Chronic Compartment Syndrome

An Unusual Cause for Claudication

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Chronic Compartment Syndrome (CCS) is usually caused by overuse injury in well-conditioned athletes (particularly runners). Less common causes of CCS include blunt trauma, venous insufficiency, and tumor. CCS is clinically manifested as claudication, tightness, and occasional paresthesia. Unlike other forms of overuse injury (tendonitis, stress fracture), CCS does not respond to rest, anti- inflammatory medications, or physical therapy. The diagnosis of this condition is confirmed by elevated compartment pressures (normal < 15 mmHg; CCS > 20 mmHg). The only effective treatment is surgical compartment release. Two hundred nine patients have been surgically treated for CCS, 100 by subcutaneous fasciotomy (group I) and 109 by open fasciectomy (group II). These procedures were usually performed in ambulatory surgery using local anesthesia. Patients treated by open faciectomy instead of subcutaneous fasciotomy had fewer early postoperative wound complications (6% *vs.* 11%) and fewer late recurrences (2% *vs.* 11%).

INTERMITTENT CLAUDICATION IS commonly associated with elderly persons and is rarely diagnosed in young healthy adults with chronic leg pain. Atypical claudication can occur without clinical evidence of venous insufficiency or arterial occlusive disease because of a condition called Chronic Compartment Syndrome (CCS).[1-4] CCS is most commonly associated with overuse injury in well-conditioned athletes (particularly runners), but it can occur in patients with blunt limb trauma, or after acute and/or chronic venous insufficiency. Unlike other exertional injuries such as periostitis, stress fracture, or tendonitis, CCS does not respond to rest, anti-inflammatory medications, or physical therapy. The only effective therapy is surgical compartment release. Symptoms of CCS include severe muscle cramping, tightness, and occasional paresthesias that can seriously effect athletic levels of performance and in some circumstances can progress to acute neuromuscular injury. Knowledge of CCS is not widespread. However the problem is not uncommon and it is becoming more prevalent in this physical fitness-oriented generation. This report describes appropriate diagnostic tests for patients suspected of having CCS, explains indications for surgical intervention, and presents arguments for using fasciectomy instead of fasciotomy to treat CCS.

Materials and Methods.

Since 1976, 209 patients have been surgically treated for symptoms of CCS at the University of Wisconsin-Madison. The majority were athletes (187, or 89%) referred from our Sports Medicine Department, and the rest had a history of blunt limb trauma (14, of 7%), venous insufficiency (6, or 3%), or soft-tissue tumor (2, or 1%). These patients (96 male, 113 female) were young (mean age, 22 years) and had long-standing symptoms (mean, 24 months) that would abate or completely disappear after extended rest but reappear again with exercise. Medical treatment, including physical therapy, icing, and the use of anti-inflammatory medicines failed to significantly alter the severity of symptoms until physical activity was restricted. The most common symptoms included claudication (188 patients, or 90%), muscle group tightness (125 patients, or 60%), and parasthesias (52 patients, or 25%). The diagnosis of CCS was based on clinical history and confirmed in most cases by elevated compartment pressures. Although the Whiteside technique has been routinely used to measure compartment pressures, we prefer to use the hand-held Stryker Digital Computer System, which provides an accurate and less cumbersome method

Presented at the 109th Annual Meeting of the American Surgical Association, Colorado Springs, Colorado, April 10–12, 1989.

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Accepted for Publication: April 17, 1989.

