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Chronic compartment syndrome: Diagnosis, management, and outcomes

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ABSTRACT

A consecutive operative series of 100 patients with chronic compartment syndrome involving 233 compartments is reported. Seven of every eight were athletes, and runners predominated. Exercise-induced symptoms of consistently recurring tightness, aching (in some, sharp pains) in anatomically defined compartments were pathognomonic. Mean months of symptoms prior to operation was 22; median age was 26 years. Bilaterality occurred in 82. The distribution of compartments was: anterior, 39%; lateral, 12%; and posterior, 48%. Incidental compartment pressures were elevated ($\bar{x}$ = 23 mmHg). Fasciotomy using local anesthesia was performed on 70 outpatients. At a median of 4.5 months, over 90% were cured or significantly improved in symptoms and/or function. Median time to walking unassisted was 2 days, and to resumption of conditioned running 21 days. Fasciotomy can be a safe, effective, and economical treatment for chronic compartment syndrome.

Chronic compartment syndrome (CCS) is most typically an exercise-induced condition characterized by a relative inadequacy of musculofascial compartment size producing chronic or recurring pain and/or disability. The chronic form of compartment syndrome is relatively recent in description and treatment, having been described as a syndrome and successfully treated surgically by Mavor in 1956.[11] The condition is also known as "recurrent compartmental syndrome,"[30] and "subacute" or "exertional compartment syndrome."[25] Mubarak and Hargens[13] prefer the term "chronic compartment syndrome," and we concur.[3] Most cases of CCS involve the leg, but a few reports exist describing the syndrome in the hand,[20] forearm,[29] and thigh.[19]

This series reports only cases involving the leg and represents a personal (DED) consecutive operative series of 100 patients treated between 1976 and 1984. One subacute case involving the forearm was treated during this period but is not included in this report.

There remain a number of questions and controversies relating to CCS. One of these involves chronic shin splints, or the "medial tibial syndrome" (MTS).[12,28] During the period of this study a number of patients with MTS were also diagnosed and treated. Several of them met the criteria of Mubarak's periostitis[12] or had stress fractures. Others fit the criteria of deep posterior CCS as described by Puranen,[18] and by Bryk and Grantham.[2] The latter group of patients is included in this series, while the former is not. A more detailed report of the MTS experience is forthcoming.

This report will present demographic data and describe the diagnosis, surgical management, operative and pathologic findings, and outcomes associated with the first author's initial 100 patients treated operatively for this condition at the University of Wisconsin Hospital. A rehabilitation program has also been developed and tested and is available upon request. Like the diagnosis of intermittent claudication, the diagnosis of CCS is primarily made on the basis of a careful history. The absence of any significant physical abnormalities beyond occasional muscle hernias and muscle soreness to palpation of the involved compartment(s) is striking. Indeed, this aspect of the syndrome, combined with the lack of awareness of its typical historical features, account for our perception that CCS is currently underdiagnosed.

CCS is predominately a problem of active people. Eighty-seven percent of patients in the series were involved in sports. Activities were distributed as follows: amateur, 36; professional, 4; recreational, 47; work-related, 2; nonsport,

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nonwork, 11. Of the athletes, runners predominated with 69 cases and were distributed as follows: track [not otherwise specified (NOS)], 17; cross-country, 11; marathon, 6; sprint, 1; steeplechase, 1; running (NOS), 33. Other sports included: racquet sports, 9; and ball/puck, 19. Nonrunning sports included: skiing, 6; golf, 4; crew, 3; gymnastics, 2; boxing, 2; dancing, 1; fencing, 1; and figure skating, 1. Many of the athletes were active in several sports. Two nonathletes described the onset of symptoms following acute development and then disappearance of a popliteal mass, apparently a Baker's cyst which triggered a compartment syndrome due to venous outflow obstruction. Two others presented late following fracture of either the tibia or fibula. Seven cases developed following knee surgery or casting.

In this series the condition occurred in nearly equal sexual distribution (51 males, 49 females); this is atypical of many series, in which males predominate. The median age of patients at the time of onset of symptoms was 20 years. The median age at operation was 23 years. Means for these parameters were 23.5 ± 9.2 and 26.1 ± 10.2 years, respectively. The reported number of physicians seen prior to a definitive diagnosis being made averaged 2.4 and ranged from 1 to 10. The median number of months with symptoms was 12 with a mean of 22 months.

