Chronic Exertional Compartment Syndrome

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KEYWORDS

• Chronic exertional compartment syndrome • Chronic compartment syndrome
• Intracompartmental pressure testing • Fasciotomy • Exercise induced leg pain
• Medial tibial stress syndrome • Shin splints • Soleus bridge • Exercise neuropraxia

KEY POINTS

- Increased tissue pressure within a fascial compartment may be the result from any increase in volume within its contents or any decrease in size of the fascial covering or due to any distensibility of the fascial covering (ie, patients with thickened fascia).
- Shin splint pain and chronic exertional compartment syndrome (CECS) can be differentiated by a careful history and by exclusion of other maladies and confirmed by compartmental syndrome testing.
- Once the practitioner makes the proper diagnoses of CECS, through clinical examination and intracompartmental testing, surgical fasciotomy along with ancillary procedures should allow the athlete to return to competitive activity.

Patients who experience intense pain, a burning sensation, tightness, and/or numbness in the lower extremities during exercise activity, whereby the pain resolves quickly after cessation of activity, can often be diagnosed with chronic exertional compartment syndrome (CECS). This syndrome was first described by Mavor[1] in 1956 in which there was increased pressure within a specific muscle compartment of the leg, which causes pressure on vessels and nerves causing the symptoms.

Mubarak and Hargens[2] studied *acute* compartment syndrome (not to be confused with CECS) that included decreased blood flow through the intracompartmental capillaries (capillary ischemia), but continued blood flow to larger arteries and veins with palpable pulses distally.

In a study performed with magnetic resonance imaging, Amendola and colleagues[3] found that CECS is not related to ischemia, but is actually due to increased fluid content within the muscle compartment. This can compromise or impair function of the muscles or nerves within a tight and constricted fascial covering.

It is also hypothesized by myself, as well as several colleagues, that the symptoms of CECS are similar to those individuals diagnosed with lumbar spinal stenosis who

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experience pain, weakness, and numbness causing them to limp. Like spinal stenosis, CECS may be the result of "temporary neurogenic claudication." Here the small capillaries that supply the leg nerves are not getting their normal blood supply because they are compressed by increased compartment content during exercise.

In addition, several colleagues and I think that in CECS, nerve(s) and its pain receptors, such as mechanoreceptors and nociceptors are stimulated by any increased abnormal pressure against them.

Detmer and colleagues[4] found in their study of 100 patients that most cases of CECS involve both legs, although other studies have indicated 80% or more involve both legs. It should also be noted in the study by Detmer and colleagues[4] that the condition affected men and women pretty equally. In my private practice working with several universities and running clubs, I would concur with these findings and note that our average patient with CECS is a competitive athlete between the ages of 18 and 25. We have found equal occurrence among sprint sport athletes as in long distance runners.

Other physical findings of CECS may include mild edema or muscle herniations over the involved compartment, and muscle weakness of a specific compartment. This may include weakness of: dorsiflexion (anterior compartment), eversion/abduction (lateral compartment), plantar flexion (superficial posterior compartment), or inversion/toe flexion (deep posterior compartment).

Symptoms present during exercise also may include paresthesia to the anterior leg or ankle, or between the first and second metatarsal due to involvement of the medial terminal branch of the deep peroneal nerve within the anterior compartment.

If the patient complains of numbness or tingling to the arch or plantar aspect of the foot during exercise activity, that may be associated with a deep posterior leg compartment syndrome and not just a tarsal tunnel syndrome. In my personal experience, however, most patients do not complain of numbness, rather they complain of pain and tightness, which most often forces the athlete to curtail his or her activity level or stop to rest.

DIFFERENTIAL DIAGNOSES

The diagnosis of CECS is initially a diagnosis made by exclusion. Later on it is confirmed by intracompartmental pressure testing.

Often times, the deep posterior muscle compartments may have symptoms of medial tibial stress syndrome or chronic shin splints. Standard treatment of nonsteroidal anti-inflammatory drugs, orthotics, shin splint taping, rehabilitation, and so forth, should be initiated. The practitioner must be keenly aware of biomechanical factors causing irritation and strain of the periosteal and other soft tissues attached to the medial posterior border of the tibia, which can cause shin pain and/or stress fractures. Radiographs should be taken to rule out bone and joint injury.

If there is a concomitant accessory soleus muscle present, which takes up space in the superficial posterior compartment, this may cause CECS. Sometimes, there is an accessory peroneus quartus muscle, which takes up space in the lateral compartment. All preoperative patients with compartment syndrome should be sent for an MRI to see if there is any abnormal pathology including accessory muscles, tendon (look for partial tears) and muscle pathology, soft tissue masses, bone pathology, and so forth. Any increased muscle mass within a fascial compartment can lead to CECS during exercise and the surgeon will need to debulk or excise the accessory muscles and perform the appropriate surgery along with the fasciotomy.