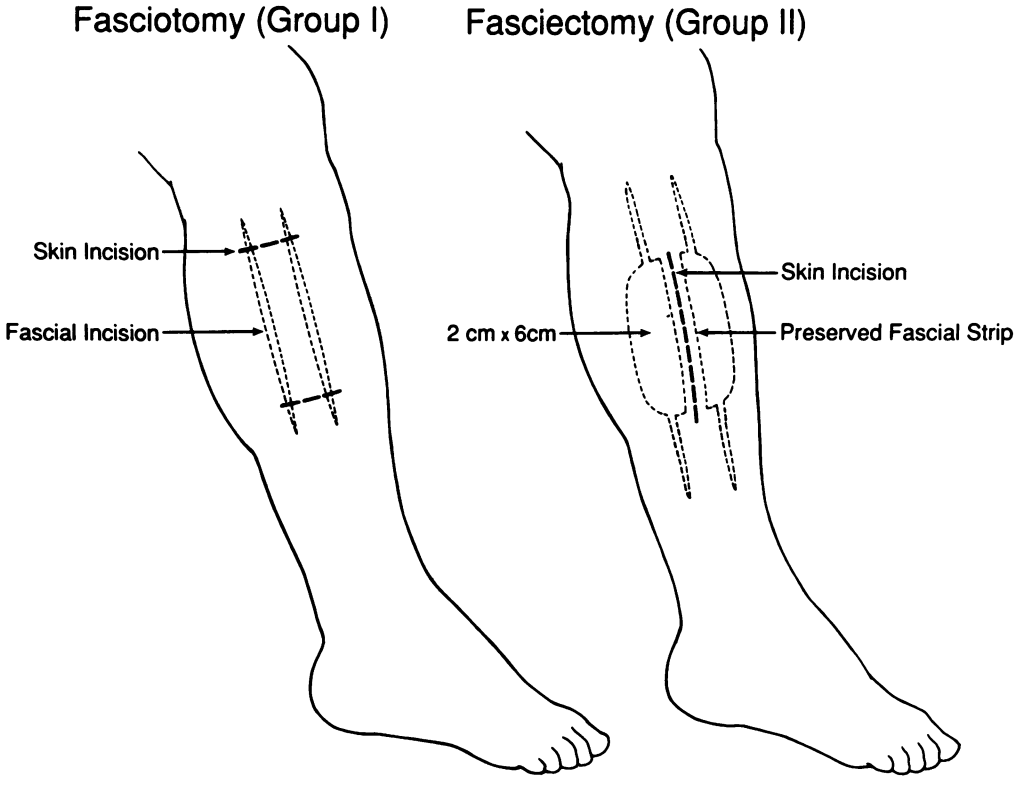


FIG. 1. Surgical compartment release techniques for the anterior and lateral compartments. Group I: Subcutaneous fasciotomy is performed using transverse skin incisions proximal and distal over the symptomatic compartment. Compartment fascia is incised by subcutaneously passing the scissors between the skin incisions. Group II: Open fasciectomy is performed using one linear incision over the medial one third of the anterior lateral surface of the leg. An elipse of fascia approximately 6 cm long and 2 cm wide is removed from the anterior and the lateral compartment, leaving a strip of fascia over the intermuscular septum. This strip protects the superficial peroneal nerve from injury or scar adherence. Extended compartment release can be achieved by proximal and distal subcutaneous fasciotomy performed under direct vision.

of obtaining compartment pressures.[4] Our experience is that normal resting compartment pressures in the lower leg are less than 15 mmHg, borderline pressures are between 16 and 20 mmHg, and pressures above 25 mmHg are markedly abnormal and uniformly consistent with CCS.[2,3,5] If patients have borderline pressures, repeat measurements should be performed after they have undergone treadmill stress or have run until symptoms develop. Patients with incipient CCS will show marked postexercise increases in compartment pressure and a prolonged return to the baseline (> 10 minutes). Accurate diagnosis of CCS requires that occult vascular disease be excluded as a cause of claudication symptoms. Noninvasive testing is routinely done to rule out vascular disorders that may occur in young adults, including premature atherosclerosis, popliteal entrapment, and chronic venous insufficiency. Resting and postexercise Pulse Volume Recordings (PVR), which provide plethysmography data and segmental limb pressure indices, are used to screen for intrinsic arterial occlusive disease. The PVR technique can also be used to screen for extrinsic compression of the popliteal artery (popliteal entrapment) by having the patient extend the knee and then dorsi flex and platar flex the foot. We performed arteriography when results of arterial screening tests were abnormal. We performed venous Doppler and Impedence Plethymography (IPG) tests on all patients who had limb swelling in conjunction with their complaints of claudication to rule out venous insufficiency.[6] Diabetic screening and EMGs were performed on patients with paresthesias to detect any peripheral nerve injury. Bone scans were performed selectively on patients who had bone pain along with muscle complaints to identify coexistent periostitis or stress fractures.[7] MRIs were done when patients were suspected of having post-traumatic intramuscular hematomas or CCS symptoms associated with a tumor mass.