Leg problems were not new to the athletes included in the study, nor were efforts to treat their syndrome nonoperatively. Associated extremity complaints were noted in one-third of patients. Orthotics were tried by 41%, and were considered somewhat helpful by 15%. A past or present history of stress fracture(s) was noted in 16%. Eighteen percent reported having had bone scans, and half of those reported having had positive scans. Stretching programs were widely used but were not considered helpful. Physical therapy modalities were used by 40%, and medications by 49%. One-third of patients described these efforts as being only somewhat helpful.

Anatomical textbooks describe four compartments in the leg. However, our experience suggests that the arrangement of muscles within these four compartments is such that CCS can develop within one subdivision of a compartment without involving other muscles within the same compartment. Since this is the case, we believe at present that appropriate clinical management requires one to conceptualize the leg as having seven functional compartments. These include the anterior, lateral, posterior superficial medial (medial head of gastrocnemius), posterior superficial lateral (lateral head of gastrocnemius), posterior deep proximal, posterior deep distal (flexor digitorum longus, flexor hallucis longus, and posterior tibialis), and posterior superficial distal (distal soleus). The 233 compartments released in our 100 patients were distributed variously among these seven compartments (Table 1).

If one knows clearly the boundaries of these functional compartments, one is much better prepared to evaluate these patients accurately. The distinction between soleal involvement and distal deep posterior involvement is, at times, a bit unclear, and compartment pressures, when elevated in the deep distal posterior compartment, may even be falsely elevated as a result of soleal involvement.

TABLE 1
Distribution of compartments released[a]

Compartment	Right	Left	Percentage
Anterior	46	46	40
Lateral	10	19	12
PSM[b]	9	9	8
PSL[c]	9	11	9
PDP[d]	17	17	15
PDD[e]	19	20	17
PSD[f]	1	—	0.4

[a] Study group: 233 compartments (100 patients).
[b] Posterior superficial medial.
[c] Posterior superficial lateral.
[d] Posterior deep proximal.
[e] Posterior deep distal.
[f] Posterior superficial distal.

DIAGNOSIS

History

Historical features are critical. The patient is able to localize symptoms precisely to one or more of the seven functional musculofascial compartments (93 patients). Muscle herniations through the fascial windows of cutaneous nerves or perforating veins or actual fascial tears may, in a few patients, mislead one into focusing the patient's symptoms too narrowly. While these areas deserve identification, all patients should be asked to outline with a finger the exact borders to the area of the pain they experience, not just where it may be maximal. An entire compartment or subdivision is nearly always outlined. Some varying overlap between the proximal and distal deep posterior compartments exists which reflects normal variation; the others are quite precisely traced out. It is important to inquire whether the posterior symptoms are superficial or very deep ["back deep inside behind the bone (tibia)"]. Consistent appearance with exercise is typical (94). Athletic performance was adversely affected by symptoms (98). In three-quarters of the patients, symptoms progressively worsened over a period of weeks or months. Rest or reduction or cessation of exercise improved symptoms in 85% of 75 athletes reporting such an experiment. Indeed, they commonly volunteer that rest is the only thing that consistently helps them relieve symptoms. Aching pain (85), tightness or a squeezing sensation (81), or sharp pains (35) complete the picture.

Mirror image bilaterality is quite common (82), and if symptoms are not mentioned by the patient as being bilateral, the patient should be questioned regarding the opposite leg. Low grade symptoms may be present in the second limb, and single-side fasciotomy will soon require a second procedure on the opposite side as the patient increases his level of exercise subsequent to the initial procedure. A very few cases were diagnosed as being bilateral on the basis of having bilaterally elevated compartment pressures, but most were bilaterally symptomatic with symptoms being equal or dominant in one limb.

The duration of pain following exercise varied as followed: 0 to 2 hours, 13; 2 to 6 hours, 8; 6 to 12 hours, 4; 12 to 24 hours, 6; over 24 hours, 20; constant, 31; and variable or uncertain, 18. Rest so typically improves the symptoms of most patients (while exercise worsens it) that failure to elicit a relationship between these variables in the patient's history greatly lessens the probability of making a correct diagnosis.

Anterior compartment patients may describe transient or persistent low grade foot-drop with or without paresthesias. Paresthesia on the dorsum of the foot with anterior compartment syndrome and in the instep with deep posterior compartment involvement is not too unusual.

Physical examination

Physical findings are decidedly unimpressive in nearly all cases. A few anterior or lateral compartment patients will have weakness of dorsiflexion. The muscles involved may be tender or not, depending upon whether the patient is symptomatic or not. Three-quarters were symptomatic when first examined. Typically, mild soreness to palpation is noted. Women may describe ankle edema which, although mild by usual clinical standards, may still be noted. Pulses are always present with chronic manifestations of compartment syndrome and play no role in the diagnosis. Indeed, absence of pulses would lead the clinician to investigate for other causes of pulselessness. Muscle hernias may be noted and may be either tender or asymptomatic. A seemingly symptomatic muscle hernia may actually be an incompetent perforator in disguise; Doppler evaluation can easily answer the question. The patient in nearly all instances is able to identify accurately which part of the leg has the problem, but having him or her return when acutely symptomatic may be necessary to evaluate the problem accurately in some.

