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Functional Popliteal Entrapment Syndrome in the Sportsperson

R. Lane [a,b,c,d,e,*], T. Nguyen [c], M. Cuzzilla [a,d], D. Oomens [d], W. Mohabbat [a,c,e], S. Hazelton [a]

[a] Dalcross Adventist Hospital, Sydney, New South Wales, Australia
[b] North Shore Private Hospital, Sydney, New South Wales, Australia
[c] Royal North Shore Hospital, Sydney, New South Wales, Australia
[d] Vascular Specialists Investigations and Management, Sydney, New South Wales, Australia
[e] Department of Vascular Research, Macquarie University, Sydney, New South Wales, Australia

WHAT THIS PAPER ADDS

- The development of a simple clinical provocative test i.e. hopping with the observation of an ischaemic weak foot. The physiological basis for hopping at the same rate of the heart is suggested.
- Related to the rapid recovery times in these athletes, automation of the systolic pressure following exercise is suggested.
- Ultrasonic differentiation of concomitant venous obstruction is defined in both syndromes both being related to muscle hypertrophy.
- Analysis of recurrences and their treatment underpins the cause and effect relationship between symptoms and functional popliteal obstruction.
- A comprehensive approach to the diagnosis and treatment to FPE.

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ABSTRACT

Objective: To define the clinical syndrome of functional popliteal entrapment comparing pre and post surgical clinical outcomes with pre and post-operative provocative ultrasonic investigations. Further, to suggest a management pathway to differentiate chronic exertional compartment syndromes and concomitant venous popliteal compression.

Methods: In 32 claudicant sportspersons, 55 limbs were characterised pre-surgery clinically, with provocative testing including hopping, and following a series of non-invasive tests. The clinical findings, ankle brachial indices (ABI) and duplex outcomes were compared pre-operatively, at 3 months post-operatively ($n = 52$) and in the long term i.e. 16 months ($n = 17$).

Results: At 3 months, all 55 limbs had clinical follow up. 52 of the 55 limbs had follow up with ultrasound with provocative manoeuvres. The ABIs normalised in 46 (88%). There were 40 of 52 (76%) that became asymptomatic post surgery with a normal scan. There were 4 of 52 (8%) who were clinically asymptomatic but with residual obstruction on duplex and who were able to resume their usual lifestyle. There were 4 (8%) that had abnormal findings both on post-operative scan and clinically. Re-operation on 2 limbs corrected the duplex findings and the symptoms. There were 4 (8%) limbs that had normal duplexes but continued with symptoms albeit varied from the presenting symptoms. In the longer term, a further 2 became symptomatic at 2.8 years requiring a further successful intervention. (Concomitant popliteal venous obstruction was present in 5 limbs (10%) on standing.)

Conclusions: In the claudicating sportsperson, where there are no well characterised specific anatomical abnormalities, the syndrome can be characterised by provocative clinical (particularly hopping) and non-invasive tests. A positive clinical outcome with surgery can be predicted by abnormal pre-surgical ultrasonic investigations and confirmed later by a similar normal post surgical study. Concomitant venous compression may occur while standing with both syndromes related to muscle hypertrophy.

* Corresponding author. R.J. Lane, Suites 13 & 14 Greenwich Square, 130-134 Pacific Highway, St Leonards, NSW 2065, Australia. Tel.: +61 2 9438 4500; fax: +61 2 9906 1573.
E-mail address: rodlane@vsim.com.au (R. Lane).

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Introduction

The role of anatomical arterial abnormalities in the popliteal fossa has been well demonstrated.[1–3] A much more common clinical situation is the presentation of a sportsperson with typical claudication symptoms without a definitive anatomical abnormality. Rignault[4] has suggested the term "functional popliteal entrapment" (FPE). The symptoms, nevertheless, are equally debilitating and may produce significant lifestyle interference, particularly for the professional sportsperson. The characterisation of the syndrome and decision to treat may be confusing particularly as non-invasive investigations such as ankle brachial indices (ABI) and duplex scanning may be abnormal in a significant proportion of asymptomatic individuals.[3–5] Turnipseed[6] has also highlighted the need to differentiate chronic exertional compartment syndrome (CECS) from FPE. Deshpande[7] has shown that many patients with FPE syndrome have had failed fasciotomies for CECS yet respond well to division of the medial head of the gastrocnemius. The aim of this communication is to define a clear clinical pathway for FPE by comparing the pre and post-operative symptoms and non-invasive investigations in 55 limbs of claudicant sporting individuals. In addition, to differentiate and characterise concomitant popliteal venous obstruction.