Other factors that must be excluded as a cause of leg pain are claudication and/or popliteal artery entrapment syndrome. With clinical suspicion, these patients should

be sent for vascular testing, preferably functional testing, where the athlete runs and is immediately tested for signs of occlusion. This test may be compared with the baseline "at rest" vascular test.

The superficial peroneal nerve may be pinched (entrapped) as it exits through the lateral compartment leg fascia superficially. This may be detected by a positive provocation sign (direct finger compression elicits point tenderness) and/or confirmed by selective nerve block of 1 mL lidocaine, which should afford temporary relief of symptoms. The sural nerve also may get pinched as it exits the superficial posterior compartment fascia superficially in the distal one-third of the leg. Affected patients may complain of numbness/tingling to the lateral aspect of the foot or ankle. Check for a positive provocation sign directly over the sural nerve at the distal lateral leg. This nerve entrapment also should be verified by a selective nerve block. Nerve entrapment pain may be present at rest, with numbness or tingling along the path of the nerve and its branches, whereas the pain of CECS is usually severe during exercise activity.

Clinically, anterior and lateral CECS frequently occurs together and the superficial peroneal nerve found within the lateral compartment is the most common nerve to be affected by intracompartmental pressures. In those with longer histories of this condition, the patients may relate pains radiating to the dorsal lateral and dorsal aspect of the foot, which I have further diagnosed as intermediate dorsal cutaneous nerve and medial dorsal cutaneous nerve neuropraxia secondary to impingement of the superficial peroneal nerve. In those with chronic deep posterior exertional compartment syndrome, the saphenous nerve has been noted to demonstrate pain at the medial malleolus, and the anterior medial ankle, which extends to the medial big toe joint. So, if patients complain of distal symptoms and leg pains, especially during exercise, the practitioner should include "CECS-induced neuropraxia" in their differential diagnosis.

CHRONIC EXERTIONAL COMPARTMENT SYNDROME ETIOLOGY

My personal observations through clinical and surgical intervention of having tested and/or treated more than 100 patients with CECS are that some individuals are genetically predisposed to this syndrome due to their anatomic muscle composition. We have noted several patients who were twins, with both siblings experiencing this condition. Some individuals are born and exhibit high muscle tone and others develop hypertrophic muscles as a result of repeated exercise activities. This is very true for runners and dancers and anyone who performs repetitive activities. It is known that the lower extremity leg muscle volume may expand 20% during exercise from both increased capillary blood flow and the retaining of extracellular fluid (Puranen[5]). Ultimately, this intracompartment expansion increases the pressure within the enclosed fascia compartment. I personally believe that there is a similarity between CECS of the leg and that which is similar to tarsal tunnel syndrome. In the latter, there is an impingement of the posterior tibial nerve, often times by a surrounding hypertrophic muscle underneath the laciniate ligament. A common example of this is the presence of a hypertrophied flexor hallucis longus muscle in dancers and runners. Typically the flexor hallucis longus should be a tendon by the time it courses under the laciniate ligament, but I have often noted the presence of an enlarged or low lying flexor hallucis longus muscle under the laciniate ligament in patients with tarsal tunnel syndrome. If the athlete with tarsal tunnel syndrome persists in playing the sport with the pain, the muscle under the laciniate ligament may engorge with blood or extracellular fluid and he or she may develop a nerve impingement and resultant symptomatic burning, numbness, or muscle weakness to the plantar foot. Anything that increases pressure

in the tarsal tunnel causes strain on the overlying laciniate ligament and impingement on the nerve. This also includes one with an inherently tight laciniate ligament. In CECS of the leg, there are similar findings in addition to intense pain and tightness.

Biomechanically speaking, I have not had great success nor has the literature supported treating patients with CECS by orthotic therapy. However, when there are biomechanical abnormalities, orthotics should be fabricated before considering surgical intervention. Detmer and colleagues[4] reported that 15% of their patients considered orthotics somewhat helpful, although it is unknown if the use of orthotics resolved their problems.

It has been my experience that some patients present with a small fascial herniation in the lower leg muscles, which may be indicative of CECS. If warranted by intracompartmental testing, appropriate completion of the fasciotomy should be performed.

As one's muscles develop through repetitive activities, it appears that the fascial coverings may not expand enough, grow, or accommodate for the increased muscle mass. On the other hand, the symptomatic patient with CECS may simply have been born with tight fascia that only becomes symptomatic during repetitive activities.

CECS occurs as a result of various factors, but always increases the intracompartmental pressures with resultant symptoms as discussed.

