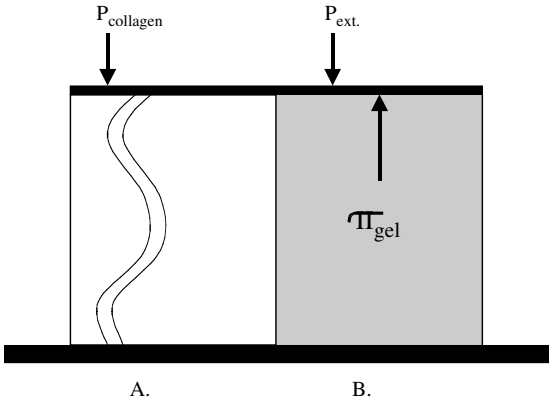

FIGURE 15.5 External forces (P_{ext}), the tensile forces of the collagen fibers ($P_{collagen}$) and the swelling pressure of the interstitial gel ($_{gel}$) create the mechanical equilibrium of the interstitial space. Solid components (A) such as collagen are under tension, whereas the gel (B) exerts a swelling pressure.

Forces and Stresses

A fluid deforms continuously under shear stress. Two kinds of forces act on fluids: (1) gravity and electrostatic forces act without physical contact; (2) compressive forces (pressure) and frictional forces require contact for transmission. These forces are called surface forces because they require a surface for their action. Stress created by gravity is a vector. It is a surface force per unit area. Stress is a vector because it has a magnitude and a direction (tensor of the second order). Fluid pressure, on the other hand, has only a magnitude and is therefore a scalar.

Shear stresses can only survive in the matrix network where they can deform the structure. In the fluid phase, shear stresses fall to zero because of fluid flow. The interstitial gel has been suggested to have characteristics between a solid and a fluid. Therefore, transmission of pressure through a gel obeys laws of pressure transmission that are a combination of both fluid and solid fiber pressure transmissions.[3]

In normally hydrated muscle and subcutaneous tissue, 50 to 100% of the force applied by external compression is transmitted into the fluid phase.[19–23] In normally hydrated tissue, 60 to 70% of the applied pressure is transmitted,[22] whereas in edematous tissue 100% of the pressure is transmitted.[22] This is also true for transmission of subatmospheric pressures.[24]

Hydrostatic Pressure Generation

Muscle fibers shorten 20% during maximal contraction. During contraction, the radius of the fiber curvature decreases by 50%. By using different mechanical models on muscular geometry, it is possible to explain by using the law of Laplace why intramuscular pressure is higher in the central part of the muscle.[25–27] Also, by applying the law of Laplace to a muscle with curved fibers and to a muscle with straight fibers, it is possible to explain why a curved muscle fiber with short radius of the fiber curvature generates higher stress to the muscle compared with that by a straight muscle fiber with a longer radius of fiber curvature. The relatively high stresses in the curved fiber generate high intramuscular pressures, as seen at the end of concentric muscular activity (Figure 15.6).[28] If the thickness of the fiber is h, and the number of fibers is n, the intramuscular pressure p can be calculated as:

$$p = p_o + s\, nh/r \tag{15.1}$$

where p_o is the hydrostatic pressure beneath the fascia, r is the radius of the fiber curvature, and s is the stress in each fiber.[25–27] The value of snh/r, i.e., the hydrostatic pressure, increases as the r value decreases.

Intramuscular Pressure as a Measure of Force Generation

Muscle contraction pressure is the interstitial fluid pressure created by a muscle as it contracts within its fascial compartment. This pressure correlates linearly with a contraction force in a specific muscle during isometric exercise of a wide range of forces.

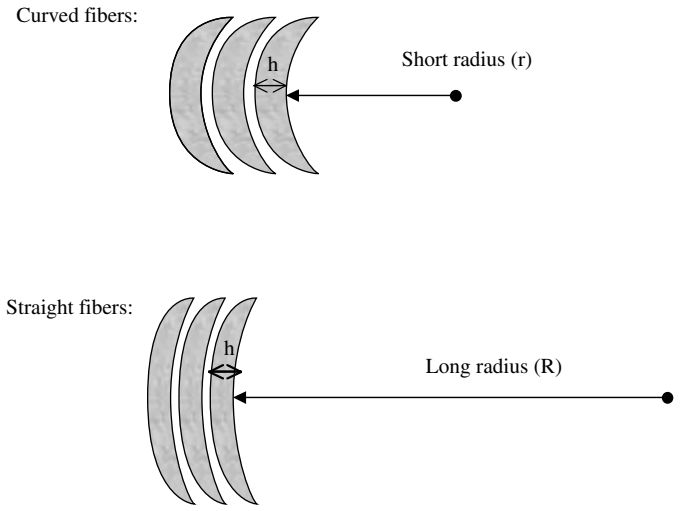

FIGURE 15.6 Different curvatures of muscle fibers with different lengths of the radius of the curvature. The number (n) and thickness (h) of fibers and the radius (R) of the fiber curvature determine the magnitude of the hydrostatic pressure.

Currently, there are no direct practical methods for measuring force production of individual muscles during dynamic exercise in humans. Implantation of a buckle transducer on a tendon is the most direct technique for individual muscle force assessment.[29]

Pressures in the Interstitial Space

Starling described the different pressures responsible for fluid shift between the capillaries and the interstitial space.[30] Scholander[31] and Wiederhielm[32] described the concept of a subatmospheric interstitial fluid pressure and physiochemical nature of the interstitium.

Following the structures in Figure 15.2, there are two kinds of tissue pressures in the interstitial space: (1) interstitial hydrostatic pressure, which is a scalar, and (2) interstitial solid tissue pressures, which are vectors. Total tissue pressure has been suggested to be the algebraic summation of the fluid and solid tissue pressure.[3] However, this concept is controversial, because a scalar and a vector must not be added. Hydrostatic pressures and forces in solid structures may be mechanically linked. They may balance forces in the solid structures of the interstitial space. Mechanical deformation of solid structures influences the hydrostatic pressure because the resistance for fluid flow through the normally hydrated interstitial space is normally high. This means that the hydraulic conductivity is fairly low.

Interstitial Fluid Pressures

The interstitial fluid hydrostatic pressure is transmitted in the tissue by fluid. In a normally hydrated tissue, most of the fluid is bound in the gel phase. Free fluid exists only in the canaliculi, which end up in the prelymphatic space.[4]

Starling, in 1896, described the regulation of fluid across capillary walls in what we presently call the Starling–Landis equation,[30] which was later modified by also taking the Staverman reflection coefficient for total protein into account:

$$J_c = K_f [(P_c - P_t) - \beta(\pi_c - \pi_t)] \quad (15.2)$$

where J_c is the net fluid flow across the capillary wall, K_f the capillary filtration coefficient, P_c the capillary blood hydrostatic pressure, P_t the hydrostatic pressure in the interstitial fluid, β the capillary membrane reflection coefficient, πc the capillary blood colloid-osmotic pressure, and πt the osmotic pressure in the interstitial fluid.

The interstitial fluid hydrostatic pressure (P_t) has a strategic position between the capillaries, cells, and lymphatics. It determines the magnitude and the direction of fluid movement between capillaries, cells, and lymphatics (Figure 15.1). It also determines whether free interstitial fluid will move in or out of the interstitial gel. Interstitial fluid pressure together with intracellular osmotic and hydrostatic pressures determines fluid flow through cell membranes. If the capillary filtration and the reflection coefficients are disregarded in Equation 15.1, the interstitial fluid pressure may be expressed as:

$$P_t = P_c - (\pi_c - \pi_t) \quad (15.3)$$

This implies that the negative free fluid pressure in the interstitium is in equilibrium with and equal to the absorption pressure of the interstitial gel, but not caused by it.[3]

Solid Tissue Pressure

Solid tissue pressures are pressures transmitted by the solid components. They are caused by forces that deform solid tissue structures. These types of pressures are never evenly distributed. Pressures within solids are rather stresses. Both pressures and stresses are subject to the same unit of measurement, namely force per unit area (Pascal or mmHg). Stresses vary throughout a solid body. Therefore, it is not possible to assign a single stress value to the interstitial fibers.

Total Tissue Pressure

Tissue pressure may also be involved in the forces from solid components operating on a vessel wall. The sum of hydrostatic and solid pressures is defined as total tissue pressure. This may be illustrated by the law of Laplace:

$$P_i - P_o = T/R \quad (15.4)$$

where P_i is the intravascular hydrostatic pressure and P_o the sum of all extravascular pressures. The equilibrium between these two pressures are balanced by the tension in the vessel wall (T_{wall}) and the radius of the vessel (R). P_o is the summation of all the partial pressures exerted on a collapsible vessel wall by cells, fluids, matrix, and the gel wall.[3,33] In Figure 15.7, the summation of all extravascular pressures is labeled P_{total}. It cannot be assumed that P_t in Equation 15.3 is equal to P_o in Equation 15.4,

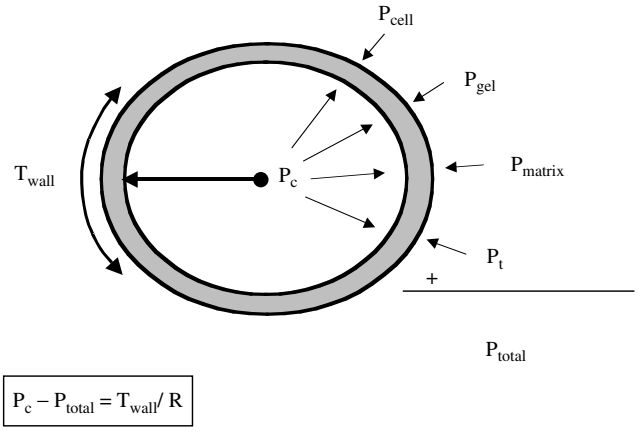

FIGURE 15.7 The total tissue pressure (P_{total}) is the sum of all the partial pressures from the cells, the interstitial gel, the matrix, and the interstitial hydrostatic pressure. The difference between the capillary pressure (P_c) and total tissue pressure is equal to the tension (T) in the wall divided by the radius (R) of the vessel.

or P_{total} in Figure 15.7. The difference between the P_c value and P_{total} value is the transmural pressure gradient. If P_o increases to abnormal levels, it may affect the local blood flow through the tissue.

The concept of total tissue pressure is controversial. The total pressure exerted on any given surface area in the tissue is defined as total tissue pressure. The total tissue pressure tends to compress blood vessels and other intratissue structures.[3,32] Total tissue pressure is not possible to measure, but it may be reflected by increased interstitial fluid hydrostatic pressure,[34] because different tissues have different abilities to withstand external forces. External forces may be transmitted into the tissue by both hydrostatic pressure and by compressive and tensile forces. The capacity of the solid components to withstand deformation as a response to external compression will decide the magnitude of tissue hydrostatic pressure. The effects of external compression on interstitial fluid pressure and solid tissue pressure was studied by Brace and Guyton.[19] They showed that the interstitial fluid hydrostatic pressure changed by 70% of the applied pressure in a normally hydrated tissue. In an edematous tissue, the fluid pressure changed by 100% of the applied pressure.[19,22] In overhydrated tissue, the interstitial fluid pressure was equal to total tissue pressure (Figure 15.8). External compression induces a blood volume shift. If the volume shift is impeded, the interstitial pressure changes by 100% of the applied pressure. The interstitial hydrostatic pressure may be considered the mechanical resultant of the interaction between hydrostatic and osmotic pressures on the one hand and mechanical forces on the other.

COMPLIANCE OF THE INTERSTITIAL SPACE

Interstitial compliance (C) is defined as the ratio between changes in the interstitial fluid volume (ΔV) and the interstitial fluid pressure (ΔP):

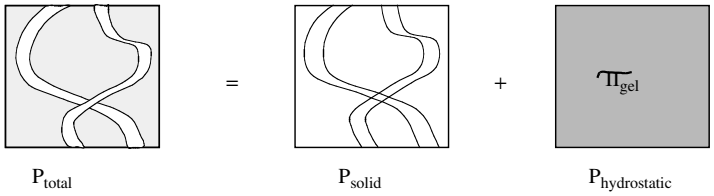

FIGURE 15.8 Total tissue pressure as the summation of pressure exerted by solid and fluid components of the tissue.

$$C = \Delta V/\Delta P \tag{15.5}$$

A low compliance implies that a small rise in interstitial fluid volume causes a large rise in interstitial fluid pressure. The compliance of a dehydrated tissue is low (Figure 15.9). Once the interstitial fluid pressure reaches atmospheric pressure level, the compliance suddenly increases manifold.[3] Substantial amounts of free fluid may be accumulated in the tissue with only minor pressure increase. Free fluid is the mobile fluid within the space. Pumping of the lymphatic system is the basic cause of the negative pressure in the interstitial space.[35] Taylor et al. showed that lymph flow increased up to 50-fold when interstitial fluid pressure increased from –6 mmHg to atmospheric level.[36]

Compliance of normally hydrated muscle tissue is high at rest.[37] The volume of muscle tissue may increase up to 20% following heavy exercise. In this situation, after activity the compliance decreases. Amplitudes of the pulssynchronous intra-

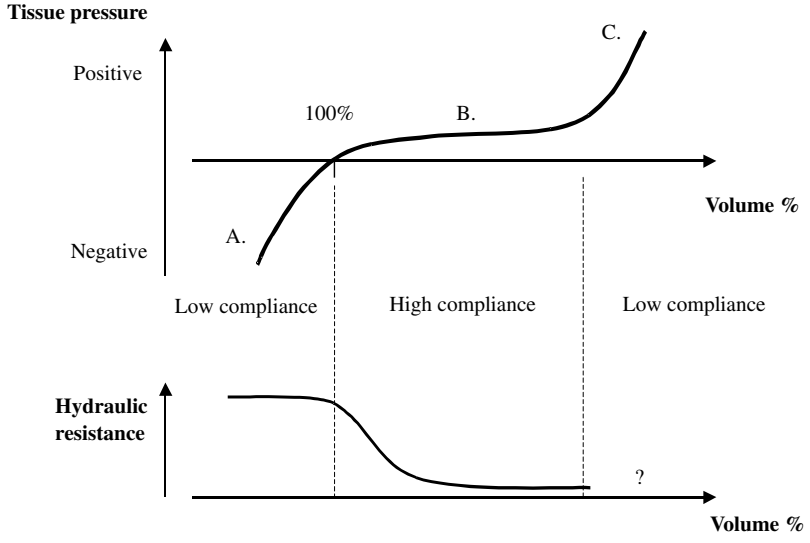

FIGURE 15.9 Relationship among tissue pressure, tissue volume, and hydraulic resistance in the tissue during different degrees of hydration. The compliance of edematous tissue is high (B). In dehydrated tissue (A) and in tissues with abnormal swelling (C), the compliance is low. The hydraulic resistance decreases once the tissue is edematous.

muscular pressure oscillations have been measured in normally hydrated muscle as well as following maximal exercise in normal subjects and in patients with chronic compartment syndrome. Patients with chronic compartment syndromes have significantly larger amplitude of the pulssynchronous intramuscular pressure oscillations after exercise as a sign of decreased compliance.

HYDRAULIC PERMEABILITY OF THE INTERSTITIAL SPACE

Glycosaminoglycans of the interstitial space have at physiological pH a net negative charge and are osmotically active. Therefore, they restrict free diffusion through the interstitium. The resistance to fluid flow through the interstitial canaliculi increases with $1/r^4$. It is high in a normally hydrated interstitial space (Figure 15.9).

Hydraulic permeability is the ability of water to flow through a tissue. The interstitial space contains interconnecting water-filled pores through which water flows. The flow rate is directly proportional to the pressure gradient over the tissue segment and to the cross sectional area. The hydraulic permeability can be described by Darcy's law:

$$Q = k\,Ad\,P/x \tag{15.6}$$

where Q is the flow rate (ml/min), k the hydraulic permeability, A the cross sectional area, dP/x is the pressure gradient across the tissue segment. The hydraulic permeability (k) varies with the degree of tissue hydration. In edematous tissue, the hydraulic permeability is high. Water flows easily through the tissue because the resistance for fluid flow is low. In dehydrated tissues, the k value is low, i.e., the resistance for fluid flow is high.

Inflammatory reactions increase vascular permeability[34,38,39] and cause a disaggregation of the ground substance. These changes favor activation and movement of fixed tissue macrophages as well as migration of inflammatory cells such as monocytes, leukocytes, and lymphocytes into the interstitial space. The increased movement of these cells contributes to increased immunological defense of the body.

SUMMARY

The volume of the interstitial space is 15% of the body weight, which is about three times more than that of the intravascular space. The volume of the interstitial space varies among tissues. It is 5% of the muscle tissue volume and 95% of the volume in the tendon. The interstitial space is an extravascular fluid system that supports the metabolic demands and acts as a fluid reservoir. It has a storage function and gives immunological support. Solid structures of matrix give elastic support and participate in muscular force transmission. The interstitial fluid hydrostatic pressure determines the magnitude and direction of fluid flow among capillaries, cells, and lymphatic vessels. Pumping of the lymphatic system is the basic cause of the negative pressure in the interstitial space. The interstitial hydrostatic pressure may be considered the result of the interaction between hydrostatic and osmotic pressures on

the one hand and mechanical forces on the other. Patients with chronic compartment syndromes have significantly larger amplitude of the pulssynchronous intramuscular pressure oscillations after exercise as a sign of decreased compliance of the interstitial space.

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16 Measurement of Intramuscular Pressures at Rest

INTRODUCTION

Accurate measurement of hydrostatic pressure is a valuable tool in the diagnosis of acute[1–5] and chronic compartment syndromes.[6–13] This measurement has been used to study the interstitial space[14,15] and fluid shift over the capillary bed.[16] The interstitial fluid hydrostatic pressure is one of the Starling forces that affects fluid transportation over the capillary bed.[17] It is an additional important parameter in physiological studies of tissue nutrition and viability.[18]

This chapter discusses the difficulties associated with invasive measurements of static intramuscular pressure at rest. Static pressure in muscle tissue is rather quasi-static, because it varies slowly, with a time constant of several seconds or minutes. The difficulties to be considered during intramuscular pressure recordings include trauma to muscular tissue by the recording device and occlusion of the catheter or needle especially during measurements for an extended period of time. Attention should also be paid to how muscle tissue is affected by the volume load following injection or infusion. The contact areas between the pressure-recording catheter and the tissue as well as the static properties of the pressure-recording equipment are important issues. Finally, measurement of intramuscular pressure, including technical knowledge and how to handle the technique for catheter insertion, has a significant learning curve.

MEASUREMENT QUALITY

A sensor converts a physical measurand, in this case a hydrostatic pressure, to a manageable quantity, e.g., an electrical signal. Important features of a pressure transducer that should be tested are accuracy, sensitivity, linearity, and hysteresis, all of which are discussed next.

Accuracy

Static accuracy is the reliability of the device to record stationary slow variations of intramuscular pressure. To obtain static accuracy, the system must be stable with a nondrifting baseline. If the recording system is not stable enough, it needs frequent baseline and calibration checks. The ability to determine the exact pressure and to reproduce a measurement achieving the same result is a desirable quality of a

transducer. Temperature drift is a common problem. Noise is unpredictable fluctuations of the output. It can be reduced by several filtering techniques.