Compartment pressure measurement

Compartment pressure measurement is used to confirm the presumptive diagnosis developed mostly from the historical information. Clearly, the most objective measurement of CCS is the compartment pressure, but as we use it, it also deserves to be evaluated in the context of the patient's symptoms and recent activity. It is definitely advocated since a sensation of fascial tightness by palpation alone is uncommon unless pressures are in excess of 30 to 35 mmHg. Initially, pressure measurement prior to and after an exercise test on a treadmill was used. Catheter wick[15] and saline-needle[31] techniques were evaluated. As experience grew it became apparent that nearly all patients with CCS differentiate themselves from normal persons by having chronically elevated compartment pressures. The upper range of normal also settled at 15 mmHg, using the simple saline-needle technique of Whitesides.[31] The mean pressure, measured in 99 involved compartments which were pain-free when studied, was 21.4 ± 7.7 mmHg (range 8 to 48 mmHg). If the compartment was symptomatic or had pain when studied, the mean pressure was 25.2 ± 11.5 mmHg (range 6 to 38 mmHg). The overall mean pressure was 23 mmHg ($N = 150$). Pressure in the proximal deep compartment was not routinely measured to avoid any possibility of injury to important deep structures resulting from a blind needle stab. Similarly, if pressures were elevated in two or more compartments of multiple symptomatic compartments of the same patient, pressures were not routinely measured in all compartments.

While others have measured proximal deep posterior compartment pressures without expressing trouble with complications, we have been somewhat reluctant to follow their lead and do not measure deep pressures routinely.

The Whitesides compartment pressure measurement technique in experienced hands is an excellent test, but proper experience is needed to assure accurate results. Its low cost and high accuracy make it a very attractive test, but its results need to be interpreted in the context of other information. If in an uncommon instance the pressure is normal and the history is pathognomonic for CCS, surgery may still be recommended. Those with less convincing historical data are advised to return when more symptomatic for the repeat study prior to making a decision; in such instances if the repeat study is still normal, operation is not recommended. Patients who are quite symptomatic and have pressures in excess of 30 to 35 mmHg are now operated upon as promptly as is feasible, and in a very few instances on an urgent basis. Those with pressures near that level are advised to limit their exercise until the procedure can be done. Patients with low grade symptoms despite heavy exercise and near normal pressures are advised to cut back on their exercise levels more or less permanently or pending worsening symptoms, or to consider fasciotomy very electively.

In many patients the decision to operate appears to be a very elective one since it is usually not strictly necessary except to allow the patient to maintain a desired lifestyle. In such circumstances if the procedure entailed substantial cost or risk, such factors would weigh heavily against recommending the procedure. The results reported below are sufficiently good so that patients wishing to remain active may in most instances look forward to returning to their sport if they undergo the procedure. Biopsy data also suggest that ischemic injury of uncertain reversibility may make operative delay unwise. A description of the procedures and a recommended approach to them follows.

OPERATIVE TECHNIQUE

There are a number of descriptions of approaches to fasciotomy.[5,14,17,21,24] One of these is much more radical than is necessary to achieve excellent results.[5] For example, we feel that there is never a situation in which a resection of the fibula is indicated to treat a compartment syndrome, whether acute or chronic. We believe that each compartment can be released safely, efficiently, and with little probability of recurrence if one consistently follows the procedures outlined below.

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All superficial releases were performed on an ambulatory basis except where other coincidental procedures necessitated inpatient care and spinal or general anesthesia. Seventy patients had their procedures under local anesthesia on an outpatient basis. Of the 23 inpatients having compartment releases only, lengths of hospital stay were as follows: 1 day, 5; 2 days, 10; and 3 days, 8.

Regardless of which superficial compartment is being released, it is wise to be alert for a definite layer of pseudofascia involving the deepest-most subcutaneous tissues. If present, this layer must also be split since it can be quite impressive—e.g., scissors are required to divide it—and an otherwise quite adequate simple fasciotomy may be performed without totally curing the patient's symptoms.