ANATOMY AND CLINICAL EXAMINATION

There are 4 muscle compartments in the lower extremities surrounded by fascia that are commonly tested in compartment syndrome. Detmer and colleagues[4] identified additional subcompartments in the legs, but most surgeons focus on the 4 compartments. Beyond the scope of this article, it should be known that there is also CECS of the foot that is documented in the peer-reviewed literature. The leg compartments include the anterior compartment, which consists of the anterior tibial, extensor hallucis longus, extensor digitorum longus, and peroneus tertius muscles. The lateral compartment consists of the peroneus longus and peroneus brevis muscles. The deep posterior compartment includes the posterior tibial, flexor digitorum longus, and the flexor hallucis longus muscles. The superficial posterior compartment includes the soleus, gastrocnemius, and plantaris muscles.

A key point to note is that the most consistent resolution of CECS leg pain/symptoms is rest, although athletes may be able to sit on the bench or stop running for 15 minutes or so and then return to activity, albeit for short periods of time and often at subpar levels. Most patients have no pain on palpation during their initial examination; however, if they were to run until they have their symptoms, they may have pain on palpation or they would be able to better identify the location of pain. After exhibiting their symptoms, patients should be asked to point their finger to the exact area(s) of their pain so that the clinician may better understand the area of potential fascial release.

TESTING FOR EXERTIONAL COMPARTMENT SYNDROME

The most reliable methods of quantifying exertional compartment syndrome is by taking intramuscular compartment pressures using the Stryker Intra-Compartmental Pressure Monitor System (Stryker, Kalamazoo, MI) by way of pressure testing as described by Whitesides and colleagues[6] and since simplified with use of the Stryker Quick Pressure Monitor Set, including diaphragm chamber and side-ported needle system (**Figs. 1** and **2**).

I typically test one leg in the athlete to spare the patient the discomfort of testing both legs. Although, if the test results are questionable, I will then test the opposite

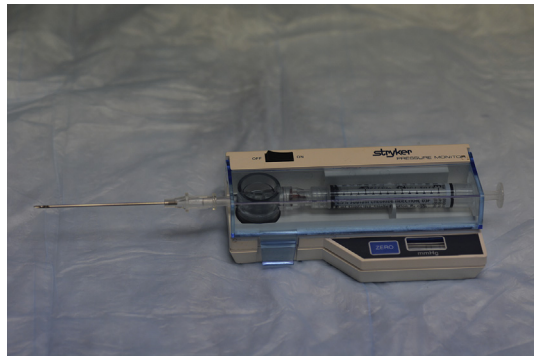

Fig. 1. Stryker intracompartmental monitor system. (*Courtesy of* Stryker, Kalamazoo, MI; with permission.)

leg if symptomatic. The athlete is placed supine on the examination table with his or her knee bent, so the sole of the foot is flat on the table. By having the lower leg upright, there is easy access to all the compartments. An indelible marker is used to circle the area for injection over each of the 4 compartments. The leg is then prepped with povidone-iodine solution. I raise a skin wheal within each circled area for each of the 4 compartments using 0.5 mL of 2% lidocaine plain. This helps reduce the superficial (skin stick) pain of the larger 18-gauge side-port needle. It is important not to anesthetize deep to the skin.

For the anterior compartment, the injection site is between the upper one-third and middle one-third of the anterior leg staying lateral to the tibia and directly over the anterior tibial muscle.

Be sure to zero balance the pressure monitor system holding the unit perpendicular to the leg and parallel to the examination table. Insert the side-port needle into the anterior tibial muscle (**Fig. 3**) approximately 1.5 to 2.0 cm deep and inject approximately 0.3 mL to 0.5 mL saline from the syringe into the muscle belly. Then record the pressure off the monitor once you reach an equilibrium state, which occurs when the LCD readout stops or fluctuates back and forth a few degrees mm Hg.

Repeat the procedure for the lateral compartment (**Fig. 4**), which should be lateral to the anterior compartment and over the area of the fibular shaft. Here, you are inserting

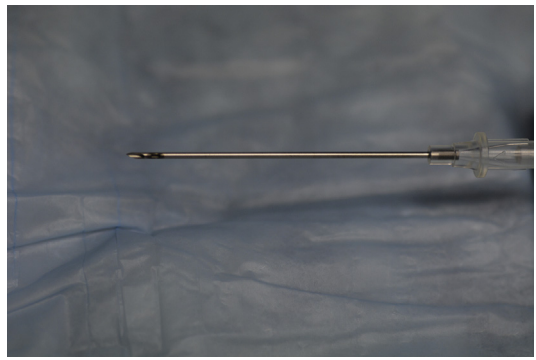

Fig. 2. Side-port needle. (*Courtesy of* Stryker, Kalamazoo, MI; with permission.)