We used surgical compartment release to treat patients with CCS when athletically induced symptoms persisted despite prolonged rest and aggressive medical management, or when the severity of claudication complaints worsened to the point that the patient's routine daily activities were adversely affected. The onset of paresthesias and the development of resting pressures greater than 25 mmHg were indications for prompt compartment release. Recreational athletes who developed CCS symptoms were encouraged to change sports or at least to modify the intensity and duration of their workouts as an alternative to surgery. In our experience most individuals involved in competitive athletics were unwilling or unable to accept behavior or training modification as a permanent means of controlling their clinical symptoms.

We performed surgical compartment release by subcutaneous fasciotomy in 100 patients (group I) and by open fasciectomy in 109 (group II) (Fig. 1). The superficial compartment releases in group I (anterolateral, posterior superficial) and the distal deep posterior releases were

performed as out-patient procedures in ambulatory surgery using local anesthesia, while the proximal deep posterior releases were done as in-patient procedures. We performed 77 ambulatory surgical procedures; 23 were done the same day the patient entered the hospital. Subcutaneous fasciotomy was performed using transverse skin incisions placed strategically at the proximal and distal limits of the compartment to assure complete release. Linear skin incisions were only used to release the deep posterior compartments. Mean follow-up for this group is now 48 months.

Open fasciectomy was used to treat primary compartment syndromes as well as recurrences after subcutaneous fasciotomy in 109 patients (group II). Open fasciectomy was performed through linear incisions made parallel to the long axis of the compartment. In most cases we used local anesthesia. The linear skin incision provided better exposure and allowed for generous excision of fascia and precise control of muscle bleeding points, and made peripheral nerve injury much less likely. An ellipse of fascia 6 cm to 8 cm long and 2 cm to 3 cm wide was routinely excised and combined with extended subcutaneous fasciotomy under direct vision, assuring complete compartment release (Figs. 2A and B). Mean follow-up for this treatment group is now 36 months.