Sensitivity

Sensitivity of a device is its ability to produce an output signal as a reaction of the pressure input. Static sensitivity of a transducer is the relation between an input (x) and output (y) signal. The sensitivity (s) of a transducer is defined as:

$$s = \Delta y/\Delta x \tag{16.1}$$

where Δy is the output change due to the input change Δx. The performance of the pressure-recording system may be tested by measuring the applied pressure and the recorded pressure at a low frequency. This may be regarded as a static calibration. The slope of the curve in Figure 16.1 illustrates the concept of sensitivity (s). A static pressure calibration curve should be linear to allow for a good sensitivity. Furthermore, the curve should maintain at origo (0,0). Environmental factors such as temperature and exposure to vibrations may change the sensitivity, and the curve may drift from origo. This is called a zero drift. Transducer-tipped catheters may be sensitive to temperature drift. The drift (mmHg/°C) must be known. Figure 16.1 illustrates sensitivity drift as well as zero drift. Fluid-filled pressure-recording systems may be repeatedly calibrated for zero drift. Bavetta et al. reported that zero

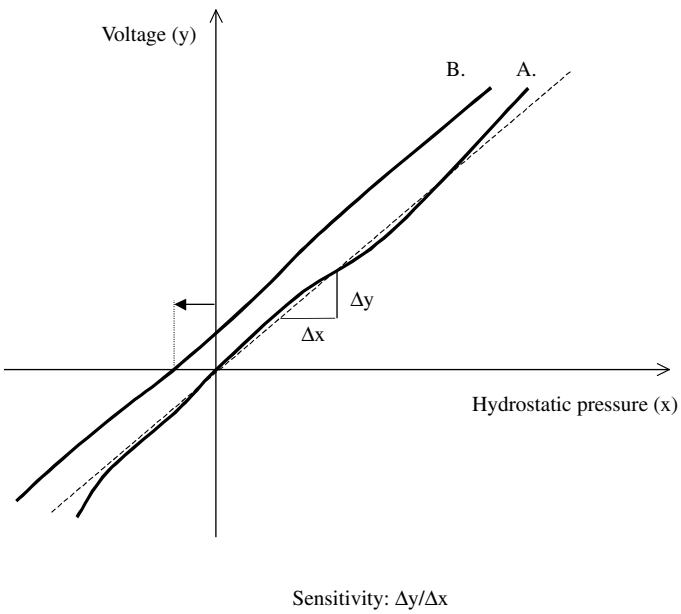

FIGURE 16.1 Linearity, sensitivity, and zero drift. The applied pressure is on the x-axis. The output is measured as voltage and plotted on the y-axis. Sensitivity is calculated from the calibration curve (A) by the slope of the least-squares line (dotted) which runs through origo. Curve B illustrates a zero drift (arrow).

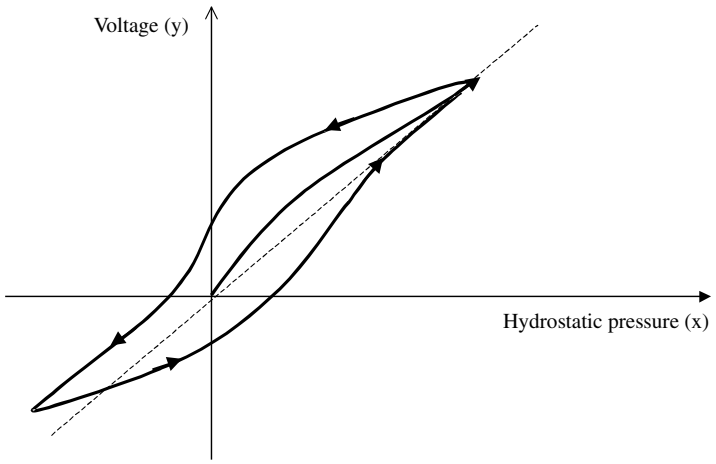

FIGURE 16.2 Hysteresis of a pressure curve. The pressure curves behave differently during pressurization and unloading. Hysteresis may be caused by both electrical and mechanical reasons.

drift of fiber-optic pressure transducers was a significant problem.[19] These catheters cannot easily be recalibrated following insertion.

Linearity, Uniqueness, and Hysteresis

Linearity is a quality of the sensor which roughly means that changing input pressure by a certain factor will result in a change of the output with the same factor (Figure 16.1). If the transducer response is linear, the sensitivity is constant all over the range of the transducer.

The system must imply uniqueness, which means that it will respond uniquely to any statically applied signal regardless of the way the signal is applied. Hysteresis exhibited by some recording systems is an example of lack of uniqueness in static response. Hysteresis of a pressure curve describes the behavior during pressurization and unloading (Figure 16.2). It may be due to electrical and mechanical components. It may result from energy being absorbed by the system during pressurization and being recovered during decompression (Figure 16.2).

COMPLIANCE OF THE PRESSURE-RECORDING SYSTEM

It is important to know the compliance of the pressure-recording system. Pressure changes will be erroneous if the volume shift at the tip of the catheter is too small to have a complete effect on the transducer. The compliance (C) of a transducer is the relation between the volume change (ΔV) and the pressure change (ΔP), given by the following equation:

$$C = \Delta V/\Delta P \tag{16.2}$$

A crucial and maybe the most difficult part of the preparation for pressure recordings is filling the transducer and the transducer line with saline that is free of entrapped air bubbles. Even fine air bubbles in the recording system increase the total compliance considerably and decrease the dynamic properties. If cold saline from the refrigerator is used, air bubble formation increases at room temperature. The same is true when saline at room temperature in the catheter is exposed to warm (35 to 38°C) skin and muscle tissue. Compliance of fluid-filled pressure transducer plus catheter ranges between 0.089 and 0.14 μL/mmHg.[10,20–22]

CATHETER INSERTION TECHNIQUE

Calibration and check of all equipment must be done before the catheters are inserted. The catheter insertion technique is important to obtain reliable intramuscular pressure recordings. The best site for catheter insertion should be decided by palpating the muscle belly during an active contraction and by using anatomical landmarks. Ca. 1 to 2 mL of local anesthesia should be installed subcutaneously. The underlying muscle fascia should not be penetrated. Local anesthesia may inhibit local muscle function and create a local swelling of the muscle by an inflammatory reaction. The patient should be asked to contract the muscle to be studied. For example, if the tibialis anterior muscle is to be studied, the patient should dorsiflex his/her ankle joint. The introducer or troachar should be inserted through the skin and fascia with a sharp steel needle while the patient keeps the muscle contracted (Figure 16.3A). The sharp inner needle should be withdrawn slightly to a point where the plastic sheath of the introducer (Figure 16.3B) protects the cutting edge of the needle tip.[10] The patient should be asked to relax his/her muscle. The plastic sheath should be inserted parallel to the muscle fibers simultaneously.[23] The steel needle or troachar should be withdrawn (Figure 16.3C). No bleeding should be visible through the plastic sheath. If this is the case, the investigation should be finished, the introducer retracted, a compressive dressing applied, and the limb kept elevated for a short while.

The catheter should be connected to a transducer line filled with saline. The catheter will be easily filled from a syringe at the transducer or from a saline bag by flushing the microcapillary. The catheter should be inserted into the tissue through the plastic sheath and the sheath retracted while keeping the catheter tip at the same spot in the muscle (Figure 16.3D). The catheter should be secured to the skin.

Gelberman et al. described a volar approach starting at the junction of the proximal and middle third of the forearm.[24,25] McDoughall et al. suggested a dorsal approach for catheter insertion in the supinated forearm.[26] By using the ulna as a guide, the catheter should be advanced through the dorsal compartment and interosseous membrane into the deep volar compartment.

It is important that the catheter be inserted parallel with the muscle fibers to minimize trauma and discomfort.[23] At least 2 cm of the catheter should be inserted into the muscle to prevent fluid leakage back along the catheter.[22] This compares in most cases at a total distance of 4 cm from the skin if the catheter is inserted parallel with the muscle fibers.[23] An unintentional muscular activation may even be painful if the catheter is placed in a wrong way. Chapter 17 discusses the matter more extensively, and describes intramuscular pressure recordings during exercise.

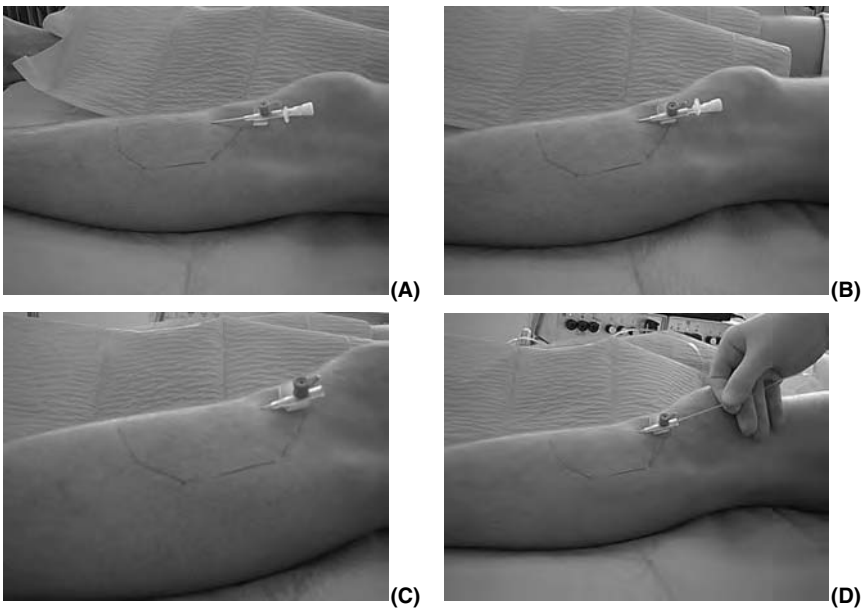

FIGURE 16.3 Pictures of catheter insertion. After the muscle fascia has been penetrated, the steel needle should be withdrawn a few millimeters into the plastic sheath of the introducer (A). In this way the cutting tip of the needle will not traumatize the muscle tissue when the introducer is advanced parallel with the muscle fibers into the preferred position (B). The steel needle is retracted (C) and replaced by the pressure-recording catheter (D). Finally, the plastic sheath of the introducer is removed. (See color insert following page 106.)

Response and Location of the Catheter Tip

Several tests are available to make sure that the tip of the catheter is located within the muscle to be studied. The response of the catheter is checked by external compression by a constant force over the muscle belly from distal to proximal by the investigator's fingertip. The pressure amplitude of the response is highest at external compression over the catheter tip (Figure 16.4). Another catheter patency test is intramuscular pressure recording during an active muscle contraction.[27] As a test of catheter location in the deep posterior compartment of the leg, intramuscular pressure recordings during a maximum flexion of the toes and then again during plantar flexion of the ankle joint are recommended (Figure 16.5). By analyzing the pressure amplitude of the response by the two activities, the degree of agonistic coactivation between the muscles may help to decide catheter location in the flexor digitorum muscle and posterior tibial muscle.[28] Another way to determine catheter location in the leg or forearm is ultrasound.[29]

Tissue Trauma due to Catheter Insertion

The extent of tissue trauma to the muscle depends on the technique for introducing the catheter, the size of the introducer, and on the design of the catheter tip. The

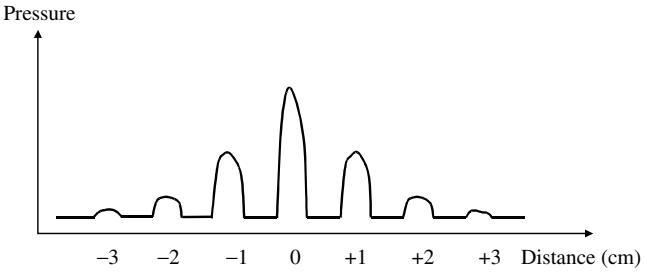

FIGURE 16.4 Amplitude of intramuscular pressure oscillations, which is synchronous with external compression by the investigator's fingertip, is useful to identify location of catheter tip. On the horizontal axis, 0 indicates the position of the catheter tip. The distance distal (negative numbers) and proximal (positive numbers) from the catheter tip are indicated.

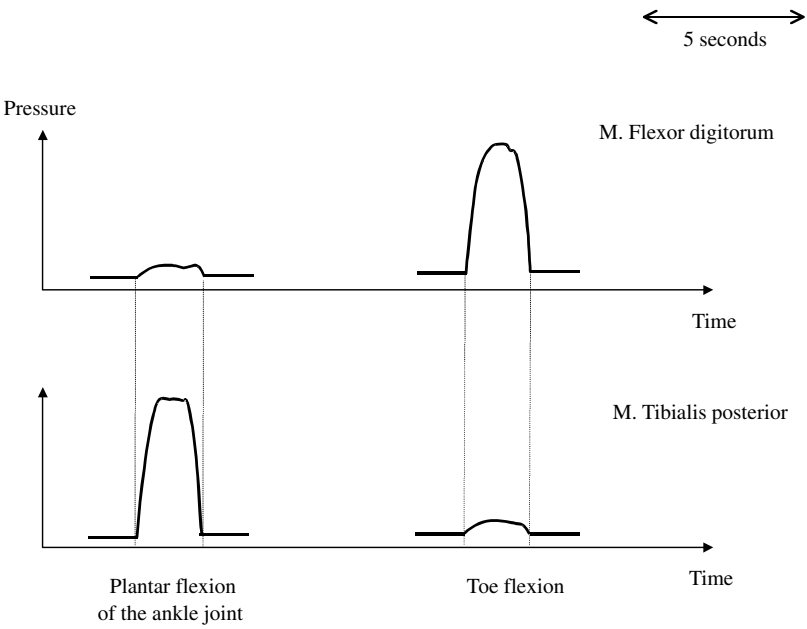

FIGURE 16.5 Amplitude of intramuscular pressure in the flexor digitorum longus muscle (upper trace) and tibialis posterior muscle (lower trace) during flexion of the ankle joint followed by toe flexion. The pressure responses indicate that catheters are correctly located. The pressure response in the tibialis posterior muscle during toe flexion is a sign of coactivation of the tibialis posterior muscle.

technique should be as atraumatic as possible. This can be achieved by choosing a blunt insertion technique once the skin and the fascia have been penetrated, as shown in Figure 16.3. By retracting the cutting needle into a position where the plastic tube of the introducer covers the tip, the muscle fibers are protected.[10,30] As mentioned previously, the catheter for intramuscular pressure measurements should be introduced parallel to the muscle fibers (Figure 16.3).

When pressures are recorded by needle techniques, a separate cutting needle may be used to penetrate the skin and subcutis. A semicutting needle with an occluded tip and open sidehole(s) can be inserted through the holes and then advanced into the muscle parallel with the fibers.

Tissue injury at catheter insertion results in local inflammation mediated by histamine, prostaglandins, and quinines.[31] Tissue injury may increase capillary permeability and tissue swelling due to interstitial absorption of fluid and proteins from the vascular bed.[32] This increases tissue fluid hydrostatic pressure. The increased capillary filtration due to increased permeability returns to normal values within 30 min. Therefore, intramuscular pressure decreases during the initial 30 min of pressure.

Catheter Occlusion

Several principles have been used to prevent occlusion of the catheter tip by tissue or by blood clots (Figure 16.6). The catheter tip can be closed and replaced by one or several sideholes.[22] Cotton or Dexon wicks that extend into the tissue from the catheter may be used to prevent occlusion.[33,34] Injection and infusion have been used

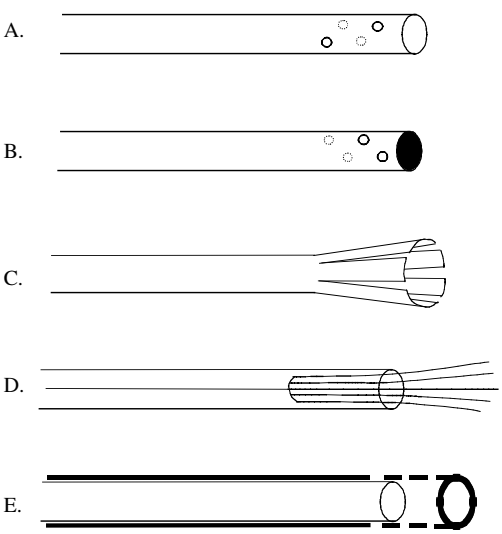

FIGURE 16.6 Examples of different principles to prevent occlusion of the catheter tip. These include multiple sideholes with (A) or without (B) an open tip, slits at the tip (C), and wick strands (D) extending into the tissue. (E) The tip of the catheter may be covered by a protective sheath, e.g., the STIC catheter.

to keep the tip open.[10,22,35–37] Protective sleeves around transducer-tipped catheters have also been used.[38–40]

Contact Area of the Catheter

The area of contact is the interface between the catheter tip and the surrounding tissue over which hydrostatic pressure equilibration takes place. Many catheter tips are designed to increase the contact area to obtain shorter equilibration time, prevent occlusion, and decrease the risks of recording erroneously high pressure due to volume load when catheters are infused or injected.

With the wick catheter method[33,34] and the wick-in-needle method,[41] the contact area is increased by the strands of wettable material extending into the tissue from the catheter or needle. The slit catheter has five symmetrical slits at the end extending 3 mm along the length of the catheter tube.[36]

Teflon catheters with multiple sideholes at their tip are another way of increasing the contact area.[10,22,42,43] Needles for pressure recordings have also been designed with one sidehole[44] or several sideholes.[43] Transducer-tipped catheters may be combined with protective saline-filled sheaths with multiple sideholes.[40] The sleeve also protects the transducer from forces exerted by solid components in the tissues.

Volume Load

The volume load of muscle tissue during pressure recording depends on the volume infused or injected during measurement and on the volume of the catheter itself. High intramuscular pressures at rest have been reported with large catheters such as the STIC catheter.[45] This may be due to the sudden volume load by the catheter, which is 2.5 mL combined with an infusion rate of 3 mL/h. Injection through needles without sideholes may also give an erroneously high intramuscular pressure reading (Figure 16.7).[43] When intramuscular pressures are recorded by the injection techniques, one should wait for at least 30 sec following injection before taking the pressure reading.

Physiologic Reactance

Physiologic reactance is the undesirable effect of any recording device on the physiologic event. The concept has been emphasized in recordings of intraarterial pressure[46] and suggested to be valid for intramuscular pressure recording.[47] The presence of a catheter in muscle tissue may alter the function of the muscle because of the discomfort or pain the subject may experience. Furthermore, most catheters used in monitoring the interstitial fluid hydrostatic pressure are at least 100 times larger than the interstitial space itself. It is unlikely that under these conditions, the fluid hydrostatic pressure in the interstitial space behaves normally. Most pressure-recording devices measure volume changes at the tip of the catheter. If the device does not have a low compliance to the flow of fluid in the interstitial space, true pressures cannot be measured.