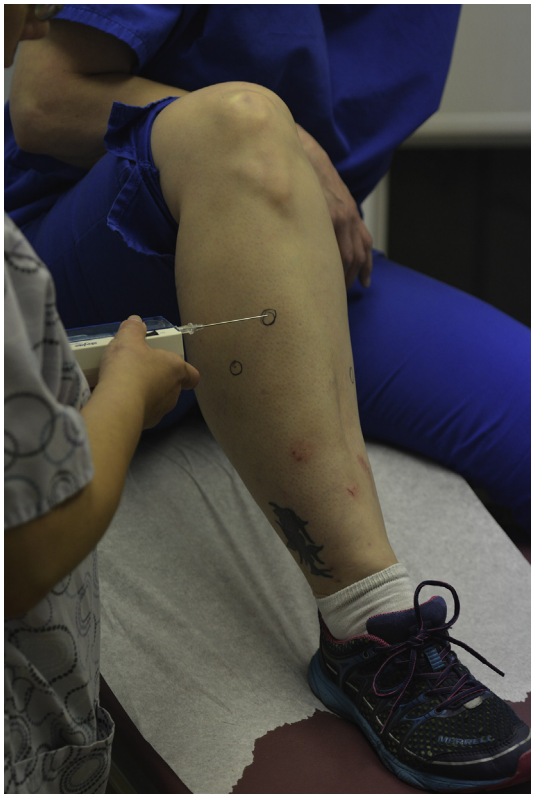

Fig. 3. Anterior compartment testing.

the needle perpendicular into the peroneus longus muscle belly in similar fashion as previously described. After the saline injection, record the pressure.

For the deep posterior compartment (Fig. 5), place the needle midway up the leg staying just posterior to the medial surface of the tibia, thereby staying anterior and avoiding the saphenous vein and nerve. The needle is inserted approximately 2.0 to 2.5 cm deep into the posterior tibial muscle belly and after the injection of 0.3 mL to 0.5 mL sterile saline, the pressure is recorded.

To measure the superficial posterior compartment (Fig. 6), which is more proximal up the leg, insert the needle into the medial head of the gastrocnemius. If the lateral head is more symptomatic, you should measure this pressure as well. Again, in a similar saline injection technique, measure the pressures.

The anterior and the lateral compartments are the most commonly involved compartments in CECS and when performing fasciotomies the anterior and lateral compartments are typically performed together. When the deep posterior or superficial compartments are involved, both compartments receive fasciotomies.

Plastic strip bandages are applied to cover each injection site. After recording the baseline pressures, have the patient run either on a treadmill or outside until the symptoms are reproduced. I have not found that cycling reproduces their symptoms as well as running. Once the athlete returns with the symptoms, the testing is repeated within 1 minute of cessation of activities. The plastic strip bandages are quickly removed and testing is repeated. The second round of injections is slightly less painful than the first

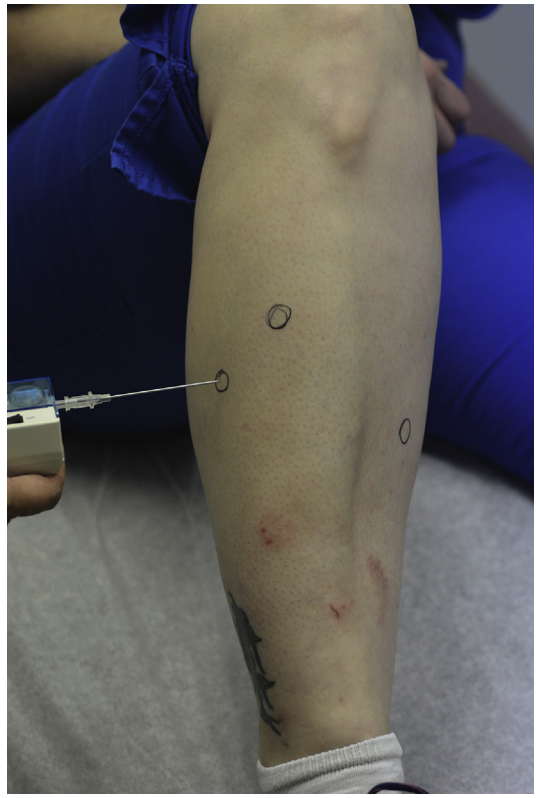

Fig. 4. Lateral compartment testing.

because the needle tract within the skin has already been established. Retest all the compartments in the same order as before and record all pressures.

A timer is used to wait 5 minutes and then all 4 compartments are once again injected with the saline and the pressures again recorded in the same order as previously.