Superficial Anterior and lateral compartment release

For anterior and lateral compartment releases one can either use the technique of Mubarak,[14,17] which involves one vertical incision, or one may approach the compartments through multiple separate horizontal incisions. In general the latter approach is preferable, although the Mubarak technique has been used and has been consistent with good results if one thing is kept in mind: when using the Mubarak approach for anterior and lateral compartment releases, one must carry the anterior compartment incision distal enough to ensure that the patient will not return with a complaint of residual distal anterior compartment tightness or pain where the muscle herniation aggravates itself on the fascial edge of the bottom V of the fasciotomy.

The individual compartment releases are performed in the following manner. The anterior compartment is roughly divided into thirds (or fourths) from top to bottom and two (or three) incisions 3 cm in length are made at the level of the junction of the thirds (or quarters). Whether three or four incisions are needed depends upon leg length. Local anesthesia is quite adequate but giving the patient 5 to 10 mg of diazepam intravenously prior to beginning helps make the patient less anxious. The skin and subcutaneous tissues are infiltrated with lidocaine and divided down to the fascia; the fascia is infiltrated with 0.5% lidocaine above and below the fascia in the direction of the planned fascial incision. The fascia is then divided medially and laterally virtually to the margins of the compartment, taking care not to damage muscle or nerve tissue beneath the fascia. The fascial incisions are brought into continuity by using Metzenbaum scissors first toward the knee, then between the two incisions, and finally distally. When the distal portion is performed, care is taken to stay very anterior, i.e., toward the midline, to avoid injury to cutaneous sensory nerves exiting from the lateral margin of the compartment. The fascia is not divided laterally on the most distal incision. If the patient is short in stature, one can do an adequate release through two incisions or even a single incision.

The lateral compartment is routinely fasciotomized by a single horizontal skin incision, taking care that it is divided medial to lateral, proximal to just below the peroneal nerve resting inferior to the fibular head and distal to approximately 4 to 6 cm above the ankle. The distal portion of the lateral compartment fasciotomy should be located very laterally to avoid sensory nerve injury. With this approach only two of our patients have experienced even transient sensory nerve paresthesias in their distal legs and feet.

The wounds are closed using a cosmetic subcuticular technique, and sterile adhesive strips are applied to the skin. Generally, quite acceptable cosmetic results are achieved.

Posterior superficial release

Releases of one or both of the gastrocnemius muscle compartments (posterior superficial medial and lateral) are nearly always performed using a single incision centered over the involved head of the muscle. In one instance a professional basketball player required two incisions in order to open the compartment adequately top to bottom, but this is decidedly unusual. The distal soleus is released medially or by a posterior superficial incision placed below the gastrocnemius. Care is taken to avoid injury to the short saphenous vein and sural nerve.

Deep posterior release

Proximal deep posterior releases are perhaps the most difficult to describe; typically one develops the ability to both see and palpate one's way to performing an adequate proximal deep compartment release. Until one develops a clear sense of the anatomy and an educated sense of touch, adequate exposure is critical since blind deep fasciotomy may injure arteries, nerves, and especially veins. The potential for injury to the deep veins is the most likely adverse outcome, and, if unrecognized, may produce a deep hematoma which delays rehabilitation and could theoretically produce a deep infection or phlebitis with an adverse long-term result. The single serious intraoperative complication of this series occurred when the distal portion of the posterior tibial artery and vein were divided during a deep compartment release in a patient with very dense fascia. The artery was successfully repaired using a segment of saphenous vein as an interposition graft and the vein was ligated. No long-term functional impairment resulted.

After the patient is anesthesized by either spinal or general anesthesia, a medial incision is made as if one were planning to perform a femoral to distal popliteal artery saphenous vein bypass graft. The vertical incision is usually 8 to 10 cm in length. Care is taken to avoid injury to the saphenous vein and nerve, and the superficial compartment fascia is first divided for a distance of approximately 10 to 15 cm. The soleus fibers are then bluntly split in the direction of their fibers and the deep posterior fascia comes into view. In most of these patients the deep fascia is too dense for blunt division, so sharp division with Metzenbaum scissors is required. One knows that the deep compartment has been entered when the examining finger encounters the direct pulsation of the posterior tibial, peroneal, and distal popliteal arteries. Under direct vision the deep fascial incision is carried proximally to divide the soleal bridge com-

pletely. This allows free and easy access to a widely open popliteal fossa proximally. The fasciotomy is then carried distally to approximately 8 to 10 cm above the level of the ankle.