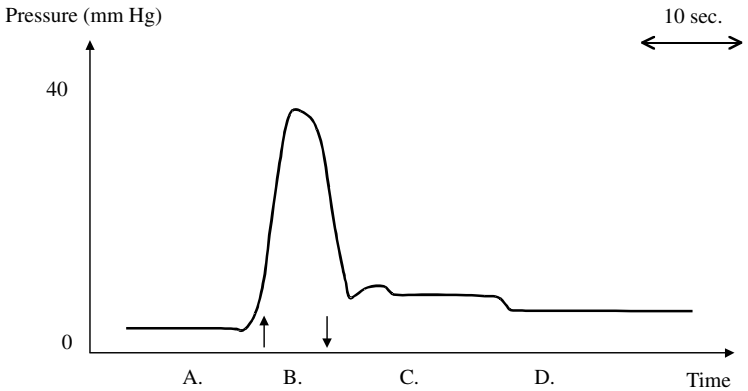

FIGURE 16.7 Intramuscular pressure measured by the needle injection technique combined with an electrical transducer and recorder. (A) Intramuscular pressure at rest recorded before injection. (B) Intramuscular pressure during injection. (C) Intramuscular pressure following injection while the meniscus was concave. (D) Pressure recording when the meniscus turned straight.[43]

CLASSIFICATION OF CATHETERS FOR INTRAMUSCULAR PRESSURE MEASUREMENT

Direct pressure sensor systems can be divided into water-perfused systems and catheter-tipped sensors. Fluid-filled systems are still the most common while the new transducer-tipped systems are developing fast. To solve some of the problems discussed previously, various catheters and methods for direct measurements of pressure within muscle tissue have been devised. Techniques for intramuscular pressure recordings can be classified based on the design of the catheter tip, location of the transducer, and the medium through which the signal is transmitted.

Design of the Catheter Tip

Intramuscular pressure may be recorded by needles with different tip designs and sizes, such as the wick catheter, wick-in-needle, Slit® catheter, Intracath® catheter, and Myopress® catheter (Figure 16.6). All these catheters require an extracorporal transducer. The transducer must always be adjusted to the same height as the catheter tip to avoid hydrostatic artifacts. Transducer-tipped catheters with or without a protective sleeve need no height adjustment.

Needle Designs

The needle technique for recording tissue pressure was described in 1884.[48] Since then, the technique has been modified and applied to clinical use.[35,37,49,50] In later studies,[37] the injected volume exceeds by far the 0.1 to 0.5 µl that was used in earlier studies.[35] Also, the design of the needle tip has been changed. To increase the contact area, needles with multiple sideholes[35,43,50] and with one sidehole and a wick[44] have been used. A needle without one or several sideholes at its tip is

less suitable for routine pressure measurements with the injection technique in the clinical setting. However, measurements of intramuscular pressure with the meniscus method following injection and by a needle with multiple sideholes eliminate the drawbacks mentioned.

Wick Catheter

Originally braided loose cotton wicks with a fiber diameter of 8 to 16 μm were pulled into the end of a Teflon™ tubing by means of a monofilament loop.[34] The tubing had an outer diameter of less than 1.5 mm. Its other end was connected to a glass tube. The assembly was filled with saline to a given mark. The wick catheter was inserted through a hypodermic needle, which was pulled back leaving the Teflon tubing with the wick in the tissue to be studied.

Mubarak et al. improved the wick catheter technique in 1976. They used soluble braided No. 1 Dexon™ sutures with a fiber diameter of 20 μm (Davis and Geck). Two pieces, each 3.5-cm long, of the suture were tied at their midpoints to a 6-0 monofilament suture which was 25 cm long.[33] The filament was passed through an epidural catheter with an outer diameter of 1 mm and an inner diameter of 0.6 mm. The filament fitted with a Luer-lock mount and a three-way stopcock. The Dexon fibers were pulled into the catheter for the distance of 1 cm, leaving 7.5 mm of the Dexon fibers outside the orifice. The monofilament was cut just inside the Luer-lock fitting. In this way, the wick fibers were retrievable should they slip out of the catheter. Before insertion, the catheter was connected to a transducer line and a transducer filled with sterile heparinized saline.[33] It is important not to pack up the Dexon wicks too tightly into the catheter orifice to avoid partial occlusion. One piece of a shorter (2.5 to 3 cm) Dexon also suits well. The wick catheter has no artifacts associated with fluid injection. The wick has an increased contact area with the tissue and keeps the orifice of the catheter open. However, the dynamic properties of the wick catheter are slow.[10] It has been suggested that the wick catheter measures osmotic pressure.[51] In many studies, the wick catheters are frequently flushed. In such cases, the device is no longer a noninfusion method, but rather an injection method.

Slit Catheter

The slit catheter consists of a 20-cm-long polyethylene tubing (PE 60). The tip of the catheter has five symmetrical slits that extend 3 mm along the length of the tubing.[36] The catheter is connected via a Luer-lock to a transducer line. The catheter is gas-autoclaved in ethylene oxide. The whole pressure-recording system including the catheter is filled with saline before insertion. The slits of the catheter prevent occlusion of the tip and increase the contact area. The catheter has no detachable pieces or parts. However, the slits may become distorted and occlude the tip of the catheter if used during exercise.[52]

Myopress Catheter

The Myopress catheter is 30 cm long with an outer diameter of 1.05 mm. The catheter has four sideholes with a total area of 1.5 mm^2 at its terminal 1 cm.[10] The

holes increase the contact area between the catheter and the tissue. The tip of the catheter may be looked upon as a Guyton's capsule[53] extended to a cylinder. The catheter has been proven to be suitable to record intramuscular pressure at rest with injection,[43] infusion,[10,54] and noninfusion.[10] The compliance of the pressure recording system is 0.083×10^{-3} mL/mmHg if a transducer with a displacement of 0.57×10^{-6} mL/mmHg is used.[10,47]

Intracath Catheter

Matsen et al. used an Intracath catheter (Model 3166) to measure intramuscular pressure at rest.[22] The catheter was prepared by occluding the end hole with adhesive cement and cutting four sideholes in the terminal 1 cm. The compliance of the total pressure recording system is 0.25×10^{-3} mL/mmHg if pump infusion is used.[10]

Wick-in-Needle

The device consists of a steel cannula with an outer diameter of 0.6 mm and a 3-mm-long sidehole about 5 mm from the tip.[44] Strands of nylon fibers with a filament thickness of 25 μm are pulled into the needle by a fine steel-wire loop. The compliance of the system is 0.06 μL/100 mmHg.

Location of the Transducer

The transducer may be located outside the tissue such as in the traditional fluid-filled system or inside the tissue at the tip of the catheter. Transducer-tipped catheters may be fiberoptic or solid-state intracompartmental catheters. Figure 16.8 shows different transducer-tipped catheters.

Fiberoptic Transducer-Tipped Catheter

The pressure-sensing mechanism of the fiberoptic system (Model 420, Camino Laboratories, San Diego, CA) is located at the tip of the catheter.[39] The fiberoptic catheter is inserted enclosed in a saline-filled sheath with an outer diameter of 2.1 mm and with 12 sideholes at the tip (Figure 16.8A). The contact area between muscle tissue and the catheter tip is 9.6 mm^2. The volume of the catheter and the sheath in the muscle is 157 mm^3. The compliance of the system is 5×10^{-6} mL/mmHg.

Solid-State Intracompartmental Catheter (STIC)

The STIC catheter is made of noncompliant polyethylene tubing. It has a 10-cm-long curved tip with 52 sideholes, which provide a total contact area of 8.3 mm^2 with the tissue.[45] A three-channel connector is attached to the catheter and then a Millar Micro-tip transducer (No. PC-340, Millar Instruments, Inc., Houston, TX). The catheter needs to be inserted by a 14-gauge needle. The cable of the Millar transducer is 120 cm long and has an outside diameter of 1.33 mm.

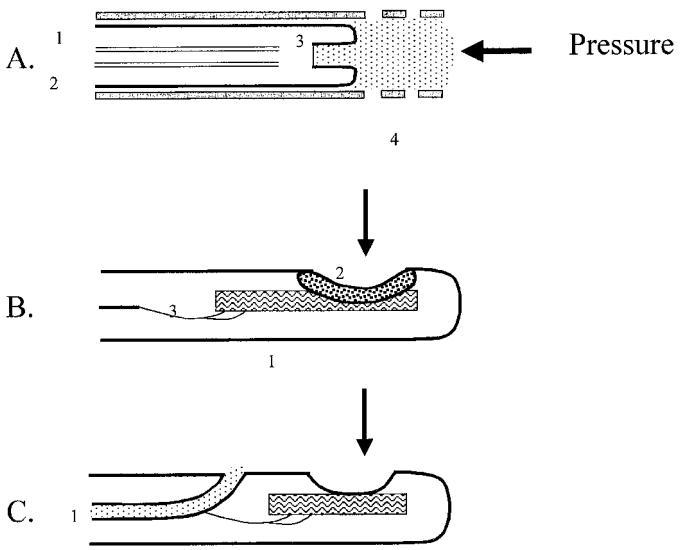

FIGURE 16.8 Principles of transducer-tipped catheters. (A) The fiberoptic transducer-tipped catheter. 1: sending fiber, 2: receiving fiber, 3: moving diaphragm, and 4: plastic sheath with 12 sideholes. (B) The Kodiag catheter. 1: stainless-steel housing, 2: unicristalline piezo semiconductor with a chip, 3: wires to the connector. The Codman catheter is constructed in a similar way. The transducer is protected in a groove. (C) Principle for transducer-tipped catheters with possibilities for injection or infusion. They may be combined with (STIC catheter) or without (Millar Micro-tip catheter) a protective sheath.

Codman Catheter

The Codman transducer-tipped catheter has been developed for measuring intracranial pressures (Figure 16.8C). It has been evaluated for intramuscular pressure measurement.[55] It may be classified as a noninfusion intracorporal pressure transducer. The transducer element consists of a silicon chip with pressure-sensitive resistors forming a bridge. The integrated strain gauge is housed in a titanium case (diameter 0.7 mm) which is placed at the tip of a Teflon catheter. The membrane of the pressure transducer is countersunk in the catheter to avoid artifacts due to mechanical contacts with the surrounding tissue. This is important because solid pressures in the tissue will not affect the transducer. The transducer is electrically connected to a digital monitor. The working range is –50 to +250 mmHg. The stability of the microsensor is good. The long-term drift is less than 0.8 mmHg per 24 h. The temperature drift is less than 0.03 mmHg/°C.

The Kodiag Catheter

The Kodiag catheter consists of a piezoresistive pressure-recording system (Figure 16.8B). It consists of a reusable probe that is 60 cm long and has an outer diameter of 0.99 or 1.32 mm (French size 3 or 4).[56] The catheter is connected to a hand-held battery-powered device. Like all unprotected transducer-tipped catheters, it should

TABLE 16.1
Classification of Common Pressure-Recording Principles (Infusion, Noninfusion, and Injection) for Intramuscular Pressure Measurements Related to the Medium of Signal Transmission

	Saline	Electricity	Light	Ref.
Infusion				
Pump infusion	+			23
Microcapillary infusion	+			10
STIC	+			39
Noninfusion				
Wick catheter	+			32
Slit catheter	+			35
Camino catheter (FOTT)			+	38
Codman catheter		+		55
Kodiag		+		56
Injection				
Needle injection		+		36

be inserted parallel to the muscle fibers to avoid contact with muscle and tendon that may press on the sensing area of the probe. If this is the case, the catheter reads forces from the solid structures of the tissue. One advantage of transducer-tipped catheters compared with fluid-filled systems is their lack of hydrostatic artifacts when subjects raise or lower their extremities. They are easy to use.

Disadvantages include the possibility of the sensor of the catheter tip to be affected by solid components of the interstitial space. Therefore, the sensor must be protected from contact with the solid structures by a protective covering sheath or by a small measuring window. The intramuscular pressure recording may be performed as a noninfusion technique as in the fiberoptic transducer-tipped catheter[39] and the Kodiag piezoresistive system.[57] It may also be performed as an infusion technique as in the STIC catheter method.[45]

Medium of Signal Transmission

The medium through which the signal is transmitted may be saline, light, or electricity (Table 16.1). It is the sensor that converts the physical measurand to an electrical signal. One example of this is fluid-filled catheters in which the hydrostatic saline pressure is converted by the transducer into an electrical signal.

TECHNIQUES FOR INTRAMUSCULAR PRESSURE MEASUREMENTS

Direct methods for intramuscular pressure measurements include needle injection techniques,[20,35,37,50] infusion techniques,[22,58] and noninfusion techniques.[33,34,36] Transducer-

TABLE 16.2
Ten Different Catheter Designs and Techniques for Measuring Intramuscular Pressure at Rest in Humans[a]

Catheter Design	Injection	Infusion	Noninfusion
Fluid-Filled Systems			
Needle	+		+
Wick-in-needle	+		
Wick catheter			+
Myopress catheter	+	+	+
Slit catheter	+	+	+
Intracath catheter	+	+	+
Transducer-Tipped Systems			
Fiberoptic transducer-tipped			+
STIC catheter		+	
Kodiag			+
Codman			+

[a] Fluid-filled systems are divided into injection, infusion, and noninfusion methods. The transducer-tipped techniques are divided into infusion and noninfusion methods.

tipped techniques[38–40,59,60] may be used with or without infusion. The servo-null technique that uses a micropipette[16,61] is not discussed here. Different catheter tip designs may be combined with different techniques for pressure measurements (Table 16.2).

Injection Techniques

Landerer first described the needle injection technique in 1884. It consists of a fluid-filled needle or a pressure catheter, transducer, three-way stopcock, a syringe, and a manometer (Figure 16.9). Since then the technique has been refined,[35,62] evalu-

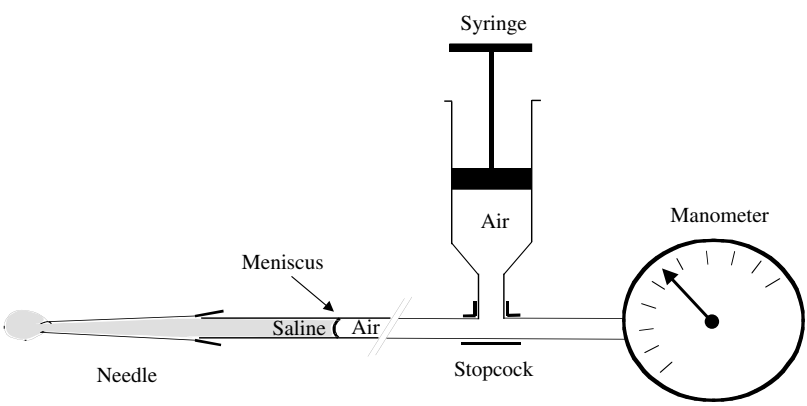

FIGURE 16.9 Needle injection technique by a manometer.

ated,[20,36,43] and applied for clinical use.[43,50,63] It can only be used to measure pressures at rest. The needle injection technique has been shown to be less accurate for pressure recordings at rest compared to other techniques.[36,43,64] The injected volume through an injection needle and the small contact area, over which equilibrium takes place, increases the measured tissue pressure artificially. In later studies,[37,65] the recommended volume of 0.1 to 0.3 mL far exceeds the 0.1 to 0.5 μL that was used in earlier studies.[35] Also, the design of the needle tip has been changed since then. With the needle injection technique, tissue pressure has been taken as the manometer reading at the moment when the saline meniscus just moved[66] or when it steadily moved,[37,67] or when the air–saline meniscus interface changed from concave to straight.[43] This means that pressures were taken during injection in the first two cases and immediately following injection in the third case.

The lack of sensitivity by the needle injection technique may be due to the low fluid conductance in a normally hydrated tissue and on the small cross sectional area of the needle tip, which leads to a small contact area with the tissue.[20,68] This results in a high fluid resistance. Therefore, pressure measurements during injection give artificially high intramuscular pressure reading,[43] which is illustrated in Figure 16.10. Measurements of hydrostatic pressure in normally hydrated tissues are not possible with injection techniques (Figure 16.10A). The rate of injection influences the measured pressure, which is illustrated in Figure 16.10C.

Infusion Techniques

There are two types of infusion techniques: the constant pump infusion technique[22] and the microcapillary infusion technique.[10] The difference between

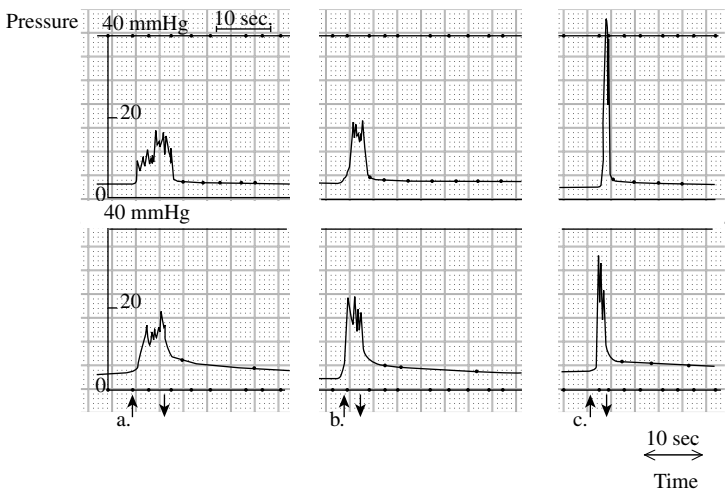

FIGURE 16.10 Results of intramuscular pressure recordings from a needle with sideholes (upper trace) and without sideholes (lower trace). Magnitudes of pressure recordings during injection depend on the time duration of the injection. Start of injection is indicated by "arrow up" and end of injection by "arrow down." Intramuscular pressure at rest after injection is significantly higher following injection by a needle without sideholes.

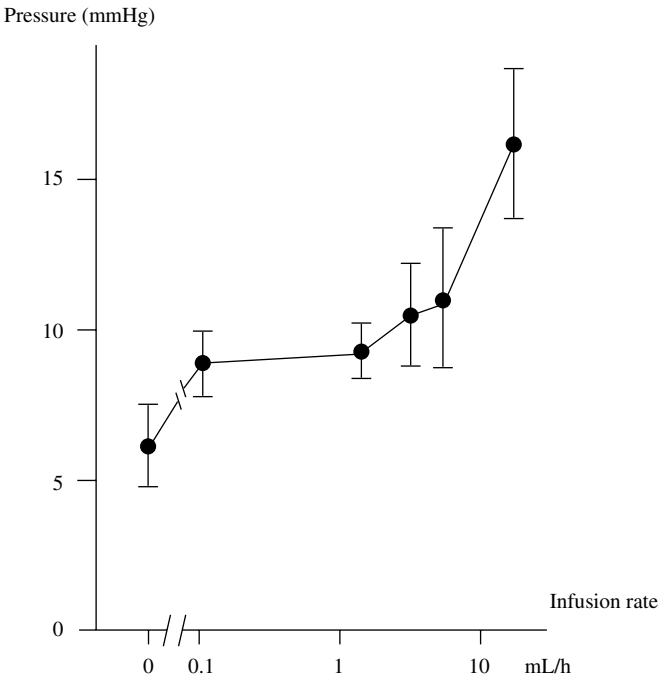

FIGURE 16.11 Intramuscular pressure at rest depends on the infusion rate. It is fairly constant when infusion rates are kept between 0.1 and 3.0 mL/h. Most of the time intermittent infusion rates of 0.1 to 0.3 mL/h are sufficient for recordings up to 24 h.

the two methods concerns only intramuscular pressure recordings during exercise. Therefore, they will be discussed in more detail in Chapter 17. These techniques may be combined with several catheter tip designs. The amount of fluid infused affects the level of intramuscular pressure recorded.[10] However, in normally hydrated anterior tibial muscle, pressure did not increase during 30 min with an infusion rate of between 0.1 and 3 mL/h. Pressure was about 4 mmHg without infusion and increased to about 6 mmHg when infusion was 0.1 mL/h. Figure 16.11 illustrates that there is a fairly wide range of low infusion rates that does not significantly affect intramuscular pressure at rest. It is well known that large volumes of fluid are required to cause even a slight additional increase of normal interstitial fluid pressure.[68] Substantial amounts of fluid can be added to the interstitial space of skeletal muscle with only small alterations of the tissue fluid pressure.[69] The relation between infused volume into muscle and intramuscular pressure at rest has been documented for shoulder muscles,[70] lumbar back muscles,[54] and leg muscles.[10,27]

Noninfusion Techniques

Fluid-filled catheter systems with external transducers, which are not injected or infused, have been used extensively.[33,36] Many catheter designs have been

combined with noninfusion techniques to measure intramuscular pressure.[33,36,52,71] The noninfusion technique is required when measuring the fluid hydrostatic pressure of a normally hydrated tissue.[34,44,51,72] Most noninfusion techniques require a small amount of fluid to be injected initially. However, no injection or infusion is allowed during the recording. If this is the case, the method is no longer a noninfusion technique.

