Diagnosis and management of chronic compartment syndromes: a review of the literature

Michael Barnes

Compartment syndromes may be acute or chronic. Acute syndromes may complicate fracture, soft tissue trauma, burns, and drug overdose. Very rarely is excessive exercise the underlying cause. Sportsmen and women (particularly footballers) are perhaps more prone to this condition than the general population because of the high incidence of tibial fractures. Acute compartment syndromes are a potentially limb threatening problem that should be dealt with urgently and hence are more in the remit of orthopaedic and accident and emergency departments than of sports medicine.

Chronic compartment syndromes are the definitive sports injury, being almost entirely due to exercise and overuse. Diagnosis and management are still a matter of debate and uncertainty.

Historical aspects
The first case of a genuine chronic compartment syndrome was documented by Mavor in 1956 as a case report of a professional footballer with bilateral lower leg pain caused by exercise. All earlier reports of exercise related compartment syndromes had been acute syndromes induced by exercise, mostly in military recruits. It is interesting to note that the clinical description and treatment in Mavor's paper are very similar to current opinions and practice.

In the same volume of the journal, an editorial by Griffiths cast doubt on Mavor's case and questioned the existence of a chronic compartment syndrome. However, over the past 40 years there have been well over 1000 documented cases (mostly confirmed by intracompartmental pressure measurement and the successful outcome of fasciotomy), so it would seem that Griffiths was wrong, and chronic compartment syndrome is now a well recognised specific condition, with its own history, symptoms, and treatment.

The correlation of the history and symptoms with raised intracompartmental pressure was first carried out by French and Price in 1962 using the needle method of Wells et al.

When Reneman published his series of 61 patients in 1975, there had been only nine previously reported cases. All 70 cases were anterior/lateral. A chronic compartment syndrome of the deep posterior compartment had been suggested by Puranen in 1974, but not confirmed by pressure measurements until 1981. Even today there is still uncertainty about the causes of exercise related pain in and around the deep posterior compartment.

Definition
In the broadest and simplest terms a chronic compartment syndrome is defined as a condition of pain on exercise that is relieved by rest. Over months the symptoms become worse with progressively earlier onset. However, this definition could cover several other conditions. Hence the need for measurements of intracompartmental pressure.

Anatomy
Some 95% of chronic compartment syndromes occur in the lower leg. This is due to its particular anatomy and that intensive exercise of the lower leg is part of virtually all sports. The remaining 5% are distributed between the forearm, thigh, hands, and, very rarely, feet. What follows, therefore, will be limited to the lower leg.

It is a generally accepted view that there are four compartments in the lower leg: an anterior, deep posterior, lateral, and superficial posterior. Chronic compartment syndromes have been diagnosed in all four, but it is predominantly the anterior and deep posterior compartments that are most often involved. This is not a universally held belief. Davey et al. suggested that the tibialis posterior muscle constitutes a separate compartment making a total of five compartments altogether. Detmer et al. has taken this even further, stating that each muscle should be regarded individually. They also describe seven functional compartments: in addition to the anterior and lateral compartments, the deep posterior should be divided into proximal and distal, and the superficial posterior should be subdivided into distal, lateral, and medial. No work has been done to confirm that these are discrete pressure units. These additional subdivisions may be relevant for correct surgical decompression.

Clinical aspects
Many chronic compartment syndromes can be diagnosed by history alone. The patient complains of pain on exercise that is relieved by rest and has progressively worsened over a period of months. The pain may be localised to a specific compartment or described as a general ache. There may be distal neurological signs of paraesthesia in the foot. There are, however, several other conditions which may mimic these symptoms—for example, popliteal entrapment syndrome, medial tibial syndrome, or stress fracture. Most of these may be eliminated by other signs or routine investigations. Medial tibial syndrome always has associated inner border tibial tenderness; stress fractures can usually be confirmed by plain radiographs.
and popliteal entrapment syndrome will affect foot pulses (chronic compartment syndrome never affects the major vessels). However, none of these conditions are mutually exclusive, so the confirmation of one does not necessarily rule out the existence of the others.