Distal deep posterior compartment release

Using local anesthesia, the distal deep posterior compartment is routinely released by a vertical incision just posterior to the tibia, medially centering the incision over the midportion of the distal deep posterior compartment musculature, i.e., the posterior tibialis, flexor hallucis longus, and flexor digitorum longus. Avoiding injury to the saphenous nerve and vein, the fascia is divided proximally and distally. It is sensible to release both the soleus as well as the deep compartment if there has been any question clinically about which of the two areas are symptomatic. In some patients with bulky muscles the soleus wraps around the deep compartment and obscures it from view, requiring soleal release to gain access to the deep compartment fascia. Awareness of the normal anatomy of the area and its variations can assure an adequate deep compartment release—this includes division of all fascia covering the deep muscles.

Typically, the total length of this fasciotomy is about 15 to 16 cm, with the skin incision roughly half that length. Unfortunately, traction paresthesias of the saphenous nerve are not uncommon and patients should be warned of this possibility. If sensation is transiently impaired, it usually returns within 3 months. Care to avoid, or to ligate, any divided, perforating vessels is wise since they frequently lie in the course of the fasciotomy.

Rarely, a patient may present with what seems to be a symptomatic muscle hernia through one of these fascial defects, and indeed such may be the case.[19] If the compartment pressures are elevated, such patients have been treated with fasciotomy as described here, but they have not been included as part of this series. Also not included have been two patients who presented in the same manner but in whom Doppler evaluation revealed an incompetent perforator and normal compartment pressures. Ligation and division of the perforator and closure of the fascial defect has been curative.

Before the skin and subcutaneous tissues are closed, a careful examination of the fasciotomized compartments for any venous or arterial bleeding is routinely undertaken. An antibiotic wash is administered to the wound, and then the wounds are closed.

Biopsy methods

Fifty-five muscle and 36 fascial biopsies were taken. Muscle specimens measured at least 2 cm^2 and were promptly prepared for hematoxylin and eosin (H & E) and modified trichrome, adenosine triphosphatase, nicotinamide adenine dinucleotide hydrogen (reduced form)-tetrazolium reductase (NADH-TR), acid phosphatase, periodic acid-Schiff, and oil red O (ORO) staining. Fascia was examined by using H & E and acid phosphatase stains. Muscle biopsy findings considered definitely abnormal included widespread intracellular nuclei, destruction of muscle fibers, substantial loss of muscle type distribution, substantial variation in fiber size, and edema around the fibers. Borderline changes included occasional dark angular fibers and single groupings of Type I or Type II fibers, some edema, clustering into groupings, and/or some internal nuclei. Fascial thickness was measured. The paucity of data on normal criteria for fascial thickness and strength in athletes makes interpretation difficult.

Statistical methods

Chi square analysis was performed comparing each of the following to outcomes: duration of symptoms, severity of symptoms, functional impact, resting compartment pressures, and biopsy data. This was done to determine whether any of these variables were sufficiently sensitive to determine prospectively which patients deserved early operative intervention.

Postoperative care

Patients are given an instruction sheet, crutches, and analgesics as needed (sheet available upon request). Experience has shown that patients recover much more quickly if they stay off their feet as much as possible for the first 48 to 72 hours and keep their legs elevated. This results in their developing much less edema, which could delay their rehabilitation. They are advised to wear elastic wraps when weightbearing for 7 to 14 days, depending upon swelling, and to resume activity as tolerated. If leg swelling persists, they are advised to continue leg elevation as necessary and elastic wrapping when walking until the problem resolves. It is usually the national class athletes who develop the most profound edema, probably due to a larger capillary bed. All are also advised not to be too aggressive in resuming athletic activity since setbacks can then occur which will add to total recovery time.

RESULTS

The 100 patients represented a total of 233 compartments released. Followup is complete to 1 week on 99 patients and complete to 2 months or longer on 97 patients. Those results dependent upon patients' statements of their outcomes were obtained by phone or personal interview by a trained phone interviewer or a physician assistant administering a written questionnaire. The lowest sample size of any of the evaluation items reported here is 95. Including all 99 cases, the median followup is 138 days; the mean is 304.8; the range is 8 to 1425 days.

Operative and postoperative complications occurred in 11 patients. Total complications noted included artery injury requiring repair, 1; hematoma/seroma, 5; superficial wound infection, 4; peripheral cutaneous nerve injury, 3; lymphocele, 1; deep venous thrombosis, 1; and other, 3. (Several patients with complications had more than one.) The patient

with the arterial repair had a good distal pulse at discharge and reported no symptoms in the distal leg or foot at 3 months. Two of the hematoma/seromas were needle-drained and none required operative evacuation. Two of the wound infections (cellulitis) required treatment with antibiotics, but none required hospitalization. The nerve injuries may or may not totally recover; other, more transient, paresthesias were not included in this list. The lymphocele healed spontaneously and occurred in a recurrent posterior deep proximal fasciotomy patient (initial procedure done elsewhere). The deep venous thrombosis patient required hospitalization for heparinization and 2 months of outpatient warfarin therapy, but the veins recanalized without late sequelae and she has been symptom-free for 7 months.