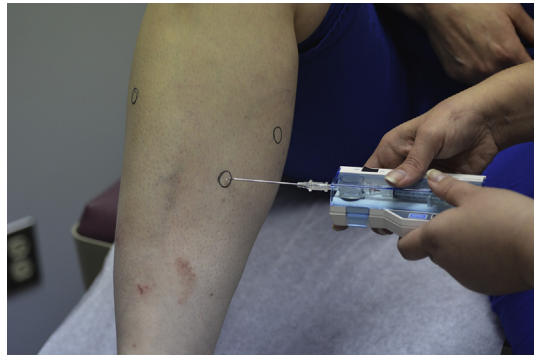

Fig. 5. Deep posterior compartment testing.

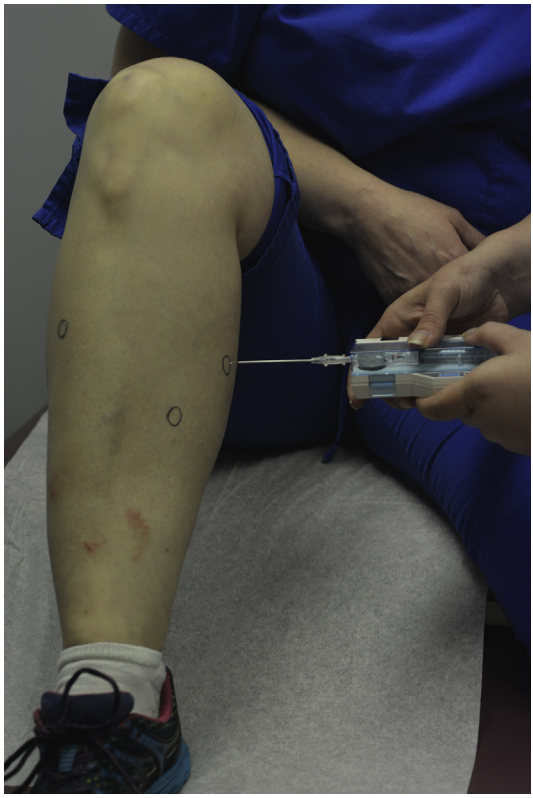

Fig. 6. Superficial posterior compartment testing.

Normal baseline pressures before exercise should be approximately 15 to 20 mm Hg. If the pressures are higher than this amount in the preexercise recordings, then exertional compartment syndrome has been established. If the immediate postexercise pressures increase greater than 30 mm Hg, then this is also considered pathologic. Finally, if the 5-minute postexercise measurements do not return to under 20 mm Hg, then this is also indicative of CECS. At any point of the 3 phases of testing where the pressures are increased higher than normal, then additional testing is not needed, but completing the test with multiple high-pressure recordings is more convincing.

There has been some recent CECS testing performed with functional MRI scanning followed by stress computed tomography (CT) angiography. The premise is that CECS may be caused by functional venous outflow obstruction. Treatment via CT and ultrasound-guided injection of botulinum toxin into the muscle adjacent to the area of venous compression has empirically been shown to help weaken a muscle or decrease spasm for several months and thereby reduce compression pressure on the obstructed vessel.[7]

PERFORMING THE CORRECTIVE FASCIOTOMY FOR CHRONIC EXERTIONAL COMPARTMENT SYNDROME

Once you have established the CECS diagnosis and conservative care measures have failed, you should proceed to the corrective fasciotomy. For a certain period of time, I

performed the fasciotomies with a small incision, endoscopically assisted. However, I was not able to fully release the deep posterior compartment and identify unexpected accompanying pathology that could better be identified in the modified open technique herein discussed.

Apply a well-padded thigh tourniquet, but it is not necessary to inflate it unless an abnormal bleeder is present during surgery. General laryngeal mask airway (LMA) or other anesthesia is obtained. Either a popliteal nerve block is performed or one can block the common peroneal and saphenous nerves proximally. A sterile scrub, prep, and drape is always performed. I prefer to use the single incisional approach, which I have modified from that of Mubarak and Hargens.[2]

For the anterior and lateral compartments, make a linear longitudinal incision approximately 8 cm long, midway up the leg between the tibial crest and fibula shaft (**Fig. 7**). Sharply dissect the incision to the level of the subcutaneous tissues down to the layer of the overlying fascia. In addition, one should finger sweep dissect the subcutaneous tissues away from the fascia, so that an unobstructed cut of the fascia may be performed (**Fig. 8**). Right angle retractors such as Army-Navy or lighted breast retractors should be used to help visualize the fasciotomy. You should be able to identify the intermuscular septum separating the anterior and lateral compartments (**Fig. 9**). Meticulously dissect the septum with tenotomy or small Metzenbaum scissors, until you locate the superficial peroneal nerve, which may be found within the intermuscular septum (**Fig. 10**) or just lateral to the intermuscular septum within the lateral compartment. Occasionally, this nerve runs just anterior to the intermuscular septum within the anterior compartment. Inspect this nerve for any obvious signs of pathology, entrapment, or adhesions and free them if necessary.