On examination there is little or nothing to see. Some authors suggest that swelling over the anterior compartment can be felt in some patients, particularly after exercise. This is a subjective finding and gives no indication as to what might be happening in the deep posterior compartment. Swelling is of course, no indication of raised compartmental pressure. It is common to find grossly swollen and tense limbs with normal intracompartmental pressures (in patients with suspected acute compartment syndrome).

Table 1  Reported incidence of muscle hernias

<table>
<thead>
<tr>
<th>Authors</th>
<th>Year</th>
<th>Patients</th>
<th>Incidence (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mavov et al.</td>
<td>1956</td>
<td>1</td>
<td>100</td>
</tr>
<tr>
<td>French and Price</td>
<td>1962</td>
<td>3</td>
<td>100</td>
</tr>
<tr>
<td>Reneman et al.</td>
<td>1975</td>
<td>60</td>
<td>60</td>
</tr>
<tr>
<td>Sudmann et al.</td>
<td>1979</td>
<td>51</td>
<td>Not mentioned</td>
</tr>
<tr>
<td>Puranen and Alasviko et al.</td>
<td>1981</td>
<td>10</td>
<td>Not mentioned</td>
</tr>
<tr>
<td>McDermott et al.</td>
<td>1982</td>
<td>9</td>
<td>45</td>
</tr>
<tr>
<td>Qvarford et al.</td>
<td>1983</td>
<td>108</td>
<td>40</td>
</tr>
<tr>
<td>Martens et al.</td>
<td>1984</td>
<td>29</td>
<td>30</td>
</tr>
<tr>
<td>Wallensten et al. and Eriksson</td>
<td>1984</td>
<td>12</td>
<td>Not mentioned</td>
</tr>
<tr>
<td>Detmer et al.</td>
<td>1985</td>
<td>50</td>
<td>Occasional</td>
</tr>
<tr>
<td>Syf and Korner</td>
<td>1986</td>
<td>80</td>
<td>See text</td>
</tr>
<tr>
<td>Allen and Barnes</td>
<td>1986</td>
<td>110</td>
<td>None found</td>
</tr>
<tr>
<td>Froncek et al.</td>
<td>1987</td>
<td>18</td>
<td>39 (&lt;5 in normal subjects)</td>
</tr>
<tr>
<td>Turnispeed et al.</td>
<td>1989</td>
<td>209</td>
<td>Not mentioned</td>
</tr>
<tr>
<td>Pedowitz et al.</td>
<td>1990</td>
<td>131</td>
<td>45.9 (12.9 in normal subjects)</td>
</tr>
<tr>
<td>Ambramowitz and Schepis</td>
<td>1994</td>
<td>28</td>
<td>25</td>
</tr>
</tbody>
</table>

Table 2  Evolution of chronic compartment syndrome pressure measurement techniques

