CHAPTER **44**

# EXERTIONAL COMPARTMENT SYNDROME

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## INTRODUCTION

Compartment syndrome is a condition that many of us have either read about or perhaps seen and treated. It is a limb-threatening and life-threatening condition in which increased pressure within a closed anatomic space compromises the circulation and function of the tissues.[1] The current body of knowledge reflects that untreated compartment syndrome leads to tissue necrosis, permanent functional impairment, and if severe, renal failure, and death. The original description of the consequences of unchecked intra-compartmental pressures is widely attributed to Von Volkmann. His 1872 publication documented nerve injury and subsequent contracture from compartment syndrome following a supracondylar fracture. The injury remains known as Volkmann's ischemic contracture.[2]

It is well documented in the literature that trauma is a leading cause of acute compartment syndrome. A variety of other conditions can lead to compartment syndrome, including arterial injury, thermal burns, crush injury, snakebites, and circumferential casts and dressings. Fracture of the tibia represents the most common cause of an acute compartment syndrome.[3]

It is also important to be familiar with another type of compartment syndrome, known as chronic exertional compartment syndrome. This is an overuse phenomenon typically involving the lower leg, especially the anterior and lateral compartments. It often occurs in athletes, particularly in relation to rigorous training. However, it may also occur in the "weekend warriors," the recreational athlete who overly exerts himself.[4]

Patients will typically experience pain and swelling, with possible sensory deficits or paresthesias, and motor loss or weakness. An important note is that this phenomenon is characterized by exercise-induced pain and swelling that is often relieved by rest.[4] A rousing game of football or a daily jog can trigger this event. The diagnosis is crucial yet may be challenging to obtain in the chronic type. This condition may end an athlete's career if not diagnosed and managed properly.

## PATHOPHYSIOLOGY

Most theories of the pathophysiology of chronic exertional compartment syndrome are based upon acute compartment syndrome. The etiology of chronic exertional compartment syndrome, unlike acute compartment syndrome, is still unclear. It was, and is still believed by most clinicians, to be based on a relative ischemia or reduced blood flow and nutrients to the muscles and nerves within a compartment. Intracompartmental tissue pressure is usually lower than pressure of arterial blood flow. For this reason, peripheral pulses remain intact. Some athletes have shown an increase in muscle volume of 20% with exercise. The surrounding fascia with its minimal elasticity does not allow the compartment to swell appropriately, in turn returning the pressure back into the microcirculation, arterioles, and veins.[4]

Matsen believes that the increase in training leads to a permanent increase in muscle size. Muscle hypertrophy occurs during exercise, due to an increase in the blood supply to the capillary bed. This combination of muscle hypertrophy and non-compliant fascia has been blamed as the causes of chronic exertional compartment syndrome.[5] Styf in 1989 suggested that exercise plays a different role in the development of a compartment syndrome. He proposed that strenuous exercise causes a micro-trauma to muscle, which in turn leads to myositis and inflammation in the capillary bed. The result is an increase in filtration and compartment pressure.[6] Friden in 1983 found that eccentric contraction causes muscle damage and releases protein bound ions. These ions increase the osmolarity of the interstitium, which increases the compartment pressure.[7]

Beckham also discussed the role of eccentric contraction in the development of anterior compartment syndrome. His study found that cycling did not

increase compartment pressure as much as running. He concluded that this was because cycling involved no eccentric contraction of the anterior tibial group.[8] The results of Jerosch's study also suggests that decreased eccentric contraction as occurs when wearing the shoe with a negative heel, causes less of an increase in the compartment pressure.[9] Whiteside had a theory that the development of a compartment syndrome depends not only on intra-compartment pressure but also on systemic blood pressure. He felt that diastolic blood pressure minus the compartment pressure should be greater than 30.[10]

Chronic exertional compartment syndrome is far more common than the acute form. The clinical presentation of chronic and acute compartment syndrome differs only in severity as well as in the onset and acuteness of symptoms. The chronic form is associated with increased pressure while muscles are relaxed. Skeletal muscle is perfused only during muscle relaxation, and relaxation pressure that exceeds 35 to 45 mm Hg results in decreased blood flow and acute myoneural ischemia. Normal tissue pressures range between 0-4 mm Hg. Studies have shown that transient increases in compartmental pressure during exercise are tolerated and don't cause symptoms in initially asymptomatic people. These pressures normalize quickly after exercise, usually within 5 minutes. In chronic exertional compartment syndrome intracompartmental exercise pressures may remain abnormally high for 20 minutes or longer after exercise before returning to normal. Ischemia and necrosis of the muscles occur even though the arterial pressure is still high enough to produce pulses. Muscle and nerves can survive for up to 4 hours of ischemia without irreversible damage. Nerve kept ischemic for less than 4 hours will show neuropraxic damage, whereas after 4 hours, nerves will exhibit wallerian degeneration.[5]