Methods

Patients: demographics

All patients were referred from sports' physicians and had extensive non-invasive screening before presentation. In particular, compartmental pressure to exclude CECS, bone scans to exclude stress fractures and periosteal abnormalities, and ultrasound + MRI to exclude musculo-tenderness abnormalities. There were 15 male and 17 female patients with a mean age of 27 ± 8 years. Of the 32 patients, 25 (78%) were involved in various types of professional sports (football, basketball, hockey) or were in the military, police or other occupations which required significant physical fitness for employment. Patients prior to surgery had prolonged conservative (non operative) treatment. The exceptions were 7 limbs that had been operated previously for popliteal entrapment. Thirteen limbs had previous tibial fasciotomies for presumed compartmental syndrome with no improvement in their symptoms. Patients with known definitive anatomical abnormalities[1] were excluded.

Symptoms and signs

All patients had significant reproducible claudication affecting lifestyle and were unable to continue in their normal chosen profession or exercise activity. Associated symptoms were weakness, foot drop, paraesthesiae and the development of "rock-like" hard calves. There were 5 patients who had complained of swelling, restless legs and cramping at rest suggestive of venous symptoms as well as claudication. The patients were examined for hypertrophied calves as well as a full arterial evaluation.

The patients were then asked to undergo exacerbating and provocative manoeuvres in the presence of the examiner. It was often helpful to ask the patient to hop on the more affected leg. The aim was to connect exercise induced pain (subjective) to vascular signs (objective). The findings were the development of a cold pale foot, numbness of the toes with associated weakness, followed by reactive hyperaemia after cessation of exercise. The observations were compared with the non-affected side. Patients who specifically developed symptomatology ascending stairs, were also asked to try and reproduce these symptoms with the clinical observations of pallor and reactive hyperaemia after cessation of the symptomatic exercise.

Investigations

Ankle brachial indices (ABI)

The ABIs were measured at rest and on a treadmill with a 10° gradient using an initial "warm up" speed of 6 km/h. The speed was then increased to produce symptoms. If typical symptoms did not develop, the patient was then asked to run outside and return with symptoms. There were logistical difficulties in measuring exercise brachial systolic pressures simultaneously with posterior tibial and dorsalis pedis vessels, as the recovery times in these patients were often rapid. The exercise pressures were compared to the baseline resting brachial pressures. The standard response to exercise is an increase in systolic pressure.[8] The end points were a reduction in indices, a failure to increase the index with exercise with a normal increase in brachial pressure >5 mmHg above resting systolic.

Duplex examination

The most important examination was the duplex examination of the popliteal vessels after standing on tiptoe repeatedly and rapidly at least 20 times, or interrupted by the reproduction of the symptomatology. The diagnostic findings were a reduction in diameter of the popliteal artery (75% or more) where the two heads of the gastrocnemii meet, a relative change in velocities in the popliteal fossa with plantar flexion, and distal waveform reduction. Reactive hyperaemic response, as shown by an increase in the velocities, was also important. The latter findings are demonstrated in Fig. 1. Similarly, a complete cessation of flow in the popliteal artery indicated complete occlusion. The popliteal vein was examined ultrasonically with the knee slightly flexed while standing, and then with the knee locked (fully extended). The AP and lateral diameters, circumference and cross sectional areas were calculated automatically by the computer. An 85% reduction in area was considered abnormal (Fig. 2).

Angiography

Standard angiography was performed with active plantar flexion and dorsi flexion. Colour rendering for flow changes were helpful in indicating the degree of obstruction, Fig. 3. Magnetic resonance angiography or computed tomography angiograms were alternatives.

MRI and compartmental pressures

Compartmental pressures and MRI had usually been performed previously to exclude alternate pathologies. Abnormal compartmental pressures with a diagnosis of CECS were treated before vascular investigations were instigated. A summary of the investigation process is shown in Fig. 4.

Surgical method

The patient was positioned prone under general anaesthetic, with the isolated limb draped and prepared to allow easy manipulation of the foot and in a plastic bag to facilitate vascular inspection. A posterior approach is preferred to inspect the origin of the medial head of the gastrocnemius. A lazy "s" skin incision with the horizontal section over the lower crease gives an appropriate exposure. The popliteal fascia was divided over the medial head of the gastrocnemius avoiding the short saphenous vein and sural nerve. The medial head of the gastrocnemius was dissected free from the neurovascular bundle. The popliteal artery was located and inspected with passive dorsi flexion and plantar flexion. The deep part of the medial head was divided at the origin