<table>
<thead>
<tr>
<th>Authors</th>
<th>Year</th>
<th>Technique</th>
</tr>
</thead>
<tbody>
<tr>
<td>French and Price</td>
<td>1962</td>
<td>Wells et al. needle</td>
</tr>
<tr>
<td>Reneman et al.</td>
<td>1975</td>
<td>Needle</td>
</tr>
<tr>
<td>Mubarak et al.</td>
<td>1976</td>
<td>Wick</td>
</tr>
<tr>
<td>D’Ambrosia et al.</td>
<td>1977</td>
<td>Needle with transducer</td>
</tr>
<tr>
<td>Sudmann et al.</td>
<td>1979</td>
<td>Needle and wick</td>
</tr>
<tr>
<td>Rorabeck et al.</td>
<td>1980</td>
<td>Silt</td>
</tr>
<tr>
<td>Puranen and Alasviko et al.</td>
<td>1981</td>
<td>Wick</td>
</tr>
<tr>
<td>Mubarak et al.</td>
<td>1981</td>
<td>Silt</td>
</tr>
<tr>
<td>McDermott et al.</td>
<td>1982</td>
<td>STIC</td>
</tr>
<tr>
<td>Raether and Lustig</td>
<td>1982</td>
<td>Whitesides et al. needle</td>
</tr>
<tr>
<td>Christenson et al.</td>
<td>1983</td>
<td>Epidural catheters</td>
</tr>
<tr>
<td>Qvarford et al.</td>
<td>1983</td>
<td>Wick</td>
</tr>
<tr>
<td>Rydholm et al.</td>
<td>1983</td>
<td>Wick</td>
</tr>
<tr>
<td>Wallensten et al.</td>
<td>1983</td>
<td>Wick</td>
</tr>
<tr>
<td>Martens et al.</td>
<td>1984</td>
<td>Wick</td>
</tr>
<tr>
<td>Detmer et al.</td>
<td>1985</td>
<td>Needle and wick</td>
</tr>
<tr>
<td>Syf and Korner</td>
<td>1986</td>
<td>Microcapillary infusion</td>
</tr>
<tr>
<td>Katz et al.</td>
<td>1985</td>
<td>Silt</td>
</tr>
<tr>
<td>Allen and Barnes</td>
<td>1986</td>
<td>Silt</td>
</tr>
<tr>
<td>Bell et al.</td>
<td>1986</td>
<td>Wick and silt</td>
</tr>
<tr>
<td>Froncek et al.</td>
<td>1987</td>
<td>Wick and silt</td>
</tr>
<tr>
<td>Turnispeed et al.</td>
<td>1989</td>
<td>Stryker</td>
</tr>
<tr>
<td>Pedowitz et al.</td>
<td>1990</td>
<td>Silt</td>
</tr>
</tbody>
</table>

and compartment syndrome found by Pedowitz et al. were reported as significant.

The only conclusion that can be drawn from such a diversity of findings is that preselection of patients may influence the results when comparing series.

Pressure measurement

Measurement of intracompartmental pressure is required to confirm the diagnosis and establish exactly which compartments are involved. The techniques and equipment used have evolved over time (table 2). The needle and manometer techniques of Wells et al. and Whitesides et al. have largely given way to wick or slit catheters and pressure transducers.

These more recent methods are not necessarily more accurate, but are much easier to use and more versatile. With flexible catheters and transducers the patient can exercise with the catheters in place, thus making it possible to measure pressure during exercise and immediately afterwards. This is obviously better than inserting a needle after exercise because of the time delay involved and the possibility that the localised trauma may give a falsely high pressure. Also, by using transducers and amplifiers an electrical signal is produced enabling a record of the fluctuations in pressure to be made.

Today most people use the slit catheter as it is readily available (or simple to make) and reliable with an adequate frequency response for measuring pressure during exercise. The older method of needle and manometer is rarely used, but does have the advantage of needing no specialised equipment so it can be used almost anywhere. The STIC (solid state transducer intracompartmental catheter) of McDermott et al. and the microcapillary infusion technique of Syf and Korner are both more complex methods designed to improve frequency response, but are both rather complicated for routine clinical use. The Stryker box is a simple hand held device containing a transducer, amplifier, and display, that con-
Table 3 Diagnostic criteria of intracompartmental pressure measurement