INTRAMUSCULAR PRESSURE MEASUREMENTS IN A SUBATMOSPHERIC ENVIRONMENT

Subatmospheric pressure has been applied on human limbs to study blood circulation, transcapillary fluid dynamics,[16,73] and countermeasures in simulated microgravity.[74] The Myopress catheter combined with microcapillary infusion technique and the fiberoptic transducer-tipped catheter (Model 110-D, Camino Laboratories, San Diego, CA) is suitable to measure intramuscular pressures in the leg when the pressure in the chamber varies between atmospheric and –100 mmHg.[75]

METHODS FOR NONINVASIVE MEASUREMENTS OF INTRAMUSCULAR PRESSURE

An indirect method to estimate intramuscular pressure in the leg was described by Kjellmer.[76] Pressure measurements were based on the following considerations: (1) flow through a collapsible tube can occur only when the pressure inside the tube is at least as high as that outside; (2) venules and veins are collapsible tubes; (3) tissue pressure acts directly on the venules and veins; and (4) intravascular pressure in the venules and veins can never be lower than the tissue pressure.[76,77] By recording arterial pressure in the contralateral thigh, venous outflow in the popliteal artery, and leg volume, the maximal tissue pressure in the leg was obtained from distensibility curves of the veins.[76]

Pressure in the anterior compartment was estimated by auscultation over the anterior tibial artery. Willey et al. estimated intramuscular pressure in the anterior compartment by auscultating the Korotkoff sounds over the anterior tibial artery above the ankle joint at rest and after exercise in the elevated limb.[78] The height (H) above the heart at which the sounds disappeared was measured. Compartment pressure (P) was calculated as the brachial diastolic pressure (D) minus the hydrostatic pressure due to the height of the column (H) in centimeters:

$$P = D - 0.8H \tag{16.3}$$

The coefficient 0.8 is calculated from the specific gravity of blood (1.06 g/cm^3) and mercury (13.6 g/cm^3).

Steinberg and Gelberman measured quantitative hardness of leg muscles. They used a low-friction probe connected to a plunger, a Teflon seal, and a hard plastic housing. Because the displacement of the plunger was directly proportional to the pressure in the probe, they could calculate the displacement of the plunger for any

pressure. They found that hardness of the muscle correlated well with actual invasive intramuscular pressure measurements.[79]

SUMMARY

No single method for intramuscular pressure measurement is superior to all methods. The choice of method must be based on pressure to be measured, clinical setting, and duration of intramuscular pressure recordings. The choice of method is influenced by economical aspects, whether the pressure is recorded at rest or during exercise, or both, with the subject lying supine, or whether the exercise includes complex movements.

The most important requirements for the pressure-recording system are that it must be stable, robust, and easy to calibrate. The size of the sensor must be small, preferably 0.3 to 0.6 mm in diameter, to minimize physiological reactance. The diameter of catheters for intramuscular pressure recordings should not exceed 1 mm. Injection techniques are prone to erroneously high intramuscular pressure readings. Electromanometry and intramuscular pressure measurements have a significant learning curve.

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17 Measurements of Intramuscular Pressure during Exercise

INTRODUCTION

History and clinical signs alone are often insufficient to establish the diagnosis of chronic compartment syndrome.[1–3] However, they are valuable tools in selecting patients with chronic exercise-induced pain for intramuscular pressure measurements.[3,4] Therefore, measurement of intramuscular pressure during or at rest after exercise has an important role in diagnosis of the syndrome.

Measurements of intramuscular pressure during exercise are also useful in physiological research and in the study of muscle biomechanics.[1,5–12] They are also valuable to study limb function,[13–18] limb swelling during and at rest after exercise,[3,10] and force generation during muscle contraction.[5,7,19–22]

Different techniques for direct pressure measurements used in clinical situations are discussed in this chapter. Most of the methods have been thoroughly evaluated and are generally accepted for pressure measurements at rest but not for measurements during exercise. Every technique for intramuscular pressure measurement has its own advantages and disadvantages. Before a technique for intramuscular pressure recording is selected, it is important to determine the exercise protocol. Will the pressure be measured at rest or during exercise, or both? Will the patient exercise on an ergometer in the laboratory or run outdoors? What is the size of the compartment? The answers to these questions are helpful in selecting an optimal and suitable pressure-recording technique. The dynamic properties of a pressure-recording system are most important for accurate measurements of intramuscular pressures during exercise. Therefore, a substantial part of this chapter is focused on this issue.

DYNAMIC PROPERTIES OF PRESSURE-RECORDING SYSTEMS

Signals may be classified as static or dynamic. Static and dynamic properties of a pressure-recording system must fulfill certain criteria to allow for proper recordings of intramuscular pressures during exercise. All static nonlinearities discussed in Chapter XVI can also affect dynamic responses. Accuracy of an intramuscular pressure recording can be expressed as the difference between the true value and the measured value divided by the true value. The ratio is expressed in percent. The dynamic accuracy of a recording system is the fidelity with which the system reproduces a

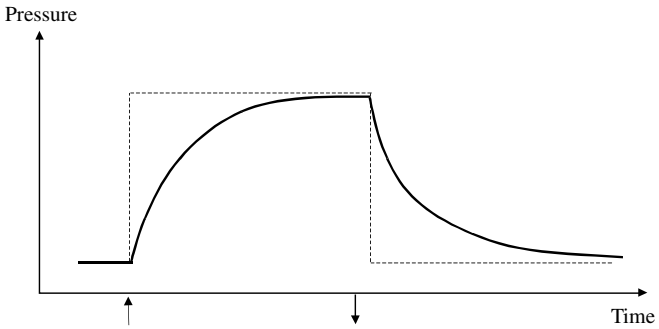

FIGURE 17.1 The dotted line indicates the true applied pressure (arrow up). The continuous line indicates the recorded pressure. The pressure rises slowly to the new value in overdamped recording systems. It decreases slowly to the initial value when pressure is released (arrow down).

dynamic event. Errors of the dynamic accuracy can have serious diagnostic consequences when intramuscular pressures are recorded during exercise in clinical practice and in research. For instance, if the pressure-recording system is overdamped, which means that the bandwidth is too low, physiological pressure changes during muscle contraction and muscle relaxation (that is, or pressure in the relaxed muscle between contractions) cannot be measured.[3,9] Therefore, chronic compartment syndromes cannot be diagnosed by pressure recording during exercise with such a technique.[9] However, they may be diagnosed by pressure recording at rest after exercise.

In overdamped pressure-recording systems, the recorded intramuscular pressure during a constant muscle contraction will rise slowly to the new value without oscillations (Figure 17.1). Overdamped systems are therefore unsuitable for recordings of fast dynamic events. They are also subject to time delay. One example of this is intramuscular pressure recording by the wick catheter system during dynamic muscular activity (Figure 17.2). As will be discussed next, they are also unsuitable for measurements of mean intramuscular pressures during repeated muscular contractions.

In underdamped recording systems, the pressure during a muscle contraction rises rapidly to the new value. It oscillates at its new damped natural frequency (Figure 17.3). The pressure oscillations fade away at a rate determined by the degree of damping in the system.

Several of the methods used for intramuscular pressure measurements at rest have been applied to measure pressures during exercise in clinical diagnosis[13,16,23,24] and to estimate muscle load in physiologic studies.[5,7,19–22] However, the dynamic properties of the pressure-recording system have been evaluated in only a few reports.[9,20,25] The dynamic properties must be known to permit proper interpretation of intramuscular pressure recordings during exercise. The dynamic properties of a fluid-filled catheter manometer system depend on catheter materials, lengths, inner diameters, number of connectors, and the compliance of the whole catheter manometer system. The interaction on the measuring system of the various parameters is rather complex. Therefore, all dynamic measurements of intramuscular pressure should be preceded by dynamic calibration of the method. The dynamic properties

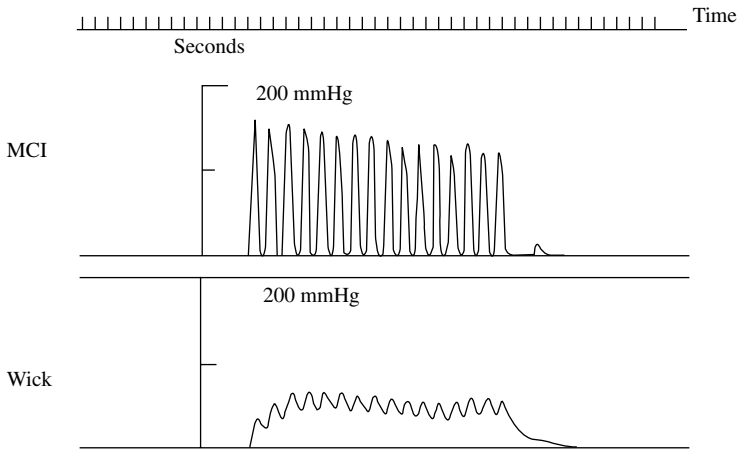

FIGURE 17.2 Intramuscular pressure recordings with a microcapillary infusion technique (MCI) (upper trace) and an overdamped noninfusion system with the wick catheter (lower trace) from the same spot in the tibialis anterior muscle of one patient. Recording by the wick catheter system does not allow for measurement of intramuscular pressure during muscle contraction or during muscle relaxation with a contraction frequency of 1 Hz. The rise time for the wick catheter may be up to several seconds.

of a pressure-recording system can be determined by measurements of rise time (or response time), the compliance, and the natural frequency (or resonance frequency) of the pressure-recording system.

Rise Time

The rise time (T_r) is used to estimate the dynamic properties of a pressure-recording system. The rise time can be calculated by the pressure step-function technique. The

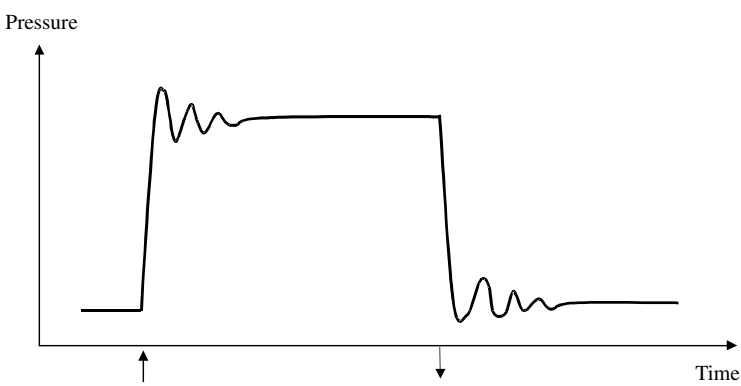

FIGURE 17.3 In underdamped recording systems, the signal will rapidly rise to the new value and oscillate around it. When pressure is released (arrow down), it will oscillate around the new value.

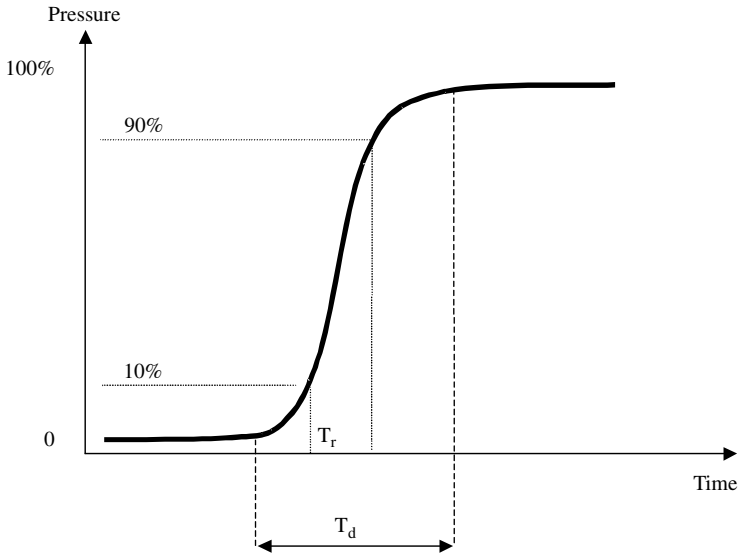

FIGURE 17.4 The rise time can be calculated by the pressure step-function technique. Rise time (*Tr*) for a pressure-recording system is defined as the time period required for the output signal to pass through the range of 10 to 90% of its final value. Time delay (*Td*) is the time period required for the signal to reach its final value.

rise time for a pressure-recording system is defined as the time period required for the output signal to pass through the range of 10 to 90% of its final value as a response to a step-like input signal (Figure 17.4).

If the overshoot of the response is less than a few percent and if the rise time (T_r) for a system is known, the frequency bandwidth (B) may be calculated by the following equation:

$$T_R \times B = k \tag{17.1}$$

where the constant k is 0.35. The wick catheter system is an overdamped system because the stranded wicks prevent rapid fluid flow over the tip of the catheter. It has a rise time of up to 4 sec.[9] By substituting this number into Equation 17.1, the frequency bandwidth will be about 0.1 Hz. This means that the duration of a contraction must be at least 5 sec followed by a relaxation of 5 sec to allow for an accurate measure of the contraction and relaxation pressures during exercise. This also implies that the maximum contraction frequency must not exceed 6 contractions/min (or 0.1 Hz).

The frequency bandwidth (B) is defined as the difference between the upper frequency (f_u) limit and the lower frequency (f_l) limit.

$$B = f_u - f_l \tag{17.2}$$

This means that the amplitude of the pressure curve is distorted and the readings are not accurate if the contraction frequency is higher than the upper frequency limit

or lower than the lower limit. Most authors recommend a bandwidth that is at least 10 times higher than the signal to be recorded. The frequency bandwidth of a recorder should always be greater than that of the signal.

Compliance of Pressure-Recording Systems

The compliance (C) of a transducer is the relation between the volume change (ΔV) and the pressure change (ΔP), given by Equation 17.3:

$$C = \Delta V/\Delta P \tag{17.3}$$

The compliance of the diaphragm is much higher than that of the saline-filled catheter or transducer cavity, provided the saline solution is bubble-free and the catheter material is relatively noncompliant.

A crucial part of the preparation for pressure recordings is filling the transducer and the transducer line with saline that is free of entrapped air bubbles and then keeping it free of air bubble formation. Even fine bubbles in the recording system increases the total compliance considerably and may decrease the bandwidth by decreasing the high frequency limit. Again, because air bubbles may be invisible, it is important to have some means for testing the dynamic performance after filling. If cold saline from the refrigerator is used, air bubble formation will increase in room temperature. The same is true when saline of room temperature in the catheter is exposed to the warm (35 to 38°C) skin and muscle tissue during exercise. Minimizing compliance is the greatest problem in obtaining good dynamic properties of the intramuscular pressure-recording system. One should look for air bubbles and leaky connections if the bandwidth of the recording system is low or the compliance is high. The compliance of the total intramuscular pressure-recording system, i.e., the catheter and transducer, should be less than 0.25 mm^3/100 mmHg.[9,26,27]

Natural Frequency

The natural frequency (F_n) is the frequency at which the pressure-recording system would resonate in the absence of any damping. The natural frequency (or resonant frequency) is influenced by length, radius, and compliance of the pressure-recording system. When a pressure applied to a pressure-recording catheter is suddenly changed from one level to another, the system will resonate at the damped resonant frequency. The natural frequency (F_n) of the system is given approximately by:

$$F_n = \tfrac{1}{2}\pi\sqrt{A^2/M \cdot C} \tag{17.4}$$

where A is the piston area of the transducer, M the fluid mass of the pressure-recording system, and C the compliance of the system. By Equation 17.4 it is possible to explain why a constant pump infusion technique has slower dynamic properties than those of the microcapillary system. The pump infusion technique has a significantly larger volume of saline that is fluid mass in the pressure-recording system.[9]

The dampening ratio is the natural logarithm of the amplitudes of the first and second oscillations in the response to the step function.[28,30] The duration of the oscillations depends on the damping. Optimal damping factor is about 0.7. The duration of the oscillations depends on the damping. The higher the bandwidth (or the natural frequency) of the system, the wider the range of frequencies over which the correct amplitude will be recorded. Also, movement artifacts of a catheter are amplified in a high-frequency response system.

In a recording system, pressure is transmitted nearly at the velocity of sound in water, which is about 1400 m/sec. Therefore, the time period it takes for a pressure wave to travel down the catheter and transducer line is less in relation to the period of the resonant frequency.

DYNAMIC TESTING OF PRESSURE-RECORDING SYSTEMS

A transducer is a device that converts energy from one form to another. As described in Chapter 16, transducers must be tested for stability, sensitivity, linearity, hysteresis, and volume displacement. The resonance frequency of the pressure-recording system may be used to judge dynamic properties of the system. This is most important because many factors, including different catheter materials and catheter dimensions and the experimental setup, may affect frequency response.[3,9,29] The ideal pressure-recording system should have a linear response, which is independent of frequency over the range of interest. Dynamic calibration of manometer systems can be tested in at least two ways, with techniques that generate pressure step response and sinusoidal pressure waveforms.[28,31]

Transient Step Response

The basis for this method is to apply a sudden step input to the pressure catheter and to record the output of the system (Figure 17.3). It is simple and most easy to perform. Performance of the pressure-recording system may be expressed as rise time in milliseconds as illustrated in Figure 17.4.

Sinusoidal Pressure Generator

Frequency response of the system is a more accurate method but is more complicated to perform and requires a sinusoidal pressure generator. The equipment to generate a sinusoidal pressure wave is complex.[31] Measurements with underdamped systems may give a hump in a frequency response–amplitude curve, as illustrated in Figure 17.5. The hump contains the resonant frequency.

It is important to have a high resonant frequency, at least 10 times or more than the highest frequency considered to be important in the original waveform. Generally a frequency response exceeding the event to be measured by up to 20 Hz is required to record the amplitude of the event accurately.[32] A frequency response greater than 40 Hz is required to limit errors to 10%.[32]

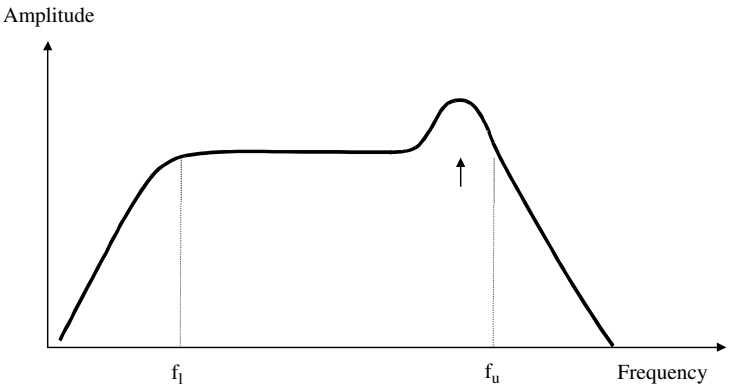

FIGURE 17.5 Measurement by an underdamped system gives a hump (arrow), which contains the resonant frequency in the frequency–amplitude curve; f_u is the upper frequency limit and f_l is the lower frequency limit.