Running is the main activity blamed for the development of the syndrome. Detmer in 1985 found that 87% of patients in his series were involved in sports. Runners made up 69% of the active patients.[11] Martens and Moyerssons also noted a high proportion of soccer players and cross-country skiers.[12]

## DIAGNOSIS

Diagnosis can be made on the basis of history, physical examination and diagnostic investigations. Historically, pain, pallor, pulselessness, paresthesias, and paralysis have been the classic findings in acute compartment syndrome. However, not every patient will demonstrate these findings, especially in the chronic form. The most impressive symptom of compartment syndrome is pain out of proportion to the primary problem as well as pain with passive stretch of the involved compartment.[4]

In chronic exertional compartment syndrome the type of pain is typically described as aching, cramping, and tightness that is usually located over the anterior compartment, which accounts for 45% of all cases. The deep posterior compartment is followed closely (40%). The lateral compartment accounts for 10%, and the superficial posterior is about 5%.[7] In the foot, the medial compartment is the most common. Pressure increases may result from limited compartment volume, poor transient expansion of the compartment, muscle hypertrophy, elevated tissue perfusion during activity, or other factors such as taping, bracing, casts, prostheses, footwear, or orthoses. Excesive talocalcaneal and talonavicular joint pronation may provide compression of the medial compartment of the foot sufficient to precipitate pressure increases. Abductor hallucis hypertrophy may also be considered as an etiologic factor.[7] Bruckner and Kahn state that the relationship of pain to exercise is the most important feature to establish in the history. The usual pattern is for pain to increase on exercise, and reduce or disappear on rest. Associated symptoms can include numbness and or tingling on the dorsum of the foot. Bilateral symptoms are common and are suggested in 82% involvement the legs.[13]

Physical examination in chronic exertional compartment syndrome is usually normal unless the patient has recently exercised. Therefore, performing the physical examination after the patient has exercised strenuously enough to reproduce symptoms is recommended. Such exercise will produce swelling and tension in the involved compartments and increased leg girth. The signs of chronic exertional compartment syndrome are typically absent at the time of examination. For this reason, the patient should perform the exercise that induces symptoms. Following the aggravating activity, the compartment is often tense, firm, and

painful to palpation. It may be very painful to passive stretch of the muscles passing through the compartment. Occasionally there will be a fascial hernia present. Vascular signs are rare and should not be relied upon to make the diagnosis.[4]

Differential diagnosis may include stress fractures, shin splints (medial tibial stress syndrome), muscle strain, blood clot, claudication, cellulitis, radiculopathy, and tumor. One should begin investigation with a plain radiograph. The next step recommended is a bone scan to rule out fracture or medial tibial stress syndrome. Nerve conduction studies are generally not recommended, unless the patient has significant motor loss.

Occasionally a MRI will be of benefit if specific soft tissue abnormalities are strongly considered. Duplex ultrasound or venography may be utilized to rule out thrombosis.[4] However, one must consider the cost/benefit ratio.

Although history and physical examination are important, a review of the literature suggests that compartment pressure tests are the only accurate and objective methods available to make a definitive diagnosis. The pressure is most commonly tested using an intracompartmental needle with pressure being measured with a closed system. A common tool is the Stryker Intracompartmental Pressure Monitor System. There is no gold standard for the diagnostic pressures to confirm the diagnosis, but the most commonly used are as follows:

- A resting pressure greater than 20 mm Hg; or
- An exertional pressure greater than 30 mm Hg; or
- A pressure of 25 mm Hg or higher 5 minutes after stopping exercise

A variety of other methods may be used to measure intracompartmental pressures including the Whiteside infusion technique (Figure 2),[10] the Wick catheter (Figure 3),[14] and the slit catheter (Figure 4).