Results

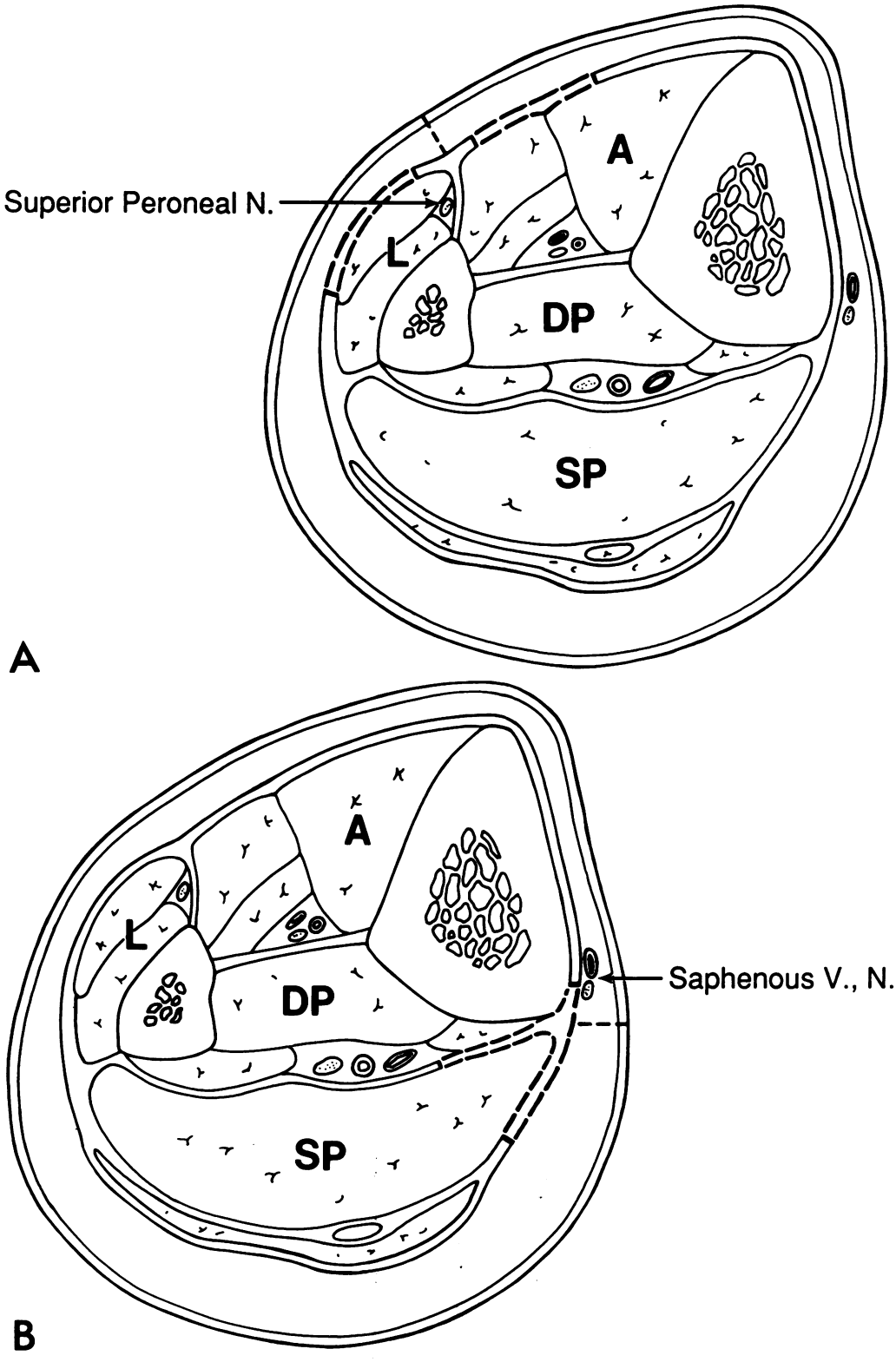
Most of the patients treated for CCS were athletes (187 patients, or 89%) who developed symptoms because of overuse injury. Other causes for CCS included blunt trauma, venous insufficiency, and soft-tissue tumor. In all we surgically released 511 symptomatic compartments. CCS was unilateral in 87 (41%) patients and bilateral in 122 (59%). The distribution of symptoms among the 511 affected myofascial compartments was as follows: anterolateral 245 (48%), deep posterior 204 (40%), and superficial posterior 62 (12%). Paresthesias occurred in 52 patients (25%) and were most commonly associated with the deep posterior compartment (39 patients) or anterolateral compartment (13 patients).

Although we routinely performed noninvasive vascular tests, only ten patients (5%) showed abnormal results. (Six abnormal IPGs suggested severe venous insufficiency, and four positional PVR changes suggested possible popliteal entrapment.) Angiograms performed on the patients with abnormal positional PVR tests showed no evidence of anatomic entrapment, but they did show external compression of the popliteal artery at the level of the soleus muscle in the deep compartment. Bone scans were performed on 20 patients who had bone pain (10%). Ten scans were positive for stress facture (four) or periostitis (six), and ten were negative. Patients with positive bone scans were not candidates for compartment release unless symptoms persisted after acute bone scan findings normalized. MRI scanning was useful for demonstrating intramuscular hematomas associated with blunt trauma (four patients) and for diagnosing soft-tissue tumors associated with CCS (two patients: one neurofibroma, one sarcoma).