Underdamped systems may be treated by partial clamping of the transducer line close to the transducer. The amplitude response should be flat without resonant peaks. This will optimize the system as regards a fast rise time and minimize distortion of the amplitude.

PRACTICAL RECOMMENDATIONS

Pressure-Recording Systems

The following steps are helpful in maintaining high dynamic properties of the pressure-recording system: (1) Using high-quality stopcocks and a minimum number of Luer locks. (2) Applying a known pressure and closing the stopcocks. If the pressure falls within a few minutes, then there is a significant leakage. (3) Testing transducers, amplifiers, and recorders for noise, linearity, stability, and hysteresis. (4) Conducting the transient dynamic test regularly at the end of the procedure. Most publications on measurements of dynamic intramuscular pressure recordings lack documentation of transient testing. (5) Applying damping of an underdamped catheter manometer system by constricting the transducer line close to the transducer with a binding screw.

Catheter Insertion Technique

Catheters for intramuscular pressure recordings during exercise must be inserted as atraumatically as possible. The introducer and the catheter should be inserted as parallel to the muscle fibers as possible and with the cutting tip of the needle retracted within the plastic sheath of the introducer (Figure 17.6). Once the introducer has penetrated the skin and the fascia, the tip of the needle should be withdrawn into the plastic sheath of the introducer. In this way, damage to the muscle fiber is minimized. Surrounding muscle fibers help guide the plastic sheath

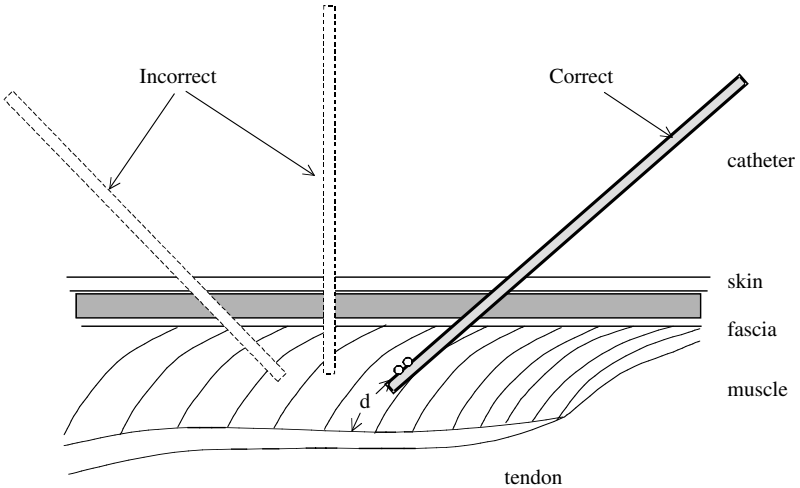

FIGURE 17.6 Catheters for intramuscular pressure recordings during exercise must be inserted parallel with the muscle fibers to allow for a pain-free investigation and minimize the risks for catheter bending. The catheter tip must not be inserted too close to the central tendon. The distance (*d*) between the tip of the catheter and muscle tendon must allow for at least a 20% muscle fiber shortening during contraction.

at insertion into the direction of least resistance, which is parallel to the fibers. Once the catheter is in place, this allows the muscle fibers to slide over the catheter tip in a parallel fashion during concentric and eccentric muscular activity. In this way, physiologic reactance to the measurement is minimized. That means that the patient or subject experiences less discomfort during muscular activity and will be able to exercise his/her muscles in a normal way without pain inhibition. Knowledge of the pennation angle and muscle fiber length is a prerequisite for a successful catheter insertion and a reliable measurement of intramuscular pressure during exercise.

Intramuscular pressure is a function of the depth of the catheter in the muscle. Depth is defined as the number of layers of muscle fibers that cover the catheter tip. In many cases, depth of the catheter is defined as the distance from the centrally located tendon within the muscle to the tip of the catheter, and not the distance from the skin or fascia.[33] Catheters should be placed in a standardized way to a depth that will allow the muscle fibers to shorten 20% of their resting length during contraction without the tendon hitting the catheter tip. If the tendon hits the catheter tip during concentric contractions, two problems may occur: (1) The patient may experience the muscle contraction as painful. In such cases, the patient cannot perform maximal contractions due to pain inhibition. Also, intramuscular pressure between contractions may be elevated because of the patient's inability to relax his/her muscles. (2) The tip of the catheter may become bent, which may create partial occlusion, which will decrease dynamic properties of the pressure-recording system. In rare cases the catheter may become completely occluded.[3,34]

Check of Catheter Position

Catheter position can be checked by sonography[35] or a function test.[36] By asking the subject to rotate different joints, different muscles in the compartment will be activated. Catheter location can be confirmed by the pressure response to these movements (Figure 17.5). External compression by a constant force by the investigator's fingertip in a proximodistal direction over the catheter tip may identify location of the catheter. The location of catheter tip can be determined by measuring the maximal pressure amplitude response to external compression by the investigator's fingertip (Figure 17.4).

Position of the Limb

Position of the subject, position of the joint over which the muscle acts,[35,37] and external compression from the underlying surface must be controlled because they affect intramuscular pressure. The heart level, which is defined as being 5 cm below the manubrium sterni in a supine subject, is a common reference point. One must be aware of the hydrostatic effects in sitting and standing patients as well as when they perform complex movements of a limb.

TYPICAL DISTORTIONS OF INTRAMUSCULAR PRESSURE RECORDINGS

In an underdamped system, the amplitudes of the higher-frequency components of the pressure wave are amplified. In such a system, it may be difficult to measure the exact pressure level during dynamic exercise. For an overdamped system, these higher-frequency components are attenuated. Overdamped systems such as the noninfusion system with a wick catheter show a time delay of up to several seconds (Figure 17.7).[9] The risk for partial occlusion of pressure catheter increases when noninfusion systems are used (Figure 17.8).[34] The transducer-tipped Camino catheter measures negative pressure values between concentric muscular contractions that are during muscle relaxation pressure.[25] This may be explained by the piston effect due to the fairly large size of the catheter. The large size of the catheter may create a negative interstitial fluid pressure as the muscle fibers slide over the tip of the Camino catheter (Figure 17.9).

Another explanation may be that the tip of the catheter was directed toward interstitial fluid flow direction during contraction and from the fluid flow direction during muscle relaxation. The fluid hydrostatic pressure sensed by a transducer varies with the direction of the fluid flow. The variation is due to the kinetic energy of the fluid. The total fluid pressure (P_{total}) per unit volume is defined by Bernoulli's equation:

$$P_{total} = P_0 + dgh + dv^2/2 \tag{17.5}$$

where P_0 is the static pressure with no flow, d the density (g/cm^3), g the acceleration due to gravity (g = 9.81 m/sec^2), h the height above the reference level, and v the

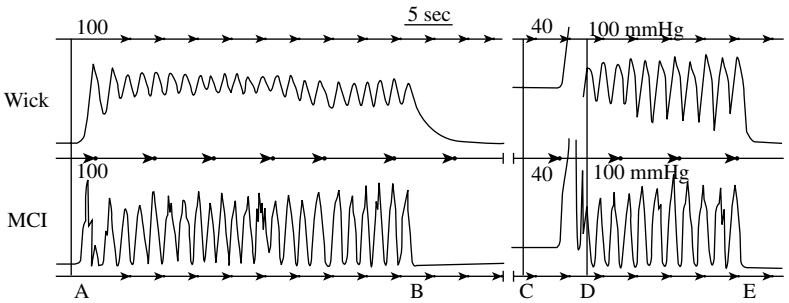

FIGURE 17.7 Intramuscular pressure recordings by the wick catheter (no infusion) (upper trace) and the microcapillary infusion techniques (infusion rate is 1 mL/h) (lower trace). Catheters are located next to each other in the same muscle. (A) Pressure recording during concentric muscular activity. Equilibrated pressures during muscle contraction and muscle relaxation cannot be obtained by the wick method due to the slow response. Recording by the wick catheter method is a frequency-dependent artifact. (B) It takes a longer time for the wick method to reach the equilibrated intramuscular pressure at rest after exercise. (C) Both catheters are flushed by 0.1 mL of saline. Pressure at rest in the flushed wick catheter is significantly increased. (D) The dynamic properties of the wick catheter system improve following injection, but not to the level of the infusion method. In this case, muscle contraction pressures are obtained because of the long duration of the muscle contraction time. However, the relaxation time is too short to allow for a proper recording of muscle pressures between contractions, i.e., muscle relaxation pressure. (E) Intramuscular pressures at rest after exercise.

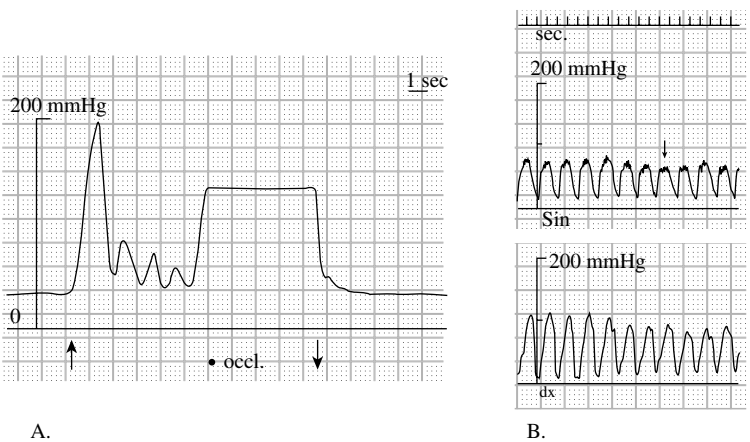

FIGURE 17.8 (A) Arrow up indicates start of contractions and arrow down indicates end of contractions. The pressure curve illustrates occlusion of the catheter tip. (B) Air bubbles in the pressure-recording system may lead to incorrect measurements of intramuscular pressure. The dynamic properties become impaired. The muscle contraction pressure in the upper trace is too low (arrow).

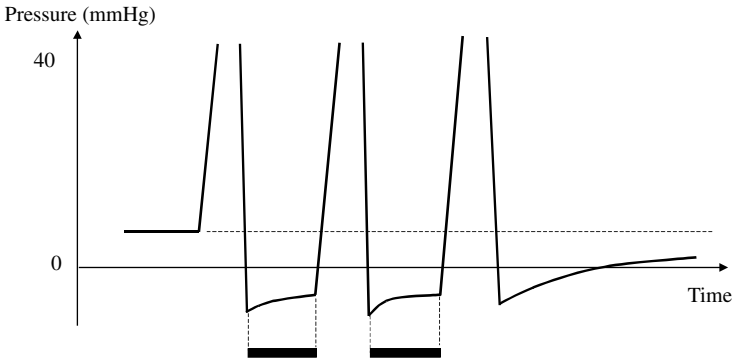

FIGURE 17.9 Intramuscular pressure recording by a fiberoptic catheter with a large diameter of the catheter tip. Pressure during muscle relaxation is negative initially due to a piston effect at the tip of the catheter. After a few minutes of muscular activity the effect disappears, possibly by normal edema formation around the tip of the catheter. The bars indicate the time period for muscle relaxation.

fluid velocity (m/sec). The Bernoulli equation may help to explain the difference observed between transducer-tipped catheters with the transducer at the distal end and the transducer at the side end of the tip. The area of the catheter tip in relation to the total fluid volume available around the tip may contribute to the piston effect and make the contribution of fluid energy by fluid flow more pronounced by large catheters without sideholes.

TECHNIQUES FOR INTRAMUSCULAR PRESSURE MEASUREMENTS

Different techniques for direct pressure recordings may be classified as injection techniques, infusion techniques, noninfusion techniques, transducer-tipped techniques. Most of the methods have been thoroughly evaluated and are generally accepted for intramuscular pressure recordings at rest.

The needle injection technique can be used to measure pressure at rest only.[38–41] This technique has been shown to be less accurate than the other techniques for pressure recordings,[40,42–44] especially if pressure recordings are made during injection.[44] The injection technique measures the tissue's resistance to fluid flow and gives an artificially high reading if pressure is measured during injection. However, a needle or catheter with one or multiple sideholes at its tip reduces the risk of recording artificially high pressures, especially when pressures are recorded by the meniscus method following an injection.[44]

Infusion Techniques

The constant pump infusion technique[27] and the nonconstant microcapillary infusion technique[9] are both suitable for recording of intramuscular pressures at rest and during exercise.[9,16,45,46] The Intracath® and Myopress® catheters are fluid-filled sys-

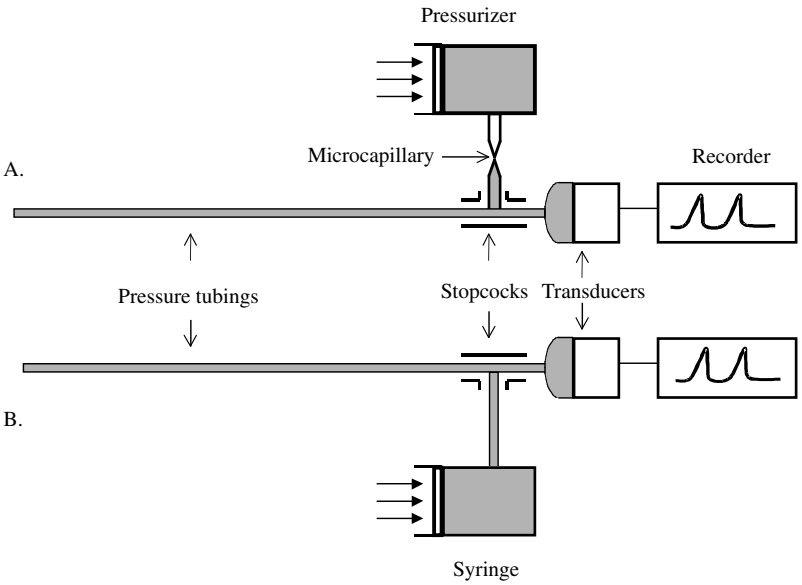

FIGURE 17.10 The microcapillary infusion (A) and the pump infusion (B) methods. The main difference between methods is the volume of the pressure-recording system, which is much larger with the pump infusion method.

tems with extracorporal transducers. They are both specially designed with multiple sideholes at their tip, which make them suitable to be used with infusion (Figure 17.10). However, they may be used without infusion as well. Both infusion techniques can be combined with different types of catheters, e.g., the Intracath®, Myopress®, Slit® and STIC® catheters.[9,27,46] The saline volume of the pressure-recording system is significantly less in the microcapillary infusion system compared with that in the pump infusion system. This helps improve the dynamic properties of the microcapillary infusion system because a smaller volume of saline, i.e., a smaller weight, has to be displaced in the measuring system.

The resonance frequency of the microcapillary infusion system is 27.8 Hz compared to 7.7 Hz with the pump infusion system, as determined by the pressure step-function technique.[9] Applying the sinusoidal pressure generator resulted in resonance frequencies of 31 and 9 Hz, respectively. During a 3-h period of pressure recording, the resonance frequency fell from 31 to 17 Hz with the microcapillary infusion technique, and from 9 to 6 Hz with the pump infusion technique, due to formation of small air bubbles in the transducer line.[9]

The magnitude of the infusion rate is important. The compliance of muscle tissue decreases during muscle contraction. Therefore, the risk of reading artifactually high muscle contraction pressure with the microcapillary infusion technique is less than with the constant pump infusion technique. The reason for this is that the infusion rate with the microcapillary infusion technique is minimal or even zero during muscle contraction (Figure 17.11). The compliance of muscle tissue at rest is high.[47,48] Intramuscular pressure is fairly constant when 0.1 to 3.0 mL/h of saline

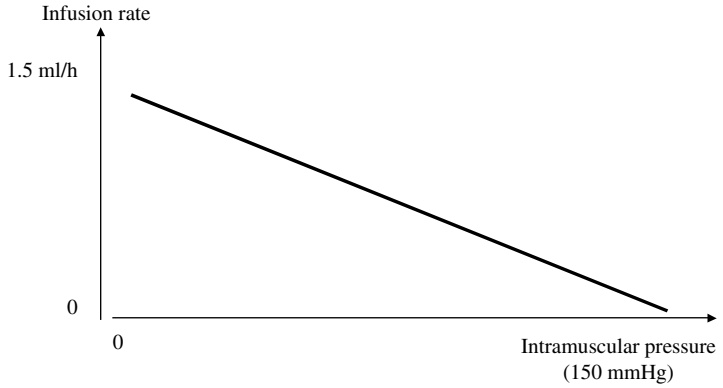

FIGURE 17.11 The relationship between intramuscular pressure and infusion rate with the microcapillary infusion technique. When intramuscular pressure approaches 150 mmHg, the infusion rate is close to zero. It is 1.5 mL/h when intramuscular pressure is atmospheric.

is infused.[9,11] However, infusion rates exceeding 0.2 mL/h are often unnecessary for pressure recordings during intermittent exercise if a Teflon catheter with an open tip and multiple sideholes at its tip is used. The microcapillary infusion technique has been combined with a Slit catheter,[34] wick catheter,[9] STIC catheter,[8] Intracath,[27] and a Myopress catheter.[9–11,22]

Noninfusion Techniques

Different non-infusion techniques[40,43] have been used to record pressures at rest and during exercise.[2,13,49,50] However, it has been shown that the wick catheter method is unsuitable for recordings of intramuscular pressure during exercise because of its poor dynamic properties.[9,20] Pressure values during muscle contraction and muscle relaxation depend on the contraction frequency as illustrated in Figure 17.12, as well as on the relative time duration of muscle contraction and muscle relaxation. Pressures during exercise were not accurately recorded with a slit catheter because the tip of the catheter occluded.[34] For this reason, slit catheters must be flushed repeatedly when pressures are recorded with a noninfusion technique.[1,20,34,45] In this situation, the device is no longer to be considered a noninfusion technique but rather an intermittent injection technique. Figure 7.7 illustrates the improved dynamic properties of noninfusion systems following flushing of catheters. Furthermore, the tip design of the slit catheter has been suggested to be more traumatic to the local tissue during muscular activity.[34] It has been described how the slits at the tip of the catheter become deformed following intramuscular pressure measurements during exercise. The tip design of Myopress and Intracath catheters make them suitable to be used with noninfusion as well.