The anterior compartment will lie anterior to the intermuscular septum. Make a nick with a scalpel blade into the overlying fascia. Using a 10-inch to 12-inch Metzenbaum straight scissor, cut the overlying anterior fascia proximally toward the patella tubercle and distally toward the anterior aspect of the lateral malleolus (see **Fig. 7**). This cut is made by using the sharp portion of the scissors distally, holding the scissors open 1 cm and sliding the scissors along the fascia to cut it much like a tape cutter is used to remove athletic tape.

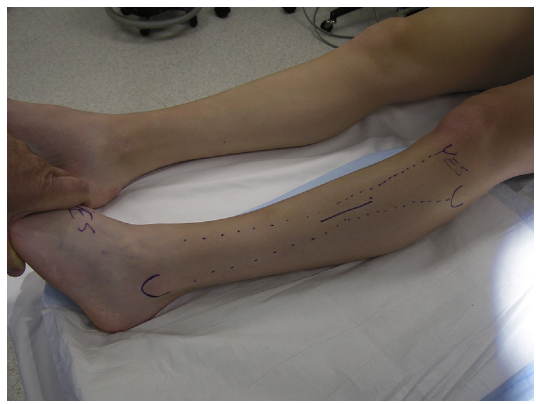

Fig. 7. Anterior and lateral fascial compartment incision. Dotted lines indicate fasciotomy: anterior compartment fascia is cut proximally toward patella tubercle and distally toward anterior aspect of lateral malleolus. Lateral compartment fascia is cut proximally toward fibular head and distally toward posterior aspect of lateral malleolus.

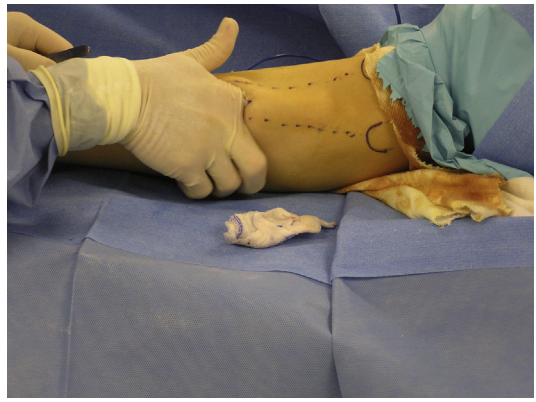

Fig. 8. Finger sweep dissection.

Release of the lateral compartment is performed after a nick is made in the fascia, lateral/posterior to the superficial peroneal nerve. The scissors are directed proximally toward the fibular head and distally toward the posterior aspect of the lateral malleolus (see **Fig. 7**). It is important to visualize and slide the tips of the scissors so that only the fascia is released and one stays posterior to the superficial peroneal nerve. By keeping the tips of the scissors along the fascia, one also avoids cutting the superficial branches of the peroneal nerve. While cutting the fascia, a slight upward pressure should be maintained to avoid cutting muscle tissue, which causes bleeding, and is also why blunt-tipped scissors are appropriate (**Figs. 11** and **12**).

To release the superficial posterior and deep posterior compartments, I recommend making one 8-cm-long linear longitudinal incision approximately 2 cm posterior to the palpated medial posterior margin of the tibia (**Fig. 13**). The incision is deepened to the level of the fascia. The subcutaneous and fatty tissues are separated from the overlying fascia using finger sweep dissection. At this time, one should be able to identify the saphenous nerve and vein, which is typically just posterior-medial to the tibia shaft overlying the deep posterior compartment. Check the saphenous nerve for overlying adhesions and free them if necessary. Retract the saphenous nerve and vein anteriorly

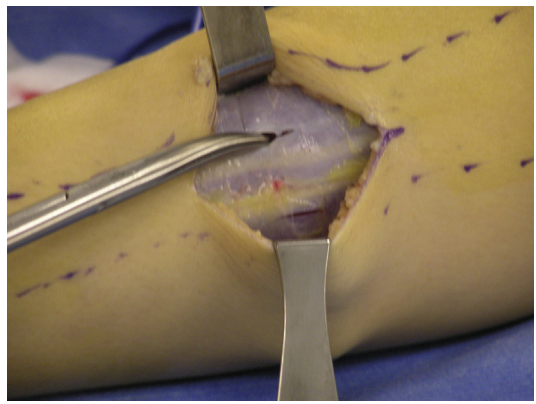

Fig. 9. Anterior compartment, intermuscular septum, and lateral compartment.