<table>
<thead>
<tr>
<th>Authors</th>
<th>Year</th>
<th>Diagnostically relevant pressure values</th>
</tr>
</thead>
<tbody>
<tr>
<td>French and Price</td>
<td>1962</td>
<td>Postexercise fall time &lt;30 min in normal subjects, &gt;100 min in patients</td>
</tr>
<tr>
<td>Reneman</td>
<td>1975</td>
<td>At six minutes postexercise pressure &gt;15 cm H2O (11 mm Hg) above resting pressure</td>
</tr>
<tr>
<td>Puranen et al</td>
<td>1982</td>
<td>Mean pressure of 55 mm Hg during running. Resting of no value, but did observe slow postexercise fall</td>
</tr>
<tr>
<td>McDermott et al</td>
<td>1982</td>
<td>Mean pressure of 85 mm Hg during running</td>
</tr>
<tr>
<td>Mubarak and Hargens</td>
<td>1982</td>
<td>Resting pressure &gt;15 mm Hg. Exercise pressure &gt;75 mm Hg. Pressure remains &gt;30 mm Hg for &gt;5 min after exercise</td>
</tr>
<tr>
<td>Puranen et al</td>
<td>1983</td>
<td>Pressures raised before, during, and after exercise. Postexercise decline 40 min. Tn 6 min</td>
</tr>
<tr>
<td>Wallensten et al</td>
<td>1983</td>
<td>No difference at rest, still raised 10 min postexercise (anterior), returned to normal &lt;10 min (deep posterior)</td>
</tr>
<tr>
<td>Detmer et al</td>
<td>1985</td>
<td>At rest, normal pressure &lt;15 mm Hg</td>
</tr>
<tr>
<td>Styf and Korner</td>
<td>1986</td>
<td>Muscle relaxation pressure</td>
</tr>
<tr>
<td>Styf and Korner</td>
<td>1986</td>
<td>Postexercise pressure &gt;35 mm Hg remained raised for &gt;6 min (Also muscle relaxation pressure was raised during exercise, &gt;20 mins to return to normal)</td>
</tr>
<tr>
<td>Allen and Barnes</td>
<td>1986</td>
<td>Exercise pressure &gt;50 mm Hg anterior, &gt;40 mm Hg deep posterior No difference in resting pressures</td>
</tr>
<tr>
<td>Fronck et al</td>
<td>1987</td>
<td>Resting pressure ≥10 mm Hg and/or ≥25 mm Hg 5 min after exercise</td>
</tr>
<tr>
<td>Rorsbeck et al</td>
<td>1988</td>
<td>Pre-exercise pressure &gt;10 mm Hg and postexercise &gt;15 mm Hg for &gt;15 min</td>
</tr>
<tr>
<td>Styf</td>
<td>1988</td>
<td>Relaxation pressure &gt;35 mm Hg, resting pressure &gt;30 mm Hg, postexercise return to normal &gt;6 min</td>
</tr>
<tr>
<td>Turnispeed et al</td>
<td>1989</td>
<td>&gt;20 mm Hg at rest (postexercise increase and slow decline &gt;10 min)</td>
</tr>
<tr>
<td>Pedowitz et al</td>
<td>1990</td>
<td>Pre-exercise pressure ≥15 mm Hg, or postexercise pressure ≥30 mm Hg at 1 min or ≥20 mm Hg at 5 min</td>
</tr>
</tbody>
</table>

Other authors have used different concepts. Puranen and Alavaikko adopted a mean value of 50 mm Hg during running and this figure was also used by Allen and Barnes. McDermott et al used a similar exercise protocol but adopted a higher threshold pressure of 85 mm Hg. In addition they state that postexercise recordings are so variable that they should not be used for diagnosis. We agree with this statement, having found that the pressure drops immediately on the cessation of exercise. We think that a slow decline in postexercise pressure is more likely to be a blocked catheter rather than a finding of pathological relevance. The justification for using pressures recorded during exercise is that this is when the symptoms occur—the pain goes when the exercise stops.

During exercise, as the muscles contract and relax, a pulsatile pressure waveform is generated, with the maximum pressure corresponding to the contraction and the minimum pressure occurring during relaxation. Styf et al considered the muscle relaxation pressure to be a more appropriate measure of diagnosis than mean exercise pressure. This is based on the idea that if the exercise pressure drops below 30 mm Hg during the relaxation phase of exercise, the muscle can still be adequately perfused. Styf et al also found a good correlation between muscle relaxation pressure and postexercise fall, and dismissed mean exercise pressures as being unreliable, having found no significant differences between legs with and without compartment syndrome. This is explained by the effect of muscle contraction force on pressure.