Swelling following the procedure is not uncommon and occurred in 22 patients. This is particularly likely in those patients who are very well-conditioned and have more severe symptoms. Two national class athletes had to be admitted to the hospital for relief of edema in the week following their releases due to their degree of capillary leak. Within 36 hours of elevation, their limbs had returned to normal size, which confirmed the impression that the problem was one of profound edema rather than hematoma. In some instances, mild ankle swelling can persist for 3 to 6 months or longer.

Most patients are walking unassisted in 2 days (median), and are running or jogging at 3 weeks (median). Time to full rehabilitation differs greatly from patient to patient and depends upon severity of the problem, their sports, and personal factors.

Functional and symptomatic results were typically gratifying. Ninety-one patients described functional improvement, and 93 reported relief of pain. Seventy-three percent of involved compartments were completely cured of their symptoms; 9% were significantly improved; 2% were slightly improved; 7% showed no change; and one got worse than prior to operation. Functional results paralleled the above findings, with 73 patients reporting complete functional cures. Eleven percent of compartments were significantly improved; 5% were only slightly improved; none were functionally worse; and 9% were unchanged. Stated in terms of patients instead of compartments, only four patients described experiencing no improvement in symptoms or function.

A distinction between functional impact and symptoms was made in describing the results. This was done as a validity check for reported results. If patients described being better symptomatically but were not improved functionally, one might wonder whether they were really better or had simply cut back on their exercise levels. Conversely, if function improves, one wishes to know if symptoms have improved or disappeared altogether. Further, while nearly all patients preoperatively are functionally limited by their symptoms of pain, a few describe being stopped by foot-drop or loss of muscle function rather than pain itself. Patients with more than one compartment involved may be stopped by pain in one compartment, while another, or others, are less involved and are not as functionally limiting.

Biopsy results were as follows: using criteria outlined above, 44 of 55 muscle specimens from symptomatic compartments were abnormal (definite abnormals, 23; borderline abnormals, 21). Fascial biopsies were beyond 600 μ of thickness in 25 of 36 specimens sampled; some were in excess of 1200 μ. How representative of all cases these specimens were is not certain; however, they were sampled nearly consecutively over a 2 year period.

Recurrences were not common. Of the patients originally operated upon in this series, five developed recurrences involving eight compartments, a recurrance rate of 3.4%. All five patients were reoperated; repeat fasciotomy was performed with total success in one patient (one compartment), partial success in two patients (two compartments), and failure in one (one compartment). The patient with the failure was subsequently cured by fasciectomy. The four other recurrent compartments were initially and successfully managed by fasciectomy. No deep compartment recurrences occurred in this series, but one patient initially operated upon elsewhere for deep posterior symptoms was cured by simple deep fasciotomy. He had noted no change in symptoms following his initial procedure. All recurrences presented by 2 months following the operation and were preceded by a brief symptom-free period.

Overall patient satisfaction with results was gratifying. When patients in the series were asked if they would have the procedure again if necessary or if a new CCS developed, 89 answered "yes," 6 answered "no," and data was missing on 5. Nine patients (15 compartments) actually did develop a second CCS in new or previously asymptomatic compartments. Seven of these patients subsequently underwent fasciotomy of those compartments with relief of symptoms. One of the remaining two patients plans to have fasciotomy performed when her hospital insurance will allow it. The sole remaining patient is currently choosing to live with her new CCS since it is low grade and is not interfering with her current lifestyle.

DISCUSSION

The understanding of compartment syndrome has been rather swift when one considers that the first report of the treatment of its chronic form was only 30 years ago.[11] Still, there is some uncertainty about the manner in which most patients develop the chronic syndrome. Certainly, a variety of etiologies exist and the common feature of all of them seems to be a chronic elevation of pressure within the compartment due to fascia that is unyielding either through thickness or tensile strength alone. Less common precipitating events include chronic intermittent external compression, displacement of compartment space by a healing fracture or a calcified hematoma, deep venous thrombosis, or venous hypertension secondary to a Baker's cyst or substantial soft tissue swelling around the knee. Most instances seem to result from a fascial compartment too small and/or too inelastic to prevent chronic elevation of intracompart-

mental pressure in reponse to muscle hypertrophy and acute muscle volume increases associated with exercise.