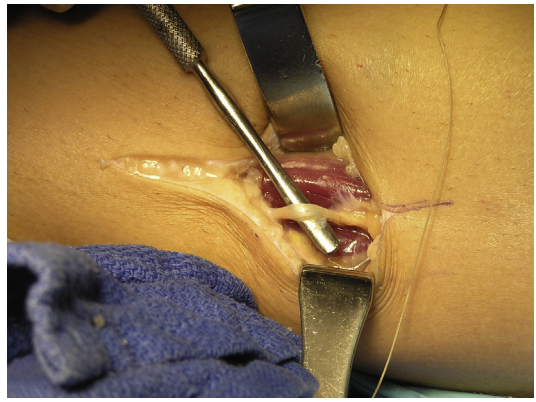

Fig. 10. Superficial peroneal nerve within the intermuscular septum.

and carefully make a superficial transverse incision and, using tenotomy or small Metzenbaum scissors, identify the intermuscular septum between the deep posterior and superficial posterior compartments.

The superficial posterior compartment fasciotomy is then performed by making a nick into the fascia over the medial portion of the soleus muscle (mid leg) and then directing the scissors proximally and diagonally across or along the medial head of the gastrocnemius staying posterior to the intermuscular septum (see **Fig. 13**). You should free the superficial posterior fascia as far distal as the musculotendinous junction. Additional release of fascia may be performed to the lateral gastrocnemius head if needed, being careful to avoid the deeper sural nerve.

To release the fascia encasing the deep posterior compartment, make a nick in the fascia, and slide the scissors proximally in a linear longitudinal manner aiming the scissor tips proximally, straight upward toward the tibial crest staying anterior to the intermuscular septum and then distally downward staying posterior to the medial malleolus (see **Fig. 13**). Be sure to retract the saphenous nerve and vein anteriorly during the proximal cut. While releasing the deep posterior compartment, it may be necessary to release the "soleus bridge," which is a connection of the soleus muscle via soft tissue to the

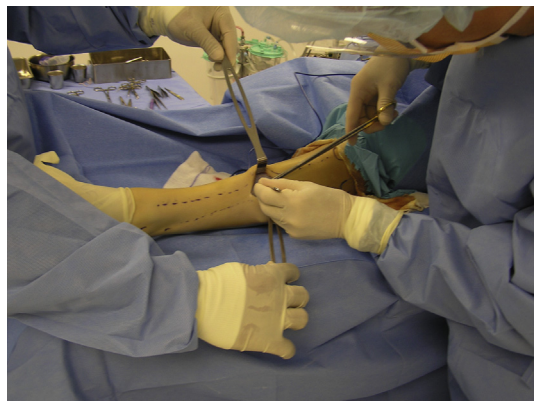

Fig. 11. Performing fasciotomy with 10-inch to 12-inch Metzenbaum scissor.

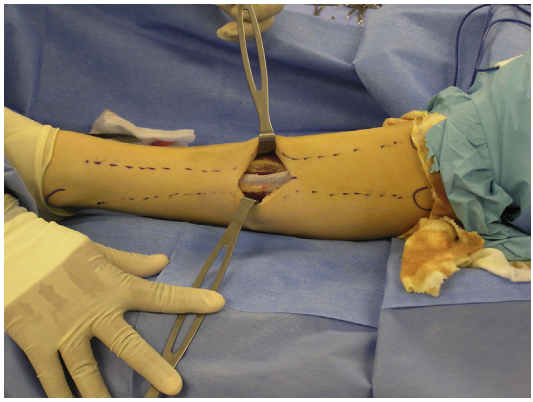

Fig. 12. Completed anterior and lateral fasciotomy.

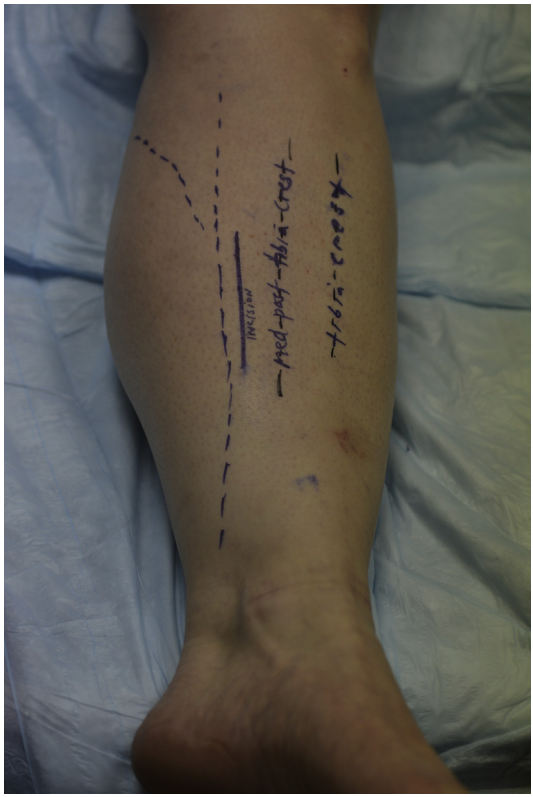

Fig. 13. Superficial and deep posterior fascial compartment incision. Dotted lines indicate fasciotomy: superficial posterior compartment fascia is cut proximally diagonally across or along the medial head of the gastrocnemius. Deep posterior compartment fascia is cut proximally toward the tibial crest and distally toward the posterior aspect of the medial malleolus.

periosteum of the mid tibia. This should be performed bluntly with a finger sweep motion or with the use of a blunt elevator to free the area between the soleus and the tibia.