Transducer-Tipped Catheters

Pressure-recording systems that are fluid filled require flushing or infusion to maintain accuracy.[9,27,34,43,46] These methods are equipped with external transducers and

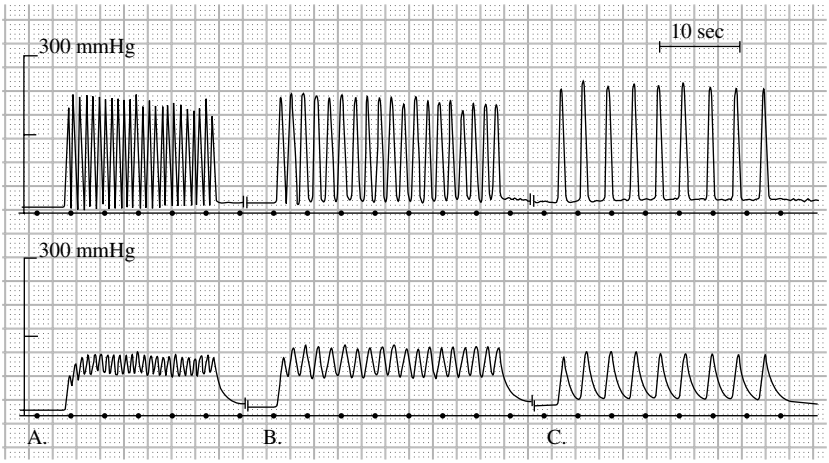

FIGURE 17.12 Muscle contraction pressure and muscle relaxation pressure, i.e., pressure between contractions in a relaxed muscle, during constant muscle load at (A) 75, (B) 37 and (C) 18 contractions/min. The readings in the upper trace obtained with a microcapillary infusion technique show the same magnitude of pressure values, independent of the contraction frequency. In the lower trace, the readings depend on the contraction frequency, because of the prolonged rise time of the noninfusion system (Redrawn from Styf, J.R. and Körner, L.M., *Clin. Orthop. Rel. Res.,* 207, 253, 1986.)

are thus prone to hydrostatic artifacts when the limb position changes relative to the level of transducer.

Transducer-tipped catheters may be classified as fiberoptic or solid state. These catheters measure the pressure at the point at which it occurs. All the problems and pitfalls that are associated with transmission of pressure through the transducer line to an external transducer are eliminated. Transducer-tipped catheters are suitable for recordings of muscle contraction pressure during complex movements of the extremity because they eliminate any problem of the changing hydrostatic column during such movements. The catheters are also suitable for recordings of pressures at rest after exercise. However, if the catheter has a large diameter, it is not recommended for recordings of muscle relaxation pressure during exercise, because of the piston effect.[25] This effect gives negative pressure values during muscle relaxation, possibly by the vacuum from sliding fibers over the tip of the catheter. Catheters with a large diameter are more prone to this effect. However, the effect diminishes after 5 to 10 min of exercise. The reasons for negative pressure recordings have been discussed previously in this chapter.

Fiberoptic Catheters

The fiberoptic transducer-tipped system for intramuscular pressure measurement was modified by Crenshaw et al. for measuring intramuscular pressure at rest[51] and during exercise.[25] They showed that the fiberoptic system (Model 420, Camino Laboratories, San Diego, CA) is most suitable to measure pressure during muscle contraction especially when complex limb movements involve changes relative to the horizontal

plane. The fiberoptic catheter is enclosed in a sheath with an outer diameter of 2.1 mm filled with saline and with 12 sideholes at the tip. The contact area between muscle tissue and the catheter tip is 9.6 mm^2 and the compliance of the system is 5×10^{-6} mL/mmHg. The volume of the catheter is 157 mm^3.

Optical transducers measure displacement of the diaphragm by optical means. The main advantage of the new fiberoptic catheter-tipped pressure transducer is the small size and the absence of electricity within the catheter, which makes it safer than other systems. The magnitude of temperature drift of fiberoptic catheters is small.[52]

Other fiberoptic pressure sensors are based on silicon micromechanics. The sensor body, which can be an interferometer of silicon, is placed on the end of an optical fiber. The sensor measures the modulation of light intensity, which occurs by interference in the cavity when its depth is of the same order as the wavelength of the light in the fiber. The technique is based on the mechanical properties of silicon. It has a volume displacement of 0.5×10^{-3} mm^3/100 mBar. The diameters of these catheters can reach 0.3 to 0.5 mm, which is a suitable size for biomedical use.

STIC Catheter

The solid-state intracompartmental (STIC) catheter is designed with an internal silicon semiconductor strain gauge transducer.[46] A multiperforated noncompliant polyethylene tubing (PE260) is tightly fitted over the tip of the transducer diaphragm. The cross sectional area for fluid flow through the perforations is 1.4 mm^2. A curved plastic sleeve with a troachar is used to insert the catheter in the muscle. The plastic sleeve is 10 cm long and the inner diameter is 2.3 mm. The STIC catheter combines the advantages of being transducer-tipped and has the possibility being infused. The infusion can be both a constant pump infusion and a microcapillary nonconstant infusion technique.

Solid-state transducers are based on silicon chips. In contrast to fiberoptic catheters, they do not require a specialized amplifier. They can be connected to the patient monitor via an inexpensive interface unit. Generally, they have fairly low zero-drift good frequency response and stable linearity. One drawback is that they cannot be rezeroed after implantation. A normal drift of 5 mmHg during 8 h is considered to be normal for both fluid-filled and transducer-tipped systems.

Codman and Kodiag Catheters

The Codman microsensor transducer (Codman, Johnson & Johnson, Raynham, MA) was developed clinically for intracranial measurements. It has been modified and tested for intramuscular pressure recordings.[53] The catheter is small and flexible and made of nylon tubing, which makes it suitable for intramuscular pressure measurements during exercise. The dynamic properties of the system are high.

The transducer element is based on a silicon chip with diffused pressure-sensitive resistors forming a bridge. It consists of an integrated strain gauge hosted in a titanium case and placed close to the tip of a Teflon® catheter with a diameter of 0.7 mm. The membrane of the pressure transducer is countersunk in the catheter to avoid artifacts due to mechanical contacts with the surrounding tissue. The pressure transducer is electrically connected to a digital intracranial pressure monitor with

fine wires passing through the catheter. The working range is –50 to 250 mmHg. The stability of the microsensor is good. The long-term drift is less than 0.8 mmHg/24 h and the temperature drift is less than 0.03 mmHg/°C. The frequency response is excellent. The Codman catheter is also suitable for pressure measurements during exercise. It withstands mechanical stress exerted on the tip of the catheter during muscle contractions. Artifacts superimposed on the true intramuscular pressure signal due to locomotion or vibration of the extracorporal part of the catheter are eliminated. Another advantage of the Codman catheter is that it has a smaller diameter than the fiberoptic transducer-tipped catheter. Patients do not report as much discomfort during intramuscular pressure recordings during exercise by this catheter.

The Kodiag catheter, described in detail in Chapter 16, may also be used for intramuscular pressure recordings during exercise.

REFERENCE VALUES FOR INTRAMUSCULAR PRESSURES

The following values of normal and abnormal intramuscular pressures in humans refer to the supine position and with the tip of the catheter at the heart level.

Intramuscular Pressure at Rest before Exercise

Normal Values

Normal pressures in most muscles vary between 2 and 8 mmHg[3,9,16,40,45,54–57] but pressures up to 10 to 15 mmHg have been reported.[2,8,58] The recorded pressure depends on the method used, the position of the leg,[35] and the depth of the catheter in the muscle.[33,59] Intramuscular pressures at rest are higher when injection or infusion techniques[9,43] are used or when the volume of the pressure-recording catheter is large.[8]

Abnormal Values

Intramuscular pressure at rest before exercise is high in patients with chronic compartment syndrome.[2,3,9,16,57,58,60] However, the wide range of pressures at rest and their dependence on the position of the ankle joint and the depth of the catheter in the muscle, as well as the position of the subject, make the use of only this parameter unreliable in the diagnosis of chronic compartment syndrome.[33,35,59] Also, pressure at rest before exercise may increase by active muscle tension from pain and by external compression by the underlying surface. For these reasons, the use of this pressure parameter alone is not recommended.[3,4,29]

Muscle Contraction Pressure during Exercise

Muscle contraction pressure during exercise is an indicator of force generation of that muscle.[5,7,19–22] Recording of muscle contraction pressure requires good dynamic properties of the pressure-recording system.[8,9,11,22,25,61] The upper traces of Figure 17.2 and lower trace of Figure 17.7 illustrate measurement of muscle contraction pressure during

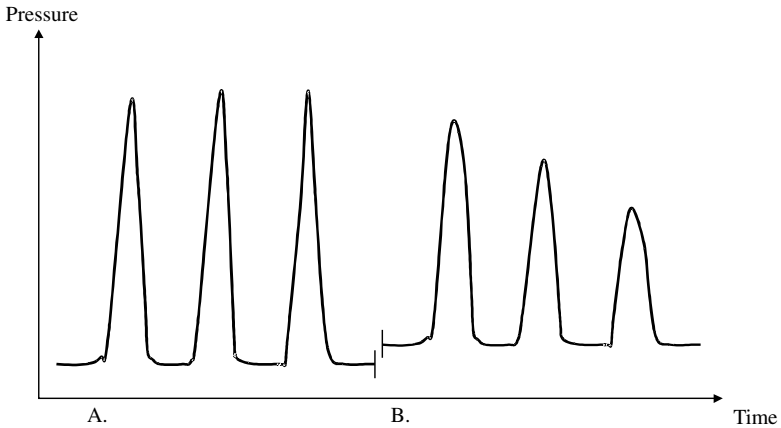

FIGURE 17.13 (A) Normal pressure recording during exercise. (B) Muscle contraction pressure decreases at the end of exercise due to muscle fatigue. Muscle relaxation pressure remains elevated due to the increased volume of the muscle.

exercise. Pressures during muscle contraction ranging from 100 to 250 mmHg have been recorded in the anterior tibial and other muscles.[3,5,6,8,9,12,19,20,22,25] This pressure parameter is not directly related to the pathophysiology of chronic compartment syndrome and should not be used to diagnose the syndrome. Muscle contraction pressure is often low at the end of exercise because the force generation decreases due to muscle fatigue or pain-induced muscle weakness (Figure 17.13).

Muscle Relaxation Pressure during Exercise

Normal Values

The magnitude of muscle relaxation pressure during exercise, i.e., the pressure between contractions, depends on the volume load of the muscle. Muscle relaxation pressure increases for two reasons. Postexercise edema increases the volume of the muscle and the regional muscle blood flow increases, both of which increase intramuscular pressure. Therefore, intramuscular pressure normally increases from about 5 mmHg before exercise in the supine subject to 10 to 25 mmHg after exercise[3,9,10,12,62,63] depending on the muscle studied. In an upright subject, the muscle relaxation pressure during exercise is lower than the pressure at rest after exercise.[17] The reason for this is the effect of the calf muscle pump, which decreases blood volume in the leg following muscular contractions. At rest after exercise, the intravenous hydrostatic column elevates the interstitial hydrostatic pressure in the leg. Recording of muscle relaxation pressure requires good dynamic properties of the pressure-recording system.[8,9,11,22,25,61]

Abnormal Values

Muscle relaxation pressures exceeding 35 to 55 mmHg correlate well with the development of pain, swelling, and impaired muscle function in patients with chronic

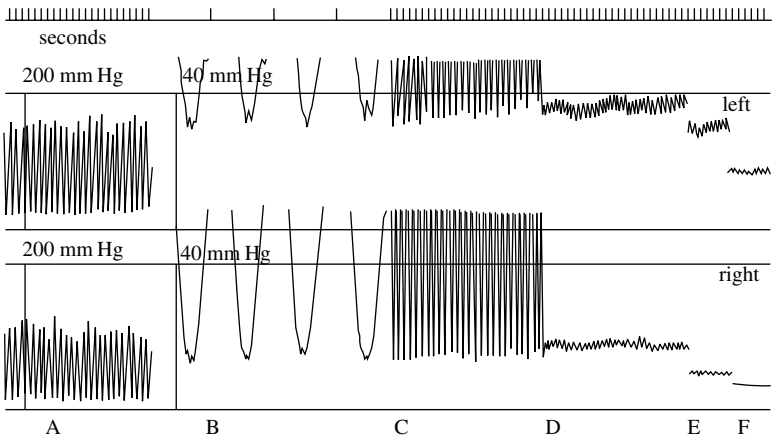

FIGURE 17.14 Results of intramuscular pressure recordings from one patient with (upper trace) chronic compartment syndrome and one without (lower trace) the syndrome. Muscle contraction pressure and muscle relaxation pressure can be read at (A). By changing the standard for intramuscular pressure from 200 to 40 mmHg, muscle relaxation pressure can be read with higher accuracy (B). At rest after exercise, intramuscular pressure and the amplitude of pressure oscillations are significantly greater in compartment syndrome (D). The elevated pressure is not normalized at 6 min (E) or at 10 min (F) after exercise.

compartment syndromes,[3,4,29] to decreased muscle blood flow[10] and decreased tissue oxygenation in these patients.[64] This pressure is related to the pathophysiology of the syndrome and is the best pressure parameter to study during exercise in patients with the chronic compartment syndrome (Figure 17.14). However, at the end of exercise, some patients are not able to relax their muscles completely between contractions because of muscle pain or muscle fatigue, or both. Therefore, muscle relaxation pressure should always be related to and compared with intramuscular pressure at rest after exercise (Figure 17.15). If these two pressures are at fairly similar levels, many pitfalls can be avoided. Examples of such pitfalls are increased muscle relaxation pressure due to inability to relax muscles or a high muscle contraction frequency.[3]

Mean Muscle Pressure during Exercise

The mean muscle pressure (MMP) is calculated from the muscle contraction pressure (MCP) and muscle relaxation pressure (MRP) by adding one third of the pulse pressure to the relaxation pressure according to Equation 17.6:

$$\text{MMP} = \text{MRP} + 1/3(\text{MCP} - \text{MRP}) \tag{17.6}$$

This means that MMP depends on the MCP and MRP.[22] Therefore, MMP is secondary to changes of both these pressures. MMP exceeding 50 to 85 mmHg have been used as criteria in diagnosing chronic compartment syndrome.[46,50] However, by definition, MMP values depend on both contraction and relaxation pressures.

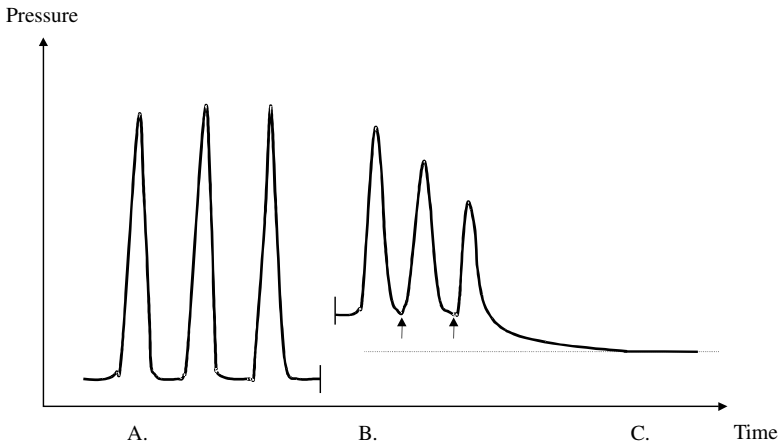

FIGURE 17.15 (A) Muscle contraction and relaxation pressures during exercise. At the end of exercise (B), the subject may have difficulties in relaxing their muscles. These patients have elevated muscle relaxation pressures and elevated intramuscular pressure at rest after the exercise test. The true muscle relaxation pressure was probably at the same level as the dotted line.

Therefore, the mean pressure is secondary to changes of both these pressures. MMP is an unreliable and physiologically unrelated parameter to use in diagnosis of chronic compartment syndrome.[3,9,10,65] The level of MMP, measured by methods with slow dynamic properties, depends also on the relative duration of the contraction and relaxation. The longer the duration of the muscle contraction is, the higher the MMP will be (Figure 17.16). Contraction cycles of short duration followed by a long muscle relaxation time give low mean muscle pressure values. Such recordings are artifacts from pressure-recording systems with slow dynamic properties that are due to the contraction frequency and relative duration of the muscle contraction and relaxation time. Overdamped pressure-recording systems with an increased rise time, such as the wick catheter system and other noninfusion systems, are prone to cause this artifact. This is also true for any catheter system that becomes partially occluded.

Intramuscular Pressure at Rest after Exercise

Normal Values

Intramuscular pressure immediately at rest after exercise is normally between 10 and 25 mmHg, which is fairly equal to the MRP at the end of exercise in a supine subject. This pressure parameter depends on the volume load of the muscle. Intramuscular pressure returns to preexercise levels within 5 to 10 min.[3,40,46,57,58,60,63,66]

Abnormal Values

Pressures exceeding 30 to 35 mmHg at rest after exercise and a time period exceeding 6 to 10 min to normalize the increased pressure have been used as criteria to diagnosis the syndrome.[2,3,9,16,60] These parameters are useful if MRP during

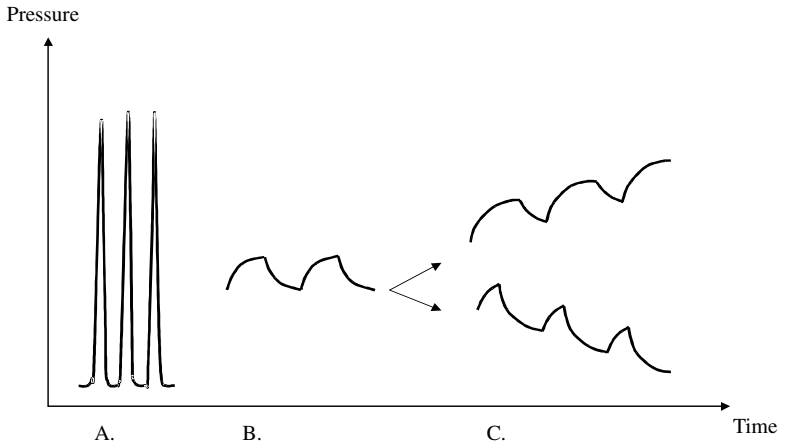

FIGURE 17.16 Redrawing from an intramuscular pressure recording during exercise with (A) an infusion system with good dynamic properties. Pressures during muscle contraction and muscle relaxation can be recorded. The pressures obtained by a recording system with slow dynamic properties like some non-infusion fluid-filled systems depend on the relative duration of the contraction. At (B), the contraction and relaxation times are equal. At (C) (upper trace) the contraction duration is about 80% of the cycle. Therefore, the recorded pressure increases closer to the true muscle contraction pressure value. At (C) (lower trace) the contraction duration is about 20% of the cycle. Therefore, the recorded intramuscular pressure decreases closer to the muscle relaxation pressure.

exercise is not recorded. Increased pressure by active muscle tension due to pain must be excluded, and external compression by the underlying bed must be avoided. There is a slight risk of making a false diagnosis of chronic compartment syndrome if the pressure at rest after exercise is used as the only diagnostic criteria.[3] It takes more than 10 to 20 min to normalize abnormally elevated intramuscular pressure at rest (Figure 17.14).

Patients with muscular hypertension syndrome have increased intramuscular pressure at rest after exercise and normal MRP during exercise.[3] The reason for this is their inability to relax their muscles at rest after exercise possibly because of pain (Figure 17.6). Following the results of pressure monitoring, this condition has been labeled "muscular hypertension syndrome."[3] Simultaneous measurements of muscular activity by electromyography (EMG) and intramuscular pressure from the same spot will help differentiate between the two conditions. In patients with chronic compartment syndrome, the pressure increase depends only on the volume load of the compartment. The EMG signal is silent in these patients. On the contrary, patients with muscular hypertension syndrome have increased intramuscular pressure at rest after exercise and a positive EMG signal.