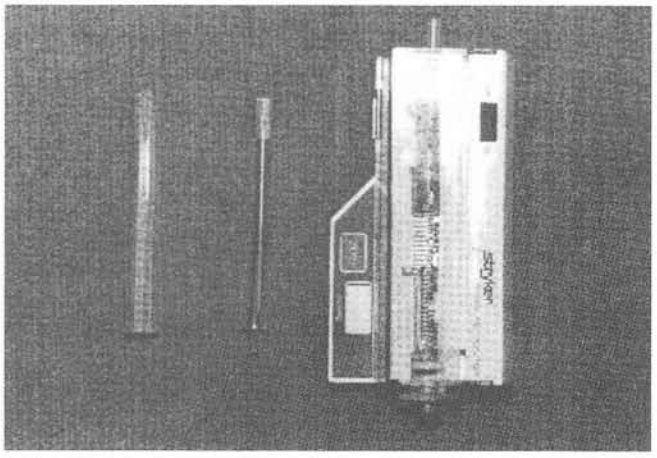

Figure 1. Stryker S.T.I.C. device.

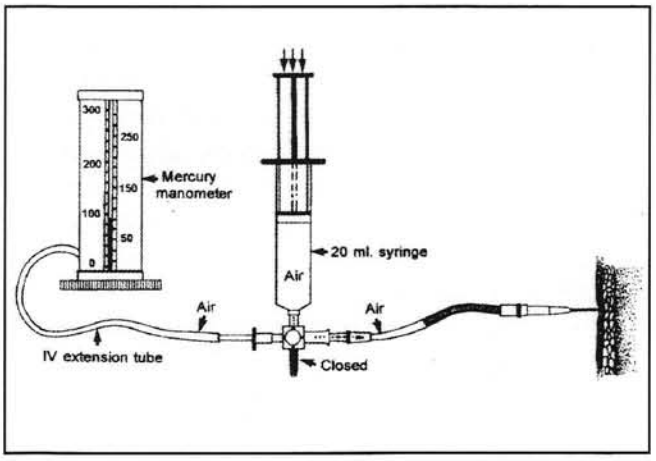


Figure 2. Whiteside's Infusion Technique

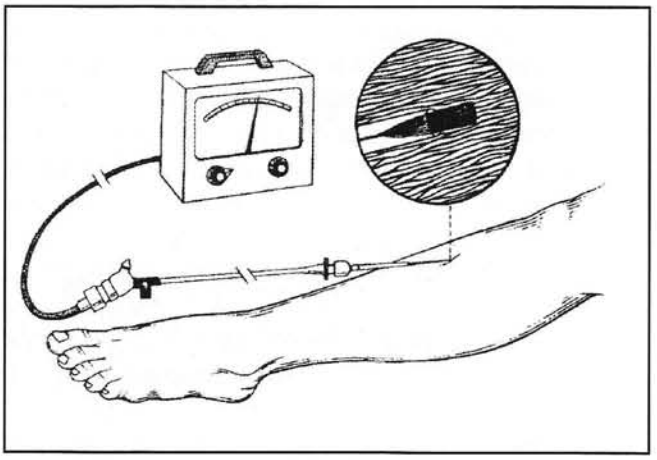

Figure 3. Wick Catheter

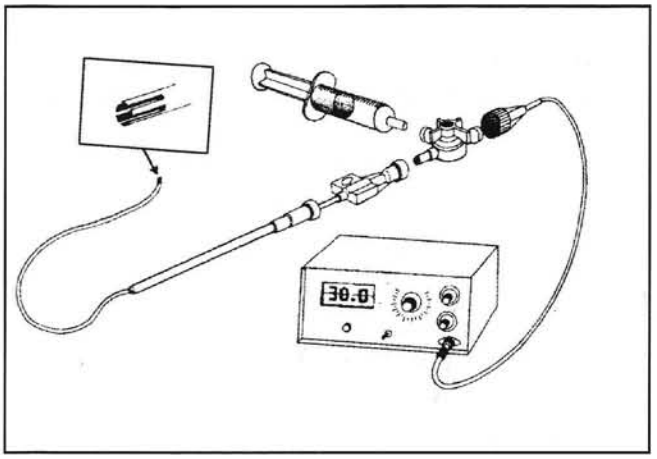


Figure 4. Slit Catheter

The needle tip location and depth of penetration, as well as knee and ankle position, are controlled to obtain reliable results. It is recommended to keep the patient supine with the knee extended and the ankle in neutral position. Although testing has been recommended before, during, and after exercise, the majority of the literature does not recommend measuring during exercise because it is technically difficult and measurements are less reliable. An effective technique is to inject small amounts of local anesthetic into the skin alone, and not into the fascial compartment, and measure the pressures before exercise and at 1 and 5 minutes after exercise. However, values obtained after exercise tend to be more reliable for confirming the diagnosis. The measurements obtained after exercise are valid only if the exercise reproduces the patient's symptoms. Bilateral measurements may be obtained if the results of pressure measurements are equivocal, or if both legs are symptomatic.[4]