In group I subcutaneous fasciotomy was performed on 100 patients with 233 symptomatic compartments. Eleven patients (11%) had 17 intraoperative or early postoperative complications. The most serious of these was a transection of the posterior tibial artery during a distal deep posterior compartment release. This vascular injury was successfully treated with an interposition vein graft. The most common problems were wound hematomas (5) and infections (4), but the most bothersome for patients were cutaneous sensory nerve injuries (3) (superficial peroneal nerve, saphenous nerve, and sural nerve). Other complications included thrombophlebitis (1), lymphocoele (3), and seroma (1). A total of 55 postoperative hospital days of care were required to treat these problems. Eleven patients (11%) developed recurrent symptoms in 16 compartments treated by fasciotomy. One third of these occurred within 3 months of surgery and were related in most cases to postoperative hematomas. The rest occurred within one year as a result of fascial scarring or herniation of muscle through the fasciotomy site. Most of the recurrences (13 of 16, or 80%) involved the anterolateral compartments and were successfully treated by an open fasciectomy. New symptoms developed in the distribution of previously unaffected compartments in five patients (5%).

In group II, open fasciectomy combined with selective extended subcutaneous fasciotomy was performed in 109 patients with 278 symptomatic compartments. Six patients (5.5%) developed eight postoperative wound complications (hematoma 5, cellulitis 2, seroma 1) that required a total of four in-hospital postoperative treatment days. There were no vascular or cutaneous sensory nerve injuries. Three patients developed delayed peripheral neuropathy (2 transient, 1 permanent) secondary to wound scar nerve entrapment. Two patients (2%) developed recurrent symptoms in the distribution of previously treated compartments (deep posterior). New CCS symptoms developed in untreated compartments in six patients (5.5%). These were successfully treated with open fasciectomy.

The best clinical results, regardless of the surgical release technique used (fasciotomy or fasciectomy), were obtained in patients who developed chronic compartment symptoms as a result of overuse injury. One hundred sixty-five of 177 patients in this category (93%) were completely cured. Four of the six patients with venous insufficiency (66%) were completely relieved of their compartment symptoms after open fasciectomy and the postoperative use of compression support with elastic stockings. Two



FIGS. 2A and B. These cross-sectional illustrations of the distal lower extremity muscle compartments (L = lateral compartment, A = anterior compartment, DP = deep posterior compartment, SP = superficial posterior compartment) demonstrate locations of fascial excisions required for release of the anterolateral and posterior compartments.

The superficial peroneal and long saphenous nerves are cutaneous branches that can be easily traumatized or encased in wound scar unless they are protected. Postoperative neuritis can negate any benefit derived from successful compartment release. Fascia overlying the intramuscular septum should be perserved so as to protect the superficial peroneal nerve from direct trauma or scar encasement. When a medial approach is used for posterior compartment releases, skin incisions should be placed posterior to the saphenous vein and nerve, and dissection around these structures should be avoided to reduce the probability of intraoperative trauma or postoperative scar entrapment.

other patients had minor relief. Only 8 of 14 patients who developed CCS as a complication of blunt trauma to the lower extremity (57%) and 1 of 2 patients with CCS symptoms related to expanding soft-tissue tumors in the lower extremity (50%) were cured by surgical compartment release and tumor excision.

Histologic examinations of the excised fascia specimens demonstrated increased thickness without inflammation