Intramuscular Pressure Oscillations at Rest after Exercise

Increased amplitude of pulse-synchronous oscillations of intramuscular pressure at rest after exercise can normally be recorded when the muscle is swollen.[3,9,29,37] The

pressure amplitude at rest after exercise is significantly higher in patients with chronic compartment syndrome (Figure 17.14). This is due to decreased compliance of the compartment. Pressure amplitude varies with the volume change induced by each arterial pulse and compliance of the compartment.

ESTIMATION OF FORCE GENERATION AND MUSCLE LENGTH BY INTRAMUSCULAR PRESSURES

Force Generation Estimated by Intramuscular Pressure and EMG

Tensile force is transmitted to the muscle tendon during muscular activity. Stress from each muscle fiber increases intramuscular pressure.[20] Figure 17.17 illustrates the pennation angle alpha, which is the angle between the muscle fiber and the tendon. It shows the magnitude and direction of forces generated at the end of each type of muscular activity. The parallelograms show that the tensile force at the end of eccentric activation of a muscle fiber is transmitted to the tendon at a relatively higher proportion. The stress that generates the interstitial hydrostatic pressure is lower compared to end concentric activity when the fiber is shorter. The reason for this is the lower pennation angle at the end of eccentric muscular activity compared with the end of concentric activity.[22] The quota between intramuscular pressure and torque generation during eccentric muscle contractions is lower than during concentric muscle contractions. The reason for this is the relatively unchanged intramuscular pressure and increased force generation during eccentric muscular activity.[21,22]

An almost spherical muscle with a large fiber curvature, i.e., with a short radius, transmits less force to the tendon than a straight fiber does. Most of the tensile forces are transformed into stresses that are transmitted into muscle tissue as a high inter-

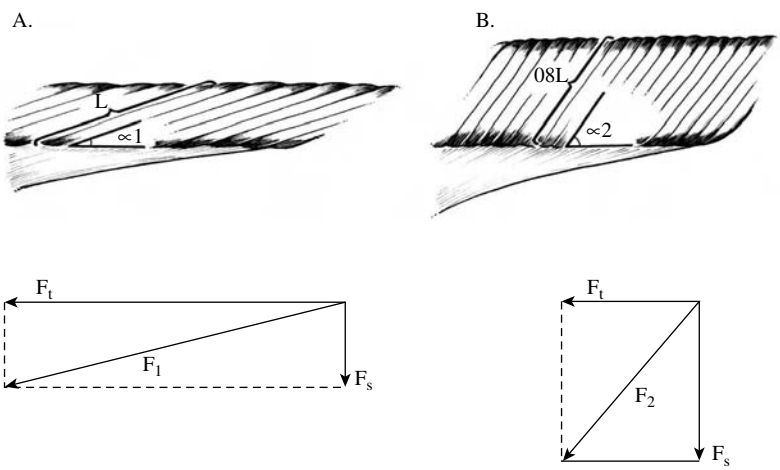

FIGURE 17.17 A muscle contraction at the end of eccentric contraction (A) and concentric contraction (B). The figures are parallelograms illustrating the tensile forces transmitted to the tendon (F_t) and the compressive forces (F_s), which generate the intramuscular hydrostatic pressure. F_s is the stress that creates the intramuscular pressure during contraction.

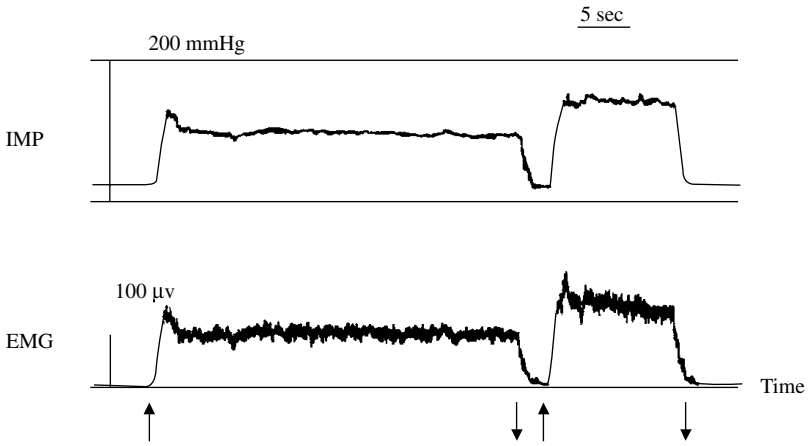

FIGURE 17.18 The close correlation between intramuscular pressure (upper trace) and the EMG signal (lower trace) from the same muscle during contraction.

stitial hydrostatic pressure. One example of such an extreme muscle is the heart muscle as a muscle pump. The heart muscle creates intravascular fluid hydrostatic pressures that are high enough to maintain tissue perfusion.

EMG has been used extensively as a technique to monitor force output during isometric contraction. Some studies report a linear relationship between force and EMG during isometric contraction,[67] whereas other studies report nonlinear.[68,69] The use of EMG as an indicator of contractile force during nonisometric exercise poses additional problems. EMG is insensitive to changes in fiber length and contraction velocity. EMG represents electrical excitation of muscle rather than its intrinsic mechanical properties and is therefore reported to be unsuitable for monitoring specific muscle function during dynamic exercise. Aratow and co-workers showed that intramuscular pressure provides a more linear index of contraction force than EMG does for the soleus and tibialis anterior muscles during concentric and eccentric activity.[5] Figure 17.18 illustrates the close relationship between intramuscular pressure and EMG during muscular activity.

Force Generation and Muscle Length

Adjustment of muscle tension is important at tendon transfer and free muscle transfer. It may be difficult to manually assess the proper tension of the muscle tendon complex during surgery. Measurement of intramuscular pressure is one possibility to optimize the tension of a transferred muscle. Another possibility is to measure the sarcomere length. The goal is to keep the optimal relationship between excursion and sarcomere length to save the optimal force generation from the fibers and thereby joint torque.

SUMMARY

Dynamic properties of a pressure-recording system are important for accurate measurements of intramuscular pressures during exercise. Catheters for pressure recordings

should be inserted parallel with muscle fibers to minimize physiological reactance. Bent catheters, partial occlusion, external compression, patient and joint position, as well as catheter depth in the muscle can result in erroneous pressure recordings. MRP and intramuscular pressure at rest after exercise are measures on the volume load of the muscle. They are the most reliable pressure parameters to study in the diagnosis of chronic compartment syndrome. MCP is a measure of the force generation from the muscle. MMP during exercise is a physiologically unrelated parameter in the diagnosis of the syndrome. Neither MCP nor MMP can be used in the diagnosis of chronic compartment syndrome. Measurements of intramuscular pressure during exercise also include a significant learning curve. It is an art of electromanometry. A safe diagnosis of chronic compartment syndrome can be established by combining clinical examination of the patient following an exercise test that elicits the symptoms with intramuscular pressure studies.

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18 Limb Edema

INTRODUCTION

Edema is defined as an abnormal accumulation of fluid within the interstitial space. It is a pathological condition and a sign of disturbed microcirculation. The formation of interstitial edema may alter the tissue's nutrition and viability. It may contribute to tissue hypoxia by increasing the distance that oxygen must diffuse from the capillary to the cell (Figure 18.1). Another harmful effect of edema is its influence on the magnitude of fluid transportation over the capillary bed by affecting the Starling equilibration as discussed in Chapter 1. Edema increases the interstitial fluid hydrostatic pressure. Therefore, the local perfusion pressure may decrease. Patients with limb edema may experience discomfort and pain.

EDEMA FORMATION

Edema of a limb is seen by clinical means when the weight of the limb increases by ca. 10% above normal.[1] An exsanguinated human limb will swell by ca. 10% of its original volume after release of a pneumatic tourniquet.[2] This compares to a 5% increase of limb circumference. The reason for this is the combined effects of reactive hyperemia and subsequent swelling. During reactive hyperemia, the intravascular volume further increases. The subsequent swelling is caused by increased interstitial volume. Following vascular reconstruction, leg volume may increase by up to 26% on the operated side.[3] The interstitial swelling is due to increased microvascular permeability and the late phase of swelling is due to impaired lymphatic drainage.

In the clinical setting, edema is seen after major orthopedic and vascular surgery to the extremities, burn injuries, and following fasciotomy due to acute compartment syndrome. Other reasons for edema formation are different conditions of venous hypertension. Edema is commonly seen in patients with reperfusion syndromes. Limb edema can worsen reperfusion injury by increasing vascular resistance and the distance for oxygen diffusion. Reperfusion of ischemic limbs is associated with a high amputation rate (12 to 22%). Only 60 to 70% of the extremities are functionally intact after a reperfusion injury.[4] Immobilized extremities are also prone to edema formation.

Edema formation can result from disturbances originating in the microvascular bed, the interstitial space or the lymph vessels. The mechanisms for edema formation may therefore be classified as vasogenic edema, myxedema, or lymphedema.

Vasogenic Edema

Capillary hypertension, low intravascular oncotic pressure, and alterations of the microvascular barriers may cause vasogenic edema.

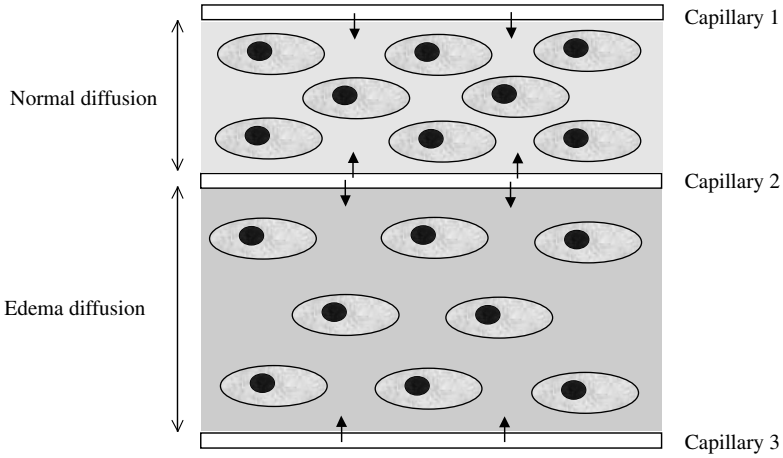

FIGURE 18.1 Effect of edema on oxygen diffusion. Edema increases the distance that oxygen must diffuse through the interstitial space from the capillaries to the cells.

Capillary Hypertension

The most common reason for increased capillary pressure in the trauma situation is abnormally elevated venous pressure due to obstruction of venous blood flow. Increased capillary pressure due to arterial hypertension is uncommon. Only 10% of the arterial pressure increment is transmitted to the capillary level.

Venous stasis induces edema formation[5,6] and may even induce acute compartment syndrome in the leg.[7,8] Venous hypertension is an important factor for the development of acute compartment syndrome in the lower limbs.[9–11] Venous return from the lower limb may decrease by elevated intraabdominal pressure,[12,13] by injuries to the pelvis and proximal thigh[14] as well as by extensive thrombosis.[15] Gross edema may occur following elevation of venous pressure by 10 to 15 mmHg.

Low Intravascular Oncotic Pressure

Hypoproteinemia is also an uncommon reason for edema in trauma cases. It may be a contributing factor in patients with cirrhosis who have a low protein synthesis and in patients with nephrosis who have a rapid escape of plasma proteins.

Alterations of Microvascular Barriers

Tissue injury induces the release of many vasoactive substances, which may alter vascular tone and endothelial cell function. Edema may be created by increased capillary filtration following ischemic damage to the endothelial cells.[16] In these conditions, a widening of the interendothelial clefts of the capillary bed occurs. The pore widening reduces the protein reflection coefficient of the Starling equilibrium. The osmotic pressure gradient over the capillary, which is the difference between intravascular osmotic pressure and interstitial osmotic pressure, falls suddenly, and large volumes of fluid are accumulated in the interstitial space.

Edema following inflammation is caused by a number of vasoactive substances, which may alter the vascular tone and the endothelial porosity. A 50% increase of the pore diameter induces a 100% elevation of the interstitial water content.[17] The capillary filtration coefficient may increase fourfold in myocitis.[18]

Myxedema

In myxedema, the amount of interstitial water and matrix is increased. The condition may thus be regarded as hypertrophy of the interstitial space. The water and the interstitial space become organized, which leads to the clinical condition of nonpitting edema. An excess of tyrotropin may induce the condition. Myxedema is also observed in patients with primary hypothyroidism and hyperthyroidism secondary to hypersecretion of tyrotropin.[17] Patients with Graves' disease may have pretibial myxedema. They have a firm bulging over the anterolateral aspect of the leg proximal to the lateral malleolus. Patients with a peroneal nerve tunnel syndrome, or entrapment of the superficial peroneal nerve, may also have a similar anterolateral swelling which is edema of pitting type.[19,20]

Lymphedema

Lymphedema may be caused by obstruction of lymph flow.[21] It occurs when lymph flow is reduced to less than 50%.[17] Impaired lymph flow may be caused by external compression that obstructs vascular return from a limb, or by destroyed lymph vessels, e.g., after dissection during vascular surgery.[22,23] Edema of the leg is commonly observed after arterial reconstruction of the lower limbs. Several reasons for this are possible, as discussed previously. Reduced lymphatic drainage due to intraoperative damage to lymph vessels is an additional possibility.[22,23]

EDEMA-PREVENTING MECHANISMS

Increased tissue hydrostatic pressure and decreased oncotic pressure in the interstitial space are two edema-preventing mechanisms.[5] The third edema-preventing factor consists of complex interactions of lymph formation, hydraulic permeability of the interstitial space, filling hydrostatic pressure of the prelymphatic space, and return of lymph fluid by the lymphatic vessels. Lymph flow is determined by the magnitude of capillary filtration. Hydraulic permeability of the interstitial space is a measure of how easily fluid flows through the interstitial space. Filling pressure in the prelympathic space is a parameter that depends on interstitial hydrostatic pressure. Finally, the ability of lymph vessels to transport the lymphatic fluid is important to prevent lymphedema.[24,25]

Competent intraluminal valves prevent retrograde lymph flow in most tissues. However, the lymph vessels in muscle tissue have no contractility and probably lack valves in the collecting ductuli.[26] Lymph flow from skeletal muscle therefore depends on active and passive muscular action. Contraction of skeletal muscle may empty a lymph vessel.[21] During muscle relaxation or when the extravascular compression is released, the intraluminar pressure decreases because of a rebound in the walls of

the lymph vessels. This creates a suction effect that possibly is transmitted to the prelymphatic space.

EDEMA-REDUCING METHODS

Limb edema may be reduced by concentric muscular activity, passive stretch of muscle, external compression of the limb, and limb elevation. Several leg and foot muscle pumps coordinate to facilitate return of venous blood from the lower limb.

Muscle Contractions

Muscle contractions work as peripheral venous pumps. Muscular activity lowers edema by lowering intravenous pressure. Muscular activity also increases lymph flow.[21,27] Intramuscular pressure in the leg is also reduced following muscle contraction of the leg in a standing or sitting subject (Figure 18.2).[28] Pressure in different muscles during contraction increases to 100 to 200 mmHg.[29–39] The recruitment pattern and muscle length changes that occur during exercise may provide a more effective muscle pump than during electrical stimulation.[40]

Passive Stretch of Muscle

Passive stretch of leg muscles may increase intramuscular pressure fivefold.[28,41–43] It may also increase lymph flow.[21,27,44,45] Intermittent passive muscle stretch oscillates the interstitial hydrostatic pressure in different leg muscles.[28,41,42,46] Pulsations of hydrostatic pressure in the interstitial space increase lymph flow.[26] These pulsations of hydrostatic pressure may also reduce edema in human legs. Passive muscular stretch may therefore be a method of treatment in patients with limb edema.

Another clinical application of intermittent passive stretch of muscle is continuous passive motion equipment, which possibly works to reduce edema by limb elevation and by passive stretch of muscles.

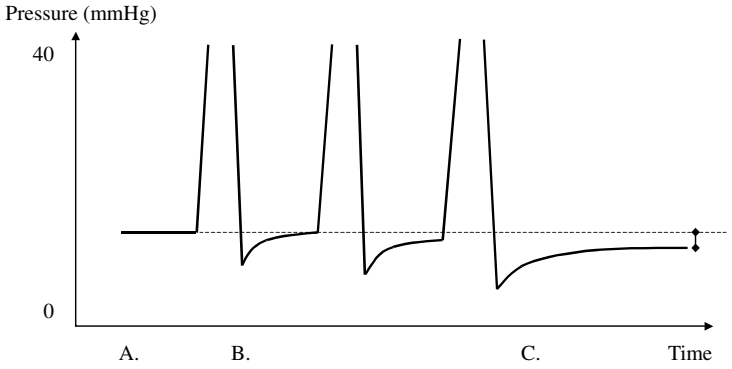

FIGURE 18.2 Pressures in a leg muscle before (A), during (B), and after (C) three active flexions of the ankle joint in a patient with vein obstruction at the thigh level. Intramuscular pressure is lower after the contractions due to decreased volume of the leg. Active muscle contractions are effective in reducing elevated pressure due to venous stasis.

External Compression

Continuous Compression

External compression of skeletal muscle increases lymph flow.[27] Compression stockings are not suitable as an edema-reducing treatment following reconstructive vascular surgery or in patients with imminent acute compartment syndrome. Compression stockings increase interstitial hydrostatic pressure and thereby decrease the local arteriovenous pressure gradient. The decreased perfusion pressure may lead to increased risk for limb ischemia in bedridden patients.

Intermittent Compression

Methods for intermittent pneumatic compression of the leg or limb have been used to treat venous insufficiency, postural edema, and prevent thrombosis.[47–49] They are proven to effectively reduce swelling and pain after fasciectomy.[50] They may also be useful in patients following vascular trauma and vascular reconstruction. They do not mimic the normal action of musculovenous pumps. Intermittent external compression of the calf muscles is still a controversial method to reduce edema.

The venous plexus of the sole of the human foot operates by weight bearing on the foot and acts in conjunction with the musclovenous pumps further up the leg.[28,51] Neither toe nor ankle movements can empty the venous plexus in the nonweight-bearing foot. Passive compression of the sole of the foot by a pneumatic pump gives oscillations of the interstitial hydrostatic pressure in the leg.[28] The mechanism for this may be intermittent passive muscle stretch of leg muscles (Figure 18.3). The tendons of leg muscles may be passively stretched by external compression at the longitudinal arch of the foot (Figure 18.4). Impulse compression of the foot reduces

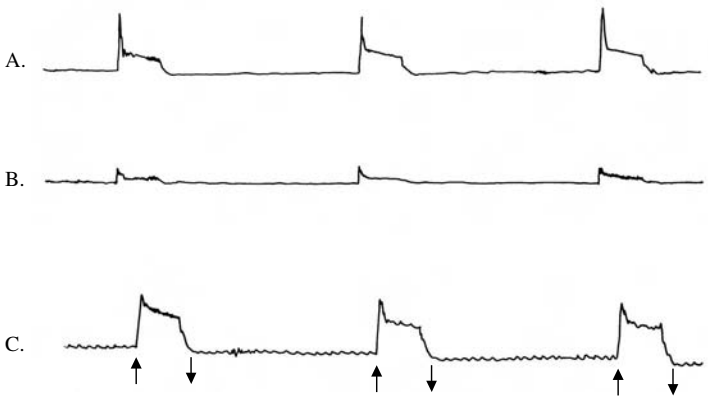

FIGURE 18.3 Pressure in the flexor digitorum muscle (A) and in the subcutaneous tissue (B) in a healthy leg without venous stasis. Intramuscular pressure during venous stasis is shown at (C). The intramuscular pressure oscillations last for about 4 sec, which is the same time the foot pump is activated. Arrow up indicates the start of pump action and arrow down indicates the end of activation.