In a study involving 14 patients, thallium chloride scintigraphy was found to be a sensitive method of diagnosis.[3] The test proved to be quite accurate when both qualitative and quantitative assessments were used. Use of the stress thallium protocol revealed reversible ischemia in the involved compartments of the lower extremity during exercise. This test may be even more reliable than compartmental pressure tests, since it accurately identified four of four patients with typical symptoms of chronic exertional compartment syndrome and elevated pressures, and also identified a patient who had only borderline elevations in pressure. Testing revealed that 7 of 14 patients had multiple involved compartments, raising the question, are multiple compartments commonly affected?

Larger studies are needed to confirm these findings, but thallium chloride scintigraphy appears to be the most promising test for use by physicians, because it could be easily ordered and is noninvasive. The patient exercises on a treadmill with enough significant grade and speed to produce a heart rate that is 85% of predicted maximum. When leg symptoms are reproduced, thallium is injected and postexercise and resting cross sectional views are obtained and compared with the preexercise images of the suspected compartment for possible defects, which would indicate ischemia.[6]

## TREATMENT

Initial treatment of chronic exertional compartment syndrome is usually conservative. The patient is rested from the aggravating activity. Cross-training with low impact activities such as swimming and bicycling may be permitted if symptoms are not exacerbated. Rest, ice, and elevation can be used to aid the involved limb, however compression is avoided. Nonsteroidal antiinflammatory drugs may be used to reduce inflammation. A gradual return to activity is then warranted. It is also important to address both extrinsic and intrinsic contributing factors. Extrinsic factors include training surface, shoe design, and training intensity, all of which can be modified. Intrinsic factors such as muscle imbalance, flexibility, and limb alignments (especially hind-foot pronation), are treated with strengthening and stretching exercises of the antagonistic muscles and orthoses.

If symptoms persist for 3 months, or if the chronic form converts to the acute form, a fasciotomy is recommended. Fasciotomies may be performed through lateral, medial or combination approaches. The lateral approach has been favored by most authors due to its ability to release all 4 compartments with one incision and reduce the compartment pressure (Figure 5). Postoperative mobilization is recommended to prevent the compartment from closing.

Figure 5. Perifibular incisional approach to release all 4 compartments. Notice retention bolsters to support wound edges

There are numerous reports supporting the treatment option of fasciotomy. Martens and Moyersoons treated 120 cases by fasciotomy and found that 80% of them were able to compete at a higher level of physical activity than before surgery. Martens treated 20 patients via fasciotomy and 16 of them had no complaints resulting from the surgery.[12] Limited information exists that illustrates the downside to surgical intervention. Bouche suggests potential complications of fasciotomy, including infection, neurovascular injury, incomplete release and residual weakness.[15]

What is the criteria for performing fasciotomy? This issue has provided fuel for argument among multiple authors. Keene decided to perform fasciotomy in athletes with typical history and a resting pressure greater than 12 mm Hg.[16] McDermott insisted that measurement of resting pressure is of no significance, as the patient is only symptomatic during exercise. According to McDermott, fasciotomy is warranted when the mean compartment pressure minus the mean resting pressure divided by 2 during exercise is greater than 85mm Hg.[17]

The fascia and skin are left open for a few days and repetitive measurements may be taken. When the pressure is reduced to zero, closure may be performed. One may also consider bicarbonate or acetezolamide to alkalinize the urine and help prevent myoglobinuria. This occurs after reperfusion of the damaged tissue, where the myoglobin in the muscle is released and precipitates in the renal tubules, leading toward renal failure.[4]

## CONCLUSION

Although many physicians are familiar with acute compartment syndrome and all of the accompanying sequelae, it is important to be familiar with the chronic form of this condition. Although the symptoms may be transient and not as intense as an acute compartment syndrome, the chronic form may convert to an acute form. It is usually diagnosed with a thorough history and physical examination as well as intracompartmental pressure monitoring. Symptoms such as cramping, tightness and paresthesias usually begin during vigorous exercise, and usually improve with rest. The anterior compartment is the most common in the leg and the medial compartment in the foot. If symptoms persist for months or if a chronic condition converts to an acute condition, a decompressive fasciotomy is recommended.

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