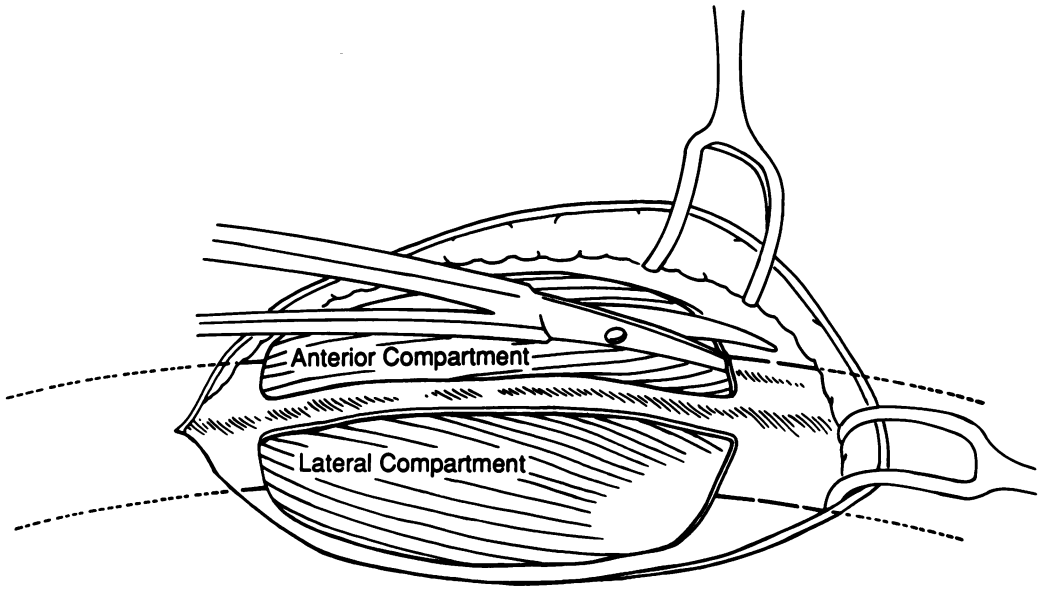


FIG. 3. Open faciectomy with extended subcutaneous fasciotomy is the procedure of choice to release the anterior and lateral compartments because it allows a more complete and precise decompression and reduces the likelihood of scar recurrence. Postoperative bleeding and neurovascular injuries have been significantly reduced because of the better exposure provided by a linear skin incision.

in 40% of cases, inflammatory changes with muscle scarring in 14%, and normal-appearing fascia without obvious abnormalities in 46%.

Despite technical differences in the extent of the surgery, there were no significant recovery differences between the two groups for uncomplicated cases. Patients returned to work or school and were ambulatory within 48 hours. Postoperative swelling was common and required elastic compression support for ten days to 2 weeks. Stretching exercises were started 1 week after surgery, and most patients were jogging 4 or 5 weeks after surgery. Return to full athletic capacity took 2 to 3 months.

Discussion

CCS is underdiagnosed and should be considered in young claudicants with normal vascular studies. CCS most commonly results from overuse injury in distance runners and athletes exposed to significant impact stress to lower extremities (basketball and football players, gymnasts and hurtlers). Occasionally, however, CCS may develop as a complication of blunt trauma, venous hypertension, or soft-tissue tumor expansion in lower extremities. Unlike the acute compartment syndrome, CCS rarely causes permanent neuromuscular injury, probably because discomfort restricts the patient's activity enough to prevent a prolonged increase in compartment pressure. Although abnormally elevated compartment pressures (>15 mmHg) occur in most patients (85%) with CCS, pressures much above 25 mmHg are uncommon in patients who are asymptomatic at rest. In symptomatic acute compartment syndromes, pressures usually exceed 40 mmHg. Compartment pressures in normal patients can increase to three or four times baseline values after vigorous activity, but they rapidly return to normal within a few minutes. Maximum postexercise pressures rarely exceed 50 mmHg. In patients with CCS, abnormally elevated baseline pressures may triple or quadruple after strenuous exercise, raising maximum compartment pressure to as high as 100 mmHg. Sustained pressures of this magnitude can cause circulatory damage to muscles and nerves. Rapid pressure decline with restricted activity probably explains why more patients do not progress to an acute form of the compartment syndrome.[2,5,7,8] It should be pointed out that diagnostic pressure measurements are safe and easy to perform when the anterolateral and posterior superficial compartments are being evaluated. However blind transcutaneous pressure measurements are not recommended for assessment of deep posterior compartments because of the potential for neurovascular injury.