It is known that collagen tissue under prolonged stretch responds by aligning its fibers and increasing both its density and strength.[4,8,9,23] Indeed, the capacity of collagen to produce a stronger layer of connective tissue in response to prolonged pressure or stretch most likely accounts for the progressive increase in intensity of symptoms described by 75% of the patients in this series.

A few patients in the series had some degree of scoliosis and the compartment syndrome. Whether there is a cause and effect relationship is not clear since mild forms of scoliosis are not rare. It is reasonable to assume that imbalanced running secondary to scoliosis might disproportionately hypertrophy certain muscles, and hence precipitate the syndrome in a few patients.

The incidence of CCS is very uncertain at present. The population from which this study was drawn included a very uncertain denominator. An average of three per year came from an annual university population of over 40,000 students, but not all students were examined for the condition. The frequency with which referrals increased over the years as local understanding of the condition spread suggests that, while it is uncommon, it is by no means rare. Yet, rare manifestations of the syndrome do exist.

The natural history of CCS is not thoroughly documented. Many patients in this series had had symptoms continuously for 10 or more years, noting relief only when cutting back on their activity. Some would note the problem only with heavy exercise regimens; otherwise they would be symptom free. Others describe a more progressive course with stable symptoms for months or years, then gradual worsening. Still others describe a somewhat sudden onset with conditioning and a dogged persistence once symptoms developed. A few patients who declined operation have stayed in contact over the years and their symptoms have typically persisted or worsened to a level where operative relief was sought. The clinical chronicity of the syndrome suggests that once the investing fascia has hypertrophied sufficiently to produce a chronic problem, it tends to persist.

There is a difference in philosophy among experts regarding the necessity for and urgency of fasciotomy for chronic compartment syndrome. Experimental work to date has focused mostly upon the effects of acute ischemia and acute compartment syndromes.[1,5–7,10,16,24,27] Much less is known regarding the particulars of chronic intermittent ischemia and tissue effects on muscle and fascia. This state of affairs at one level is certainly not resolved by this report. There were no statistically significant correlations between duration of symptoms, severity of symptoms, functional impact, resting compartment pressures, biopsy data, and outcomes. Some correlations to the 0.10 level were noted, but none to the 0.05 level. Why is this? Possibilities are numerous and include, among others, an inadequate number of observations, use of single incidental rather than repeated compartment pressure measurements, widely varying pain thresholds among patients, wide variation among patients in sensitivity of muscular, and nervous tissues to ischemic injury. It is possible that comparing outcomes with continuous pressure data over 48 to 72 hours might be more revealing and perhaps not. It seems reasonable that some patients may show higher pressures over time in response to exercise than may others. Certainly the availability of substitute muscle groups in the posterior portion of the leg may mask significant tissue injury to one isolated compartment.

We do believe that there are a few compelling reasons to recommend fasciotomy for those patients with an excellent history of CCS and elevated compartment pressures. This philosophy is partially supported by the muscle biopsy data since 80% showed abnormality in a presumptively healthy individual. A more aggressive surgical stance is especially warranted if the anterior, or lateral or distal deep posterior compartments are involved. Irreversible injury to muscles or nerves in these compartments cannot be substituted for by other muscle groups.

An increase in fascial thickness was also apparent in 25 of 36 specimens sampled. The impressive thickness of fascia in some patients (greater than 1200 μ) strongly suggests that once established, a chronic compartment syndrome may be unlikely to reverse itself easily. Further, the ability to predict which patients may have significant muscle pathology is limited, as is the predictive capacity to determine prospectively who may not recover well following operation. This inability to determine prospectively who needs the procedure probably relates to the tremendous differences in pain thresholds and/or pain tolerances among individuals. Very high pain thresholds and/or tolerances can mask significant muscle injury and ischemia.

The anterior compartment group is best examined in detail when comparing biopsy findings and their relation to prognosis. While an abnormal biopsy did not allow prediction of a poor outcome (19 such patients experienced full recovery), none of the 4 patients with poor outcomes had normal biopsies. These observations suggest that neither the muscle tissues biopsied nor the patients who were biopsied were unrepresentative, but that some muscles and nerves recover better than do others, or that pathologic criteria for precisely assessing the degrees of reversible tissue injury are lacking. It is known that muscle does have the ability to regenerate itself to a considerable degree unless subjected to complete ischemia for several hours.[26] In chronic settings it is quite difficult to gauge the duration and severity of ischemia. A reexamination of the biopsies from patients experiencing less than complete recovery is currently underway in an effort to identify any findings predictive of later poor outcomes in comparison to their more fortunate colleagues.