The clinical relevance of pressures measured during exercise has been criticised on several grounds: the value being dependent on depth of catheter insertion and the strength of muscle contraction. Hargens et al found strong correlations between the pressure and the depth into the muscle and percentage voluntary contraction (MVC). Higher pressures were recorded at greater depth and higher %MVC. This finding has been confirmed by Abramowicz and Schepis.

However, this work was done in the vastis medialis of the thigh which is anatomically very different from the muscles in the lower leg compartments. In addition it is possible to avoid these criticisms by inserting the catheters to a fixed depth and adopting a quantified exercise protocol.

Some have used the pre-exercise resting pressure for diagnosis (table 3), or at least taken it into account. However, as pointed out by NKelle et al there is a range of nearly 20 mm Hg in normal subjects, making any use of this figure questionable, and similarly Allen and Barnes found a wide variation in the resting pressures of patients with chronic compartment syndrome.

Hence, there is no consensus on definitive diagnostic values. This probably reflects the lack of understanding about the underlying pathophysiology.

However, intracompartmental pressure measurements remain the most direct and best
objective method of diagnosis. Many other methods have been used in the investigation of chronic compartment syndrome, but these have largely been in an attempt to find out more about the underlying causes, rather than as practical diagnostic techniques.

Other methods of investigation

BONE SCANS
Radioisotope imaging is a useful technique in the diagnosis of lower leg pain; in particular, bone scans are the best method to confirm stress fractures. Several authors have attempted to use bone scanning to diagnose compartment syndrome, but the results have been inconclusive. This is not an appropriate technique for the diagnosis of chronic compartment syndrome, but is necessary for the elimination of other diseases.

METHOXY ISOBUTYL ISONITRILE
Methoxy isobutyl isonitrile has been used to image ischaemic heart muscle for some time. Recently, this technique has been applied to skeletal muscle in the hope that it can distinguish between normal and pathological compartments. The disadvantages of this technique are that it subjects the patient to radiation, the radiopharmaceutical is very expensive, and it requires 3D imaging techniques. Should it prove to be reliable it has the advantage of being more widely available than pressure measurements and is less invasive.

ELECTROPHYSIOLOGY
There are two types of investigation that may be applied to chronic compartment syndrome. Electromyography (EMG) can be used to give an indication of the strength of muscle contractions, and hence may be useful in shedding light on the causes of compartment syndrome. Nerve conduction velocity (NCV) may show neurological changes due to raised intracompartmental pressure either directly exerting pressure on the nerves or affecting them by disrupting the blood supply. Both may be useful research tools, but it is rare for intra-compartmental pressures to become sufficiently elevated that the nerves are affected, so the diagnostic use is limited.

MAGNETIC RESONANCE IMAGING
Magnetic resonance imaging (MRI) has been used to investigate chronic compartment syndromes. It has the obvious advantages of being non-invasive, producing an image of all the compartments at the same time, and perhaps providing additional information as to the causes. At present it is still a very expensive diagnostic technique, and rather an indirect method of diagnosis.

ULTRASOUND IMAGING
Gershuni et al. have used ultrasound to measure changes in the size of the anterior compartment after exercise. This has the advantages of being totally non-invasive and widely available, but is not really a valid diagnostic technique. It can only be used on the anterior compartment, and there is no evidence to suggest that an increase in the compartment volume is related to an increase in the compartment pressure. It could be argued that the compartment pressure increases because there is no increase in the compartment volume.

MUSCLE BLOOD FLOW
This technique has been used for many years. Struyf et al. correlated muscle relaxation pressure with muscle blood flow during exercise. Qvarfordt et al. found that muscle blood flow decreased during exercise in patients with raised pressures. These findings may shed light on the underlying pathology, but are again more a research than clinical tool.

Treatment

DO NOTHING
The real incidence of chronic compartment syndromes in the population at large is unknown. This is because it is a self-limiting condition, the pain occurs during exercise and goes with rest. Many potential patients will simply give up their activity rather than seek medical treatment. This attitude may unfortunately be adopted by some members of the medical profession who recommend that if it hurts don’t do it”. Only a small percentage of potential patients may ever progress to reach a definitive diagnosis.