If there is concomitant chronic shin splint pain, then one should palpate the periosteal tissues along the tibia where complaints of pains have been identified. If there are any areas of fibrosis or scarification noted, this area of periosteal tissue may be excised.[8] During surgery, I have often noted micro-nodules along the medial distal tibia border. After excising the nodules or stripping a small amount of periosteal tissue, I rasp the area to smooth any sharp or irritating border of the tibia. I believe that rasping the bone also promotes regrowth or adherence of sharpey's fibers or periosteal tissue to the overlying soft tissues.

Additionally, if there is any concomitant gastrocnemius equinus deformity then the medial gastrocnemius aponeurosis is cut in a transverse manner from medial to lateral just distal to the gastrocnemius muscle (make sure to see the soleus muscle deep to it) until the appropriate ankle dorsiflexion is achieved. Usually no more than 2.5 cm is transected medially to avoid exorbitant weakening of calf plantar flexion.

The surgical sites are then irrigated with sterile saline solution. The fascia is not sutured. It is my preference to insert a closed suction drain for 24 to 48 hours for the posterior compartment and another for the anterior/lateral compartments to help reduce the incidence of hematoma. The subcutaneous tissues are closed in standard technique with absorbable suture. Staples are often used for the skin. The surgical site is dressed with sterile gauze and elastic bandages. The patient is placed in a removable below-knee walker-type cast or well-padded posterior splint cast.

POSTOPERATIVE CARE

The patient follows up within 24 to 48 hours for removal of the closed suctioned drain. Patients are then instructed on range-of-motion exercises to be performed within a few days of surgery, several times per day. This includes toe flexion and extension exercises, ankle range of motion exercises, and knee flexion and extension exercises. Patients remain in a walker-type boot cast non–weight bearing for 3 weeks. Suture and/or skin staples are removed at approximately 3 weeks and then patients attend rehabilitation. Patients may perform upper body exercises and/or cycling soon after the surgery, but must elevate their legs afterward to compensate for any swelling.

POSTOPERATIVE COMPLICATIONS

In my experience the most common complication has been hematoma formation. This may be evident from dissecting through small vessels or due to inadvertent cutting of muscle tissue while performing the procedure(s). A surgeon can either perform the procedures wet and cauterize any bleeders as encountered, or use a tourniquet and let it down before closure to check for bleeders. Hematoma (and seroma) formation delays the healing process and has a higher incidence of infection. One must be aggressive with physical therapy when a hematoma or seroma is identified and apply heat packs, massage, and electrical muscle stimulation to assist in breaking up the hematoma. If necessary, aspirations should be performed to diagnose either hematoma or seroma and to relieve pressure and allow for better soft tissue healing. To be on the safe side, I have been using a closed suction drain in all recent cases and have not had any more issues with hematoma formation.

Wound dehiscence is another complication, especially when performing additional surgery for chronic shin splints that requires dissection along the posterior-medial tibia. Wound dehiscence can delay surgical healing, leave a hypertrophic or unsightly scar, and expose the soft tissue to infection. It is usually caused by significant swelling

with tension on the skin or improper closure of the skin. If the wound margins gap open, then it is important to bring the patient back to the operating room and undermine the skin. Release the tension and then apply either retention sutures or an external tissue expander device. This is usually augmented with new sutures applied after a few days and the retention sutures or tissue expander removed.

During surgery, patients are usually placed on intraoperative intravenous antibiotics, and after surgery they continue with prophylactic oral antibiotics, so infection has been rare. Localized skin hypoesthesia is often a complication. This is a fairly common complication after any leg surgery. It is possible that during the procedure, the surgeon may cut a small aberrant nerve branch or small nerve(s) within the skin incision site are sacrificed. As with any surgical procedure, potential risks and complications must be discussed and documented with the patient before the surgery.

In summary, increased tissue pressure within a fascial compartment may be the result of any increase in volume within its contents or any decrease in size of the fascial covering or due to any distensibility of the fascial covering (eg, patients with thickened fascia). Therefore, the initial clinical diagnosis of CECS is made by a careful history and by exclusion of other maladies and confirmed by compartmental syndrome testing. Once the practitioner makes the proper diagnoses of CECS, through clinical examination and intracompartmental testing, surgical fasciotomy along with ancillary procedures should allow the athlete to return to competitive activity.

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