Four patients with poorer outcomes have been unable to resume prolonged running even after 2 years following fasciotomy, yet none of the four have evidence of recurrence of CCS deserving fasciectomy; i.e., their symptoms do not include the typical tightness of the compartment but rather stiffening and weakness of the muscles. Repeat biopsy in these patients would be interesting, but has not been done. Continuing clinical improvement up to 2 years following

fasciotomy was noted in these four patients, but no further progress has occurred.

A comment should be made regarding patients with distal deep posterior compartment involvement. The patients included in this report had elevated resting compartment pressures and were similar in all particulars to other CCS patients with respect to symptoms. A few had concomitant problems referred to as periostitis by Mubarak et al.[12] We definitely believe that both conditions exist as distinct entities. One operated patient who was cured of symptoms from proximal deep compartment involvement increased her exercise levels and developed distal involvement which was cured with a distal release. The level of functional separation between the proximal and distal components of the deep posterior compartment seems to be where the posterior tibialis moves from its location directly behind the tibia to a medial location just below the midportion of the leg.

Interpreting poor outcomes is important since recurrences can be treated successfully with a second procedure. Historical data can be very helpful since some poorer results will be related to prior muscle or nerve injuries rather than to CCS recurrence. Those with simple muscle injury describe soreness without the characteristic tightness of CCS. Muscle or nerve injury patients do typically express definite relief of tightness, but not soreness or greatly improved function. It is very common for patients to express relief from tightness soon after fasciotomy. Indeed, some describe relief immediately upon release of the fascia while they are still on the operating table.

No reliable indicators of which patients are likely to develop recurrences could be identified. For example, preoperative compartment pressures in compartments which later developed recurrences were not appreciably higher than average. The capability to measure consistently accurate pressures postoperatively has been thwarted by apparent scar tissue scattered between the incised fascia, new overlying pseudofascia, and the underlying muscle. This is particularly true during the first few months following operation. When normal pressures are obtained, they are likely to be accurate. However, elevated pressures present difficulties since one cannot be certain that one is not simply measuring a subcompartmental space rather than the pressure within the entire compartment.

In this series all recurrences appeared to be due to exuberant excessive healing of scar tissue over the fasciotomized muscle. Evidence for this is found in three sets of observations. First, all patients who developed a recurrence first described a period of symptomatic relief following their initial release. This supports the impression that the initial operation adequately decompressed the compartment. Indeed, all but one reported relief of symptoms until typical symptoms of CCS returned at approximately 2 months and then gradually worsened. The remaining recurrence developed 1 year following the initial operation. Second, at reoperation fascial margins had not rejoined, but rather a thick abundant scar tissue was found to have created a constricting pseudofascia which in some instances was much more impressive in density and strength than the original fascia. Third, the observation that fasciectomy (removal of all remaining fascia and pseudofascia) typically results in a cure, suggests that only with an extensive excision can one be assured that one has gotten ahead of the exuberant tissue healing response and thereby prevented the potential for future CCS. Because some strength is sacrificed at least temporarily, if not permanently, when the fascial barrier is not present for the muscle to work against, it is a sound rule that fasciotomy should only be performed in compartments in which one is convinced CCS is present. For the same reason fasciectomy should be reserved for cases of CCS recurrence following fasciotomy. This seems quite reasonable since recurrence following simple fasciotomy is acceptably low, i.e., below 3.5%. If one finds a second layer of pseudofascia overlying the fascia at the time of original fasciotomy, this should be divided with at least two longitudinal incisions if one is to rest assured that an adequate compartment release has been obtained.

The experience with this small group of recurrences has led us to favor fasciectomy for those patients who initially experience a good result and then develop new symptoms. Like fasciotomy, fasciectomy can be performed adequately under local anesthesia on an outpatient basis.

A number of patients have vigorously pursued a wide variety of nonsurgical approaches prior to deciding to have a surgical release. The relative rareness of CCS, the poor results of nonoperative modalities described by these patients, and their satisfaction with fasciotomy when performed on an outpatient basis make a good but not fully scientific argument for operation. Until a randomized trial is performed it seems reasonable to recommend fasciotomy for those patients with significant symptoms or disability and elevated compartment pressures.

It is worth keeping in mind that the results reported here can be improved upon if one operates on patients before they develop severe symptoms or serious functional impairment. Even including all comers as they arrived for care, when one compares the poor results which follow denial of operation or long delays prior to operation and the severity and chronicity of symptoms in many of these patients to the ease, safety, and generally excellent results when fasciotomy is performed, fasciotomy emerges as an excellent treatment for managing CCS.

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