It is reasonable to assume that the symptoms of claudication, parasthesia, and postexercise muscle group tightness associated with CCS are caused by local neuromuscular compression and ischemia. Increased muscle compartment pressure can be caused by several different factors, including muscle hypertrophy, altered fascia compliance associated with changes in fascial thickness and elasticity, myofacial scarring, venous hypertension, and post- traumatic soft-tissue inflammation. Unlike exertional injuries such as stress fracture, tendonitis, or periostitis, CCS can not be cured by nonsurgical methods of treatment. Although CCS symptoms improve temporarily with rest, anti-inflammatory medications, and physical therapy, symptoms quickly return once vigorous physical activity is resumed.[8-11] The only effective treatment for this condition is surgical compartment release. The objective of surgical therapy is to permanently reduce intracompartmental pressure. The technique most com-

monly used to treat CCS is subcutaneous fasciotomy. More recent clinical experience suggests that open fasciectomy is a safer and more effective treatment.[12] Open fasciectomy has fewer postoperative complications and fewer recurrences than blind subcutaneous fasciotomy. Open fasciectomy is done through a skin incision made parallel to the long axis of the muscle compartment. This incision improves exposure, makes identification of anatomic structures more precise, allows for direct control of bleeding points, and makes it much easier to perform a fasciectomy and extended subcutaneous fasciotomy under direct visualization. Better exposure reduces the hazards of intraoperative trauma to neurovascular structures. Fasciectomy combined with extended subcutaneous fasciotomy allows for complete decompression of the muscle compartment and greatly reduces the possibility of a recurrent compartment syndrome secondary to postoperative scarring (Fig. 3). Although fasciectomy is a more extensive procedure than fasciotomy, postoperative hospitalization, early complication rates, and late recurrences have been significantly reduced with little or no effect on the time required for rehabilitation.

We are now conducting prospective studies to determine whether there may be long-term functional differences between clinically successful fasciectomy and fasciotomy procedures. Our impression thus far is that no significant reductions in strength or endurance occur as a result of fasciectomy. Physiologic and histochemical evaluations of fascia from patients with CCS are being compared with specimens taken from comparable age groups requiring fasciectomies as part of orthopedic reconstructive surgery. We hope that these studies will allow us to better understand why some patients develop CCS and why others do not, and allow us to determine whether long-term functional differences between fasciotomy and fasciectomy exist.

Clinicians should be aware of CCS and consider it as a possible cause of claudication in young adults. This condition will not improve without surgery. Surgery is more effective for patients with CCS caused by overuse injury than for patients with CCS caused by trauma, venous hypertension, or tumor. Open fasciectomy causes fewer complications and fewer recurrences than closed subcutaneous fasciotomy and should be considered the surgical procedure of choice for treating CCS.

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Discussion

Dr. Robert W. Barnes (Little Rock, Arkansas): I am indebted to Dr. Turnipseed and his colleagues for providing me with one more reason not to jog. I am pleased that they have apprised us of this unique disorder that is somewhat analagous to mimo-causalgia or sympathetic dystrophy, which likewise can cause chronic, disabling, painful syndromes, which are often unrecognized by physicians and which can also be cured with appropriate therapy.

In reviewing the manuscript, I have three concerns about this study and three questions for the authors.

My first concern relates to the fact that we are not provided with detailed results of compartmental pressures at rest or after exercise in normal individuals and asymptomatic athletes or other subjects matched for age, sex, and causative factors similar to the patients reported. Second the method of determining the deep posterior compartmental pressure is not clear. My final concern relates to the implication that open fasciectomy is superior to semiclosed fasciotomy in the absence of evidence that these procedures were carried out concurrently or at random and in the absence of data reported in life table format.

My first question is to ask if the authors could correlate the development of symptomatic chronic compartment syndrome with inadequately supervised acute exposure to excessive muscular exercise. Second has the recent abuse of anabolic steroids contributed to an increased incidence of this syndrome? Finally, how can one adequately decompress the deep posterior compartment by fasciectomy under local anesthesia?

Dr. F. William Blaisdell (Sacramento, California): I have a problem with this syndrome.

It is hard for me to imagine that muscle hypertrophy would be such as to crowd a compartment because the fascia should dilate or stretch in response to any chronic muscle enlargement.

I could accept the problem if it is the result of some type of fascial abnormality, that is, injury to the fascia that resulted in thickening so that the fascia could not enlarge as muscle hypertrophy develops.

I would ask whether there was any evidence on pathologic examination of the specimens that the fascia was abnormal. Also were any studies done of the fascia using ultrasound or CT that might have indicated if there was fascial abnormality?