Diagnosis, Treatment Options, and Rehabilitation of Chronic Lower Leg Exertional Compartment Syndrome

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Chronic exertional compartment syndrome of the lower leg is a well-described and documented cause of exercise-related pain in recreational, elite, and military athletes. Although this condition is common, the exact underlying mechanisms, those most at risk, long-term effects on muscular strength if unrecognized, and prevention strategies are relatively uncertain. Runners are most commonly affected and can be markedly impaired by the recurrent, often predictable pain that develops with exercise. An accurate history, high index of suspicion, and compartment pressure testing before and after symptomatic exercise confirms the diagnosis. Conservative therapy is minimally effective. Fasciotomy is the treatment of choice for athletes who are unwilling to modify their exercise or sport.

Pathophysiology
The lower leg is composed of four muscular compartments surrounded by osseofascial structures that comprise the walls of the compartments. Some authors debate that there are actually five or more compartments with many subdivisions of the deep posterior compartment [5•]. Most clinicians clinically evaluate only the four commonly described compartments.

The lateral compartment contains the peroneus longus and brevis muscles, a branch off of the anterior tibial artery/vein, and the superficial peroneal nerve. The anterior compartment contains the tibialis anterior, extensor digitorum longus, the extensor hallucis longus, the peroneus tertius, anterior tibial artery/vein, and the deep peroneal nerve. The superficial posterior compartment contains the gastrocnemius, soleus, and plantaris muscles. It also contains a branch of the tibial artery/vein, as well as the sural nerve. Finally, the deep posterior compartment of the lower leg contains the tibialis posterior, flexor digitorum longus, flexor hallucis longus, popliteus muscle, posterior tibial artery/vein, and the tibial nerve.

With normal exercise, muscle volume increases by 20% and intramuscular pressures exceed 500 mm Hg with contractions [3••]. Therefore, like cardiac muscle, peripheral muscles perfuse when the muscle is relatively relaxed, allowing the arterial/venous gradient to increase. In the lower leg, the arterial/venous gradient must be at least 30 mm Hg to overcome normal intramuscular resting pressure [3••]. Gershuni et al. [6] report that CECS occurs when the exercising muscles enlarge normally but are unable to expand secondary to non-compliant fascia, resulting in increasing intramuscular and
compartment pressures. When this pressure exceeds 30 mm Hg the perfusion gradient is lost. Ischemia and pain ensues, only to be relieved with rest and resolving pressures over time. Pedowitz et al. [7] demonstrated decreased thallium-201 distribution in muscles affected by CECS by using single photon emission computed tomography (SPECT). This suggests a lack of perfusion in the postexercise symptomatic muscles. Fugl-Meyer [8] and Schepsis et al. [9] suggest that CECS of the anterior compartment occurs secondary to the occlusion of large vessels in areas of local muscle herniation as they cross the interosseous membrane. Hurschler et al. [10] biopsied fascia from patients with CECS and found that 25 out of 26 had abnormally thickened, noncompliant fascia. The question that remains is whether the documented high pressures in the compartments result in profound ischemia and pain. Some authors feel that the pressure does not cause ischemia but does stimulate pain receptors in the fascia and peristem [5•]. It is known that as the high pressures persist or increase pain, paresthesias, muscular weakness, and even foot drop may develop. However, the degree of pressure elevation does not correlate with the degree of pain, nor does it predict outcomes post fasciotomy [4••].

In summary, the pathophysiology of CECS is probably multifactorial (Table 2). Noncompliant, inelastic fascia and muscles that are too large for their space play a large role in contributing to the abnormal high pressures that occur with exercise. The resulting pain and neurovascular compromise eventually dissipates with rest and shrinkage of the muscles in the respective compartments.

### Risk Factors
The use of creatine supplementation and androgenic steroids increases muscle volume throughout the body. The use of these substances has been implicated as a potential cause of abnormal intracompartmental pressures in exercising athletes [2,11]. Eccentric exercise in postpubertal athletes may decrease fascial compliance over time, and in those congenitally predisposed, create a favorable environment for CECS [2,11].

### Clinical Presentation
Chronic exertional compartment syndrome has a very typical history in most instances. Athletes report that after a predictable intensity or duration of a specific exercise their symptoms gradually progress until pain, cramps, muscle tightness, distal paresthesias, or altered muscle function requires them to stop. After a predictable rest period, usually minutes to hours, symptoms resolve. The symptoms may occur in one leg; however, bilateral symptoms tend to occur in the lower extremities 85% to 95% of the time [12]. The athlete may experience symptoms on the lateral, anterior, or posterior portion of the lower extremity. Other concomitant injury, such as tibial stress syndrome, may delay the diagnosis of CECS.

The physical examination of the lower extremities is usually unrevealing. Athletes lack bony tenderness, gross edema, popliteal fullness, or masses. Gait and flexibility is usually normal. When symptoms are present, palpating the muscular compartments may reveal tightness and tenderness. Passive stretching of the calf musculature increases the pain when pressures are elevated and is a key diagnostic finding [3••]. Interestingly, 10% to 60% of symptomatic athletes with CECS have been found to have small fascial defects in the lower leg [2,11]. Rarely these fascial hernias may be palpated, but their exact contribution to the pain associated with CECS is unknown.

### Differential Diagnosis
Hurschler et al. [10] classified exertional leg pain into three types: type 1) stress fractures; type 2) medial tibial perios­talgia; and type 3) CECS. This scheme describes the three most common causes of exertional leg pain. A more complete list of differential diagnosis is provided in Table 1.

### Diagnostic Tests
Athletes with persistent symptoms suggestive of CECS should be further evaluated using diagnostic modalities. The gold standard test continues to be intracompartmental pressure testing, both at rest and postexercise [3••,9]. A Stryker catheter (Stryker Surgical, Kalamazoo, MI) is commonly used to measure these pressures. Most sports medicine specialists, physiatrists, and orthopedic surgeons are trained and competent in performing this testing. The key to making an accurate diagnosis using this tool is to check resting pressures and then have the athlete perform the sport-spe-