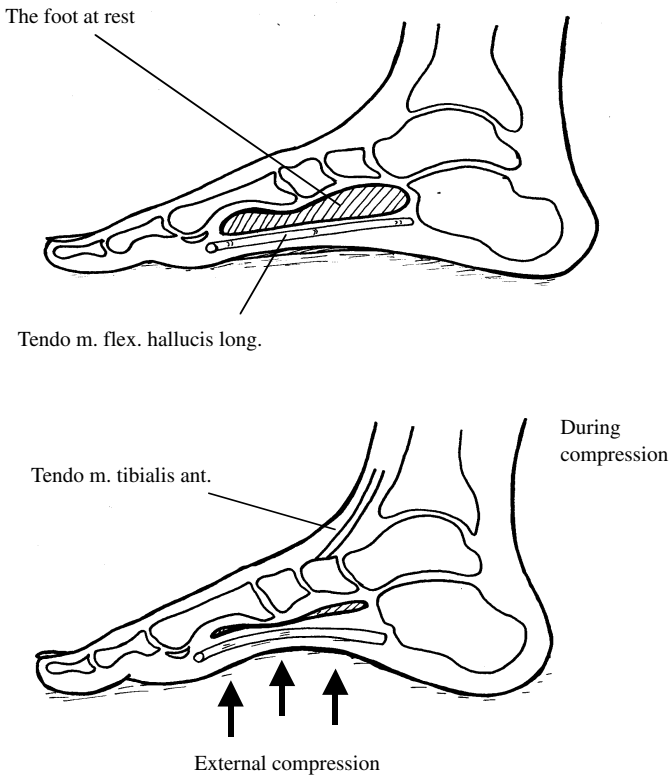

FIGURE 18.4 Foot at rest (A) and during compression by the pneumatic foot pump (B). The figure illustrates how the tendon to the muscle is deviated when it passes the longitudinal arch of the foot. The pressure amplitude in the lower leg during pump activation is explained by passive intermittent muscle stretch. (Redrawn from Styf, J., *J. Clin. Physiol.,* 10, 77, 1990.)

posttraumatic swelling and pain, but cannot reverse abnormally high intramuscular pressure in patients with acute leg compartment syndrome.[52]

Treatment by the venous foot pump simulates the effects of weight bearing by increasing venous return in immobilized patients.[28,52] Gardner and Fox[51] used the phlebographic technique to show that mechanical activation of the venous plexus of the foot could return the venous flow up to groin level without any assistance of muscular activity. Venous return from the lower extremity may thus be stimulated by a passive, mechanical activation of the venous plexus of the foot.[28,51–53] Venous return by this mechanism is similar to that seen in standing and walking.

Limb Elevation

The desirable effect of limb elevation early after trauma and surgical treatment is to diminish local bleeding by decreased perfusion pressure (Figure 18.5). Limb elevation is recommended as a treatment in patients with venous hypertension

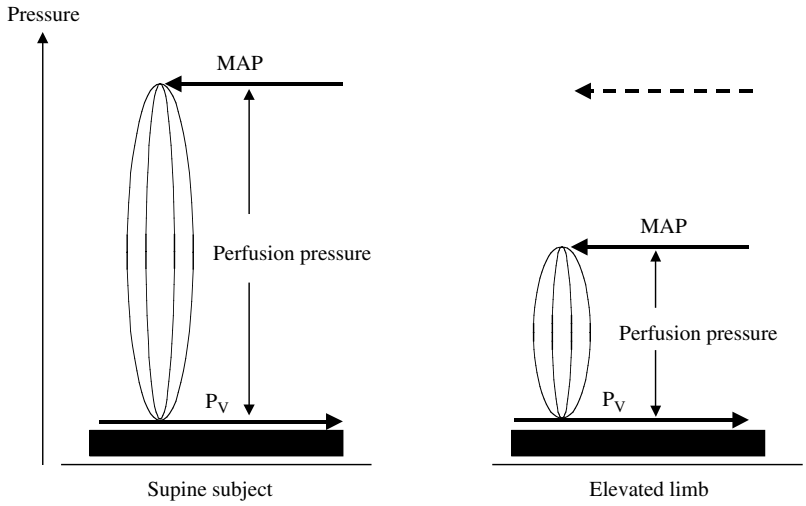

FIGURE 18.5 Normal perfusion pressure in a supine subject with the limb at heart level (left figure) and the decreased perfusion pressure in an elevated limb. The main effect of limb elevation is deceased local perfusion pressure due to decreased mean arterial pressure (MAP). The venous pressure (P_v) remains unchanged.

following trauma.[54] However, elevation of a limb that is externally compressed may impede local muscle blood flow[55,56] and may induce dysfunction of the deep peroneal nerve.[56] Limb elevation during prolonged surgery may also induce acute compartment syndrome.[57–59] Limb elevation combined with external compression in patients with arterial disease of the lower limb increases the risks for insufficient perfusion pressure. Finally, limbs should not be elevated in patients with abnormally elevated intramuscular pressure (or imminent acute compartment syndrome) because limb elevation may induce ischemia due to decreased perfusion pressure.[60] The effects of an elevated limb on the interstitial hydrostatic pressure, arterial pressure, and the intravenous pressure in healthy individuals and in patients with abnormally increased tissue pressure are further discussed in Chapter 4 and Chapter 5.

Leg and Foot Muscle Pumps

The calf has two ways of acting as a musculovenous pump, one passive, which is distal, and the other active, which is proximal. The distal muscle pump is activated by dorsiflexion of the ankle joint. The distal part of the posterior compartments is narrower than the proximal parts. When calf muscles are pulled distally by ankle joint dorsiflexion, they are compressed by the osteofascial boundaries of the posterior compartments. It is believed to function as a conically shaped piston, which is passively compressed by the osteofascial boundaries of the leg.[41,43,52] The proximal musculovenous pump is activated by concentric muscle contraction during plantarflexion of the ankle or by eccentric muscle activity during dorsiflexion of the ankle joint.[28,52,61]

Intramuscular pressure in the calf muscles may increase by three ways. First, pressures may reach 100 to 300 mmHg during concentric and eccentric plantarflexion of the ankle joint.[62–64] Second, calf muscles are antagonistically coactivated during dorsiflexion of the ankle joint to intramuscular pressure values of at least 30 to 60 mmHg. Finally, passive dorsiflexion of the ankle joint may increase pressure in calf muscles to 40 mmHg.[41,43,63]

Weight bearing on the foot is a powerful edema-reducing mechanism. It activates all the leg muscle pumps and empties the venous plexus of the foot.[28,51] The muscle pumps of the leg and foot are synchronized. The most powerful edema-reducing mechanism is muscle contractions during plantar flexion of the ankle joint. By intermittent external compression of the foot, the venous maximal flow reaches its peak when the compression interval is about 1 min.[48]

Transplantation of Venous Valves

Patients with insufficient venous valves in the deep venous system experience swelling, heaviness, and pain in their legs, because of edema. These patients have elevated intramuscular pressure due to venous insufficiency.[15,65] The symptoms may improve and the muscle relaxation pressure during exercise may normalize by transplantation of patent venous valves (Figure 18.6). Transplantation of a single valve to the superficial femoral location of an incompetent venous system may temporarily correct venous hemodynamics.[66] Microsurgical flap reconstruction maintained good clinical and hemodynamic results over time.[67]

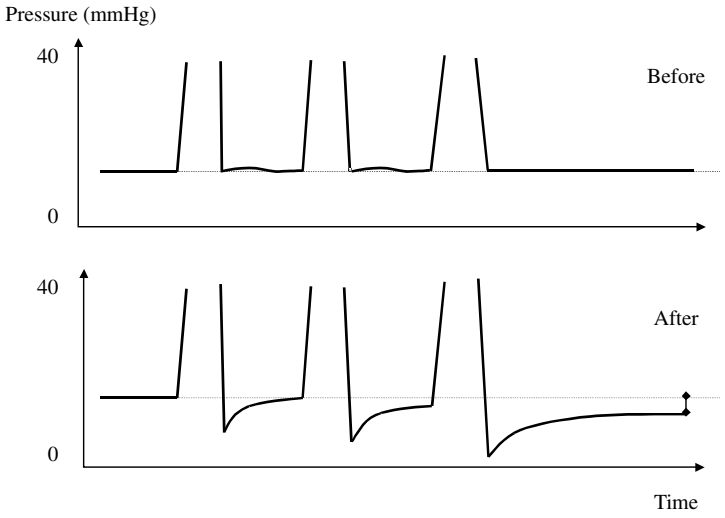

FIGURE 18.6 Intramuscular pressure recording before and after operation for insufficient valves. Before surgery (upper trace), MRP is elevated due to the reflux over the valves. After operation (lower trace), MRP as well as intramuscular pressure at rest after exercise are normalized as signs of valve competence.

Hyperbaric Oxygenation

Treatment by hyperbaric oxygenation increases tissue oxygenation in hypoxic tissues to levels that maintain viability. It may also reduce edema by vasoconstriction.[68–70] The increased partial pressure of oxygen in the blood causes vasoconstriction by direct action on blood vessels.[69] The mechanism is supposed to include decreased blood flow and perfusion pressure to the area of injury. Plasma hyperoxygenation compensates for the decreased flow associated with vasoconstriction. Improved tissue oxygenation reduced capillary pressure and decreased edema formation lead to resorption of extravascular fluid and decreased interstitial fluid pressure.

SUMMARY

Edema is always a sign of disturbed microcirculation. Following trauma or surgery edema may alter the conditions for tissue nutrition and viability. Patients may experience swelling, pain, and impaired neuromuscular function. Several active and passive mechanisms to stimulate peripheral musculovenous pumps are available. Concentric muscle contraction is probably the most powerful edema-reducing mechanism. Passive muscle stretch and several methods for external compression and limb elevation are additional methods. Hyperbaric oxygenation improves tissue oxygenation, reduces capillary pressure, and decreases edema formation.

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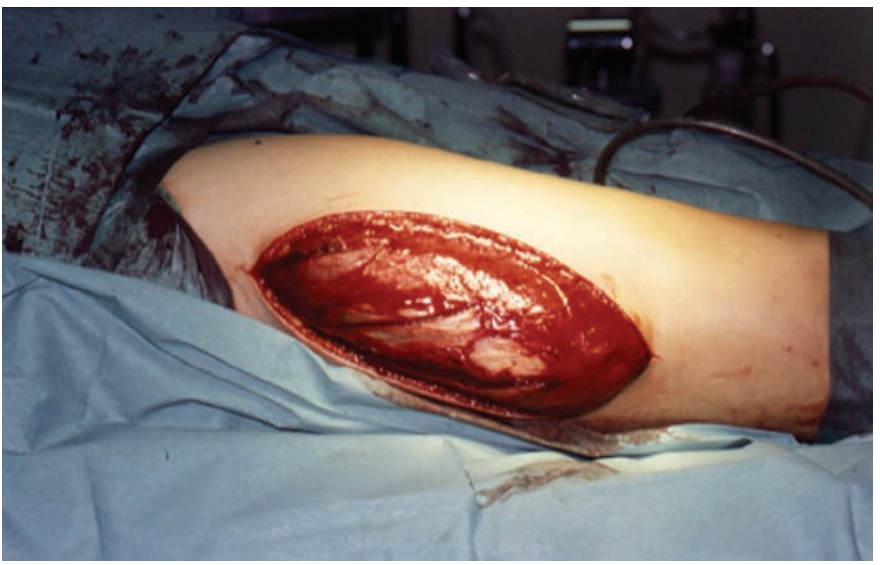
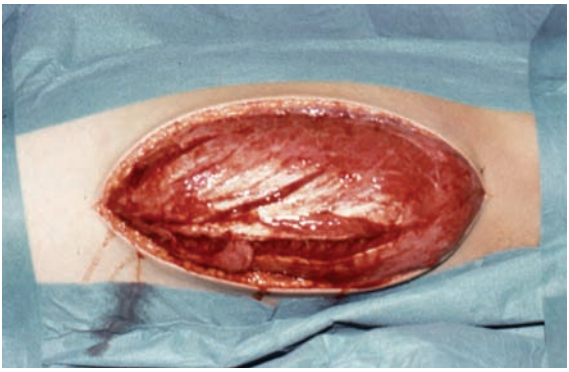


COLOR FIGURE 6.6 Intraoperative photo of the lateral thigh. The skin incision should be complete from the greater trochanter to the lateral femoral condyle.



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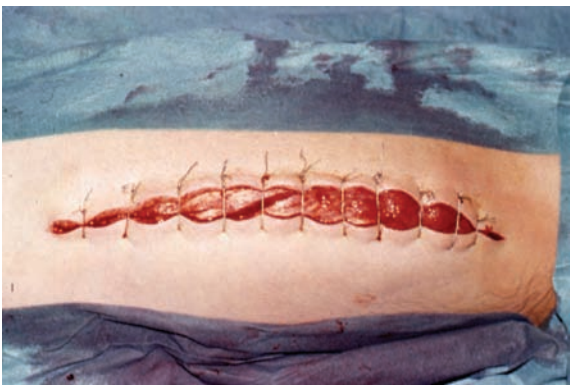

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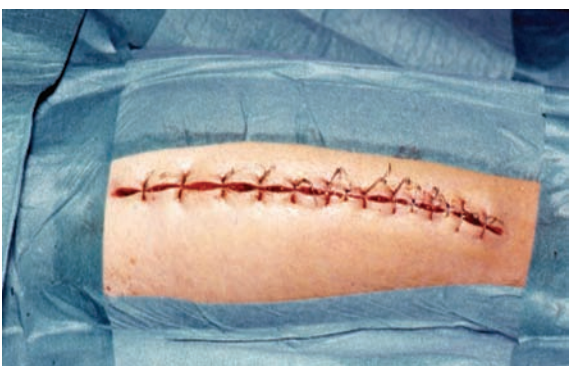
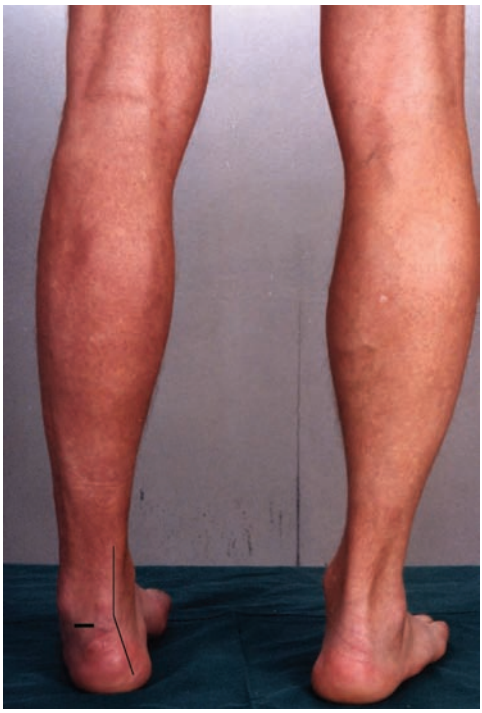
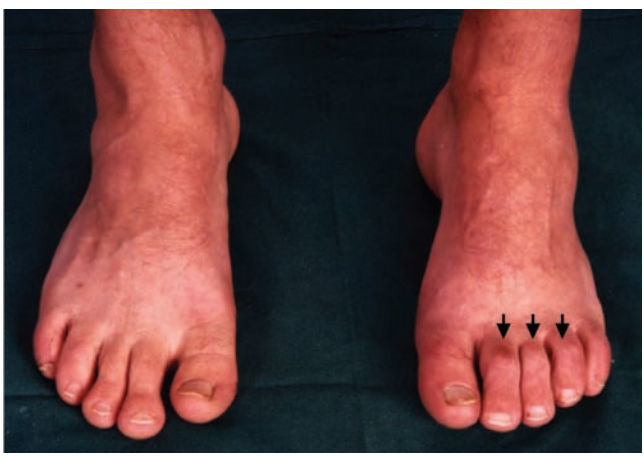


(C)

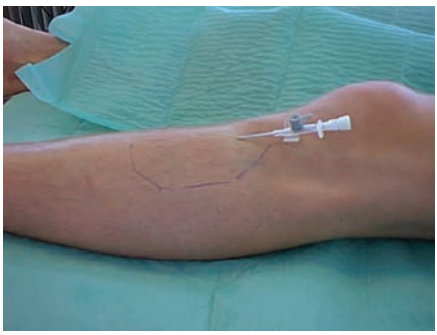
COLOR FIGURE 6.12 Secondary wound closure by wire sutures in a patient 3 days after fasciotomy for acute compartment syndrome of the thigh. (A) The lateral thigh wound 3 days after fasciotomy. (B) Wire sutures are applied 3 days after fasciotomy. (C) On Day 5 the sutures are tightened to completely close the wound.



COLOR FIGURE 8.3 Ischemic contracture following untreated acute compartment syndrome of the left leg. The left side shows a short cavus foot with an equinu-varus deformity. The distance between the lateral malleolus and the Achilles tendon (black bar) is shorter.



COLOR FIGURE 8.4 Mild claw toe deformities of dig. II to IV in patient with ischemic contracture of the flexor digitorum longus muscle and intrinsic dysfunction. The soft tissues along the longitudinal arch are hypotrophic. The foot is shorter and has a cavus deformity.



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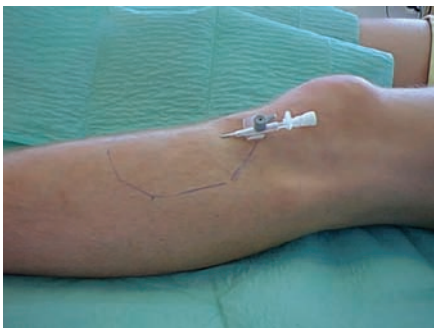

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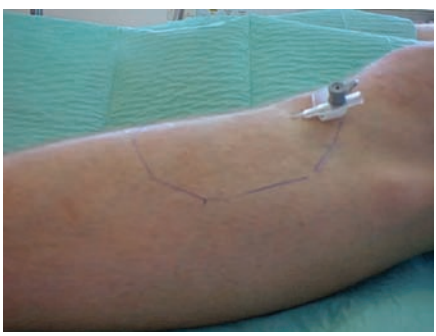

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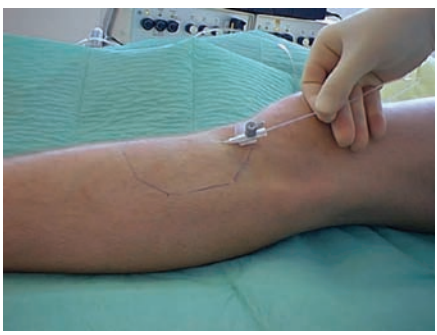

(D)

COLOR FIGURE 16.3 Pictures of catheter insertion. (A) After the muscle fascia has been penetrated the steel needle should be withdrawn a few millimeters into the plastic sheath of the introducer. (B) In this way the cutting tip of the needle will not traumatize the muscle tissue when the introducer is advanced parallel with the muscle fibers into the preferred position. (C) The steel needle is retracted and (D) replaced by the pressure-recording catheter. Finally, the plastic sheath of the introducer is removed.

Orthopedics

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Jorma Styf

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