Many patients can trace the earliest symptoms back to an increase in or a change of activity. Reducing activity back to previous levels often prevents the pain, and some patients consider this to be the best option.

There is one potential danger with this method of treatment. There is a small amount of evidence to suggest that a chronic compartment syndrome may, under certain circumstances (excessive overuse), become acute and therefore potentially limb threatening. This can only be inferred from the very few cases of exercise-induced acute compartment syndromes which sometimes list chronic symptoms in the history. There is no direct evidence.

CONSERVATIVE MEASURES

Preventive
It is widely believed, although there can be no real objective scientific evidence, that inappropriate training may be a major contributing factor to the development of a chronic compartment syndrome. Such factors as incorrect or worn out footwear, running on hard surfaces, and changing or increasing activity have all been suggested as possible causes. There is also the suggestion that chronic compartment syndrome may be caused by biomechanical abnormalities and in particular excessive pronation. If this is the case then correcting the gait by the use of orthotics should prevent the symptoms. As yet this theory and the effectiveness of orthotics have neither been proved nor disproved.

Curative
As well as restricting activity to within comfortable limits, total rest has also been suggested as a treatment. It is our experience that
Compartment syndromes

rest has no effect at all on genuine cases. Even when patients do nothing more strenuous than everyday walking for several months, the symptoms return within days of recommencing to run. This contradicts the theory that muscle hypertrophy is a contributing factor, as muscle will waste very quickly with this level of inactivity.

Many other conservative treatments have been tried. Anti-inflammatory drugs, painkillers, ice, heat, ultrasound, physiotherapy, and steroid injections have all been used at some stage. Most of these treatments are effective at alleviating the symptoms to a degree, but they do not cure the underlying problem. In the series of 80 patients described by Styf and Kornner® all had tried a range of conservative measures, including diuretics, without success. Martens et al® treated nine patients conservatively with prolonged rest, physiotherapy, anti-inflammatory drugs, and stretching exercise of the flexor muscles, but this could not be considered successful as all the patients had to reduce their sports activity to some extent. Fronke et al® initially treated 18 patients conservatively with rest, stretching, and modification of activity. However, 13 of 18 found this unsatisfactory and went on to surgery. The remaining five did not improve, but continued to restrict their activity.

Hence all have found conservative measures to be unsuccessful.

Surgery

It is generally accepted that surgery is the only really effective treatment for a chronic compartment syndrome. Various different fasciotomy techniques have been described, but all have the same effect—to divide the restricting envelope of the fascia and thus effectively increase the size of the compartment.

The underlying cause of chronic compartment syndrome is still unknown. One of the possible causes may be due to changes in the mechanical properties of the fascia—for example, reduced compliance due to increased thickness. If, as seems most likely (as long term rest and all conservative measures are ineffective), these changes are irreversible, then surgery must be the only effective treatment.

Outcomes

The outcome of surgery is generally very good with some success rates reaching 100%. A successful result can vary from an increased exercise tolerance to complete absence of symptoms. Several authors have also measured postoperative pressures for confirmation, although these can be difficult to interpret owing to the presence of scar tissue. There are some poorer results, particularly with the deep posterior compartment. Abramowitz and Schepsis® found that all 16 patients who underwent anterior compartment fasciotomy had good results. However, only 13 out of 20 had satisfactory results in the deep posterior compartment. Similarly Rorabeck et al® obtained complete success in the anterior compartment but in only 10 out of 12 with the deep posterior compartment. Both these authors suggest that the failures were due to incomplete fasciotomy, a theory supported by Puranen and Alavaikko® who went on to try his failures with more extensive surgery, obtaining successful outcomes.

In one of the largest series Turnspeed et al® decompressed 209 patients, 100 with a subcutaneous fasciotomy and 109 with the unusual method of open fasciotomy. The success rate was higher (98%) with the open procedure than with the closed (89%). However, no mention was made of recurrence rates in specific compartments.

The comparatively low success rate of surgery in the deep posterior compartment may result from several reasons. The possibility of incomplete fasciotomy has already been mentioned, and some authors are cautious about excessive dissection in an athlete, possibly leading to muscle weakness. A further reason may be incomplete decompression of the tibialis posterior, if as suggested by Davey et al® it is a separate compartment. There is also the possibility of multiple diseases with similar presentations. Detmer® has subdivided patients with exercise pain in the deep posterior compartment into three distinct groups, which are not all treatable by surgery and may well coexist.

Postoperative Management

The successful outcome of surgery may also depend on the immediate postoperative management. The earliest possible mobilisation is required to maximise the effect of the fasciotomy. If the patient remains inactive in bed for several days after surgery, then the fascia may well heal back to its original size and make the surgery ineffective.

Rorabeck et al® encouraged patients to walk as much as possible on the first postoperative day; Bell® kept his patients in bed for five days, but encouraged them to perform active foot dorsiflexion one day after surgery; Detmer et al® kept the legs raised for 48 to 72 hours to reduce oedema and hence speed recovery, whereas Wal lensten® allowed his patients to walk immediately after surgery.

Styf and Kornner® kept the patient’s legs raised for 12 hours and then encouraged walking.

Complications

Some authors have admitted to surgical complications. Detmer et al® in a series of 100 patients had a total of 11. These included one arterial injury repair, five haematomas, four wound infections, three nerve injuries, and one deep venous thrombosis. Fronke et al® reported only one wound infection in a series of 18 patients. The published complication rates are not sufficiently high to consider that surgery may be too risky; but there are other possible contraindications which should be considered.
POSSIBLE DETERIMENTAL EFFECTS OF SURGERY

There is some speculation concerning the possible detrimental effects of surgery. As has already been mentioned there are concerns about excessive surgical dissection causing muscle weakness. Garfin et al. found that muscle force decreased with fasciectomy, which may be a problem for athletes. This work was carried out on dogs so it only shows an immediate effect; there was no long term follow up. No measurements have been done to see if this is a genuine long term problem. A more recent study has shown that preoperatively patients with compartment syndrome have weaker muscles than normal subjects. Our current research shows that patients with chronic compartment syndrome have greater power but less endurance than normal subjects.

It is widely thought that fasciectomy is successful because it increases the size of the compartment, thereby increasing the volume and reducing the pressure generated by exercise. However, if the cause of increased pressure is stronger muscle contractions, then perhaps the effect of fasciectomy is to reduce the power of the contractions and thus to reduce the pressure. This theory correlates with the work of Hargens et al. who found that intracompartamental pressure was related to muscle contraction force.

REPEAT SURGERY

As mentioned earlier there have been some failures of surgery. Many of these have then been successfully treated by a second more extensive operation. It is important to distinguish between patients who have never had relief of symptoms and those who have had a recurrence months or even years later. Bell had five patients who still had symptoms and elevated pressures after fasciectomy. These were successfully treated by fasciectomy. In the series of Detmer et al. of 100 patients undergoing surgery, five had had a recurrence of symptoms within two months. All five were then cured by a second operation. We have found a small but important group of patients who were free of symptoms for several years before the symptoms returned. These patients have also responded well to a second operation, but it is difficult to explain why the symptoms returned. This may cast doubt on the long term effects of fasciectomy. No comprehensive long term follow up studies have yet been done.

Conclusion

Chronic compartment syndrome of the lower leg is a relatively common life-threatening injury. It is usually easy to confirm the diagnosis from history alone, but in more difficult cases, intracompartamental pressure measurement is required. The treatment of choice is fasciectomy, which has a very high success rate. Despite the abundance of literature on the subject over the past 40 years, there are still several areas of disagreement, principally that of an exact diagnostic pressure value. This may become clearer as more work is done to establish the underlying cause.

60 See PAK, Miles KA, Emmerson S, Jenner JR. Leg muscle scintigraphy with 99Tc MIIBI and single photon emission tomography (SPECT) in the assessment of suspected compartment syndromes of the calf. British Institute of Radiology Congress 1994;38.