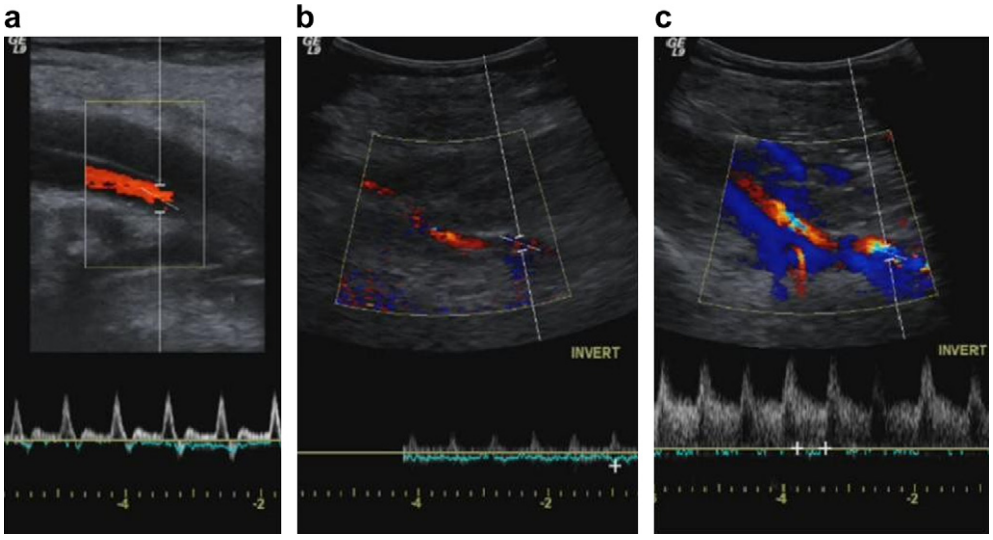

Figure 1. Doppler findings with FPS a) Popliteal artery longitudinal duplex scan in neutral position. b) Same popliteal longitudinal scan with plantar flexion. c) Reactive hyperaemic response following resumption of normal position.

from the lateral aspect of the medial condyle and posteriorly between the intercondylar notches. The only part remaining was the superficial gastrocnemial muscle arising from the dorsal surface of the medial condyle. The popliteal artery was traced distally to ensure no residual compression or any other anatomical abnormalities were present. Occasionally it was necessary to divide the entire medial head of the gastrocnemius particularly in recurrent cases, who were approached through the same scar posteriorly. A segmental division was performed using a right angle and diathermy, with retractor protection of the neurovascular bundle lying laterally. A posterior fasciotomy was performed extending two thirds down the calf in the subcutaneous plane and a large drain was inserted. The popliteal fascia was not reconstituted, and interrupted subcutaneous sutures as well as a continuous subcuticular skin suture was used. Compression bandages to the calf and popliteal fossa completed the procedure. The drain was removed when there was little or no drainage and the patient was mobilised and sent home, usually on the same day. Prophylactic anticoagulation was given subcutaneously using a low molecular weight heparin both at the commencement of the procedure and on discharge from hospital, but further anticoagulation was not continued.

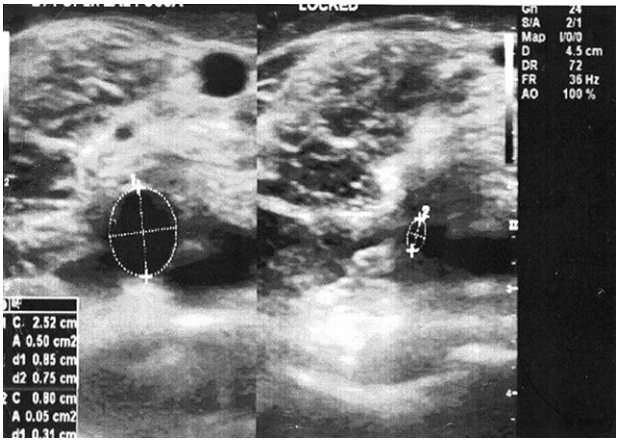

Figure 2. Venous compression syndrome. Area deduction with and without knee locking (extended). There is concentric reduction in both axial diameters with 90% total area reduction. The computer has measured the cross sectional areas of the flexed knee at 0.50 cm^2 going down to 0.05 cm^2 with knee flexion.

Patient follow up

The patients were reviewed before discharge from hospital and seen 6 days later after the compressive bandages were removed. The patient was again reviewed at 3 months with non-invasive investigations, or beforehand depending on clinical need. Long-term patients were also reviewed on clinical need and yearly. Symptoms were recorded as progressive (worse), unchanged, residual (improved) or asymptomatic.

Statistics

A χ^2 analysis of the pre and post-operative variables was performed. Students "*t*" tests were performed for the venous variables.

Results

Table 1 summarises the pre and post-operative ABI results.

Table 2 summarises the clinical outcome and post-examination duplex examinations performed in 52 limbs. Three patients had successful clinical outcomes but were unable to have ultrasonic investigation for logistical reasons.

Longer term follow up

There were seventeen limbs available for follow up in the long term. Two were symptomatic with abnormal duplex, fifteen were asymptomatic, eight were available for duplex scans, all of which were normal, and seven of the eight had normal ABIs. Two limbs had successful repeat procedures.

Table 3 summaries the symptomatic failures and complications.

Table 4 summarises the pre or post-operative popliteal vein cross sectional areas with and without knee locking (extended) in the 5 limbs with venous symptomatology as well as claudication. All 5 patients were free of claudicants symptoms at follow up. One patient had considerable residual swelling at 3 months. In the last 11 cases, particular observations of the origin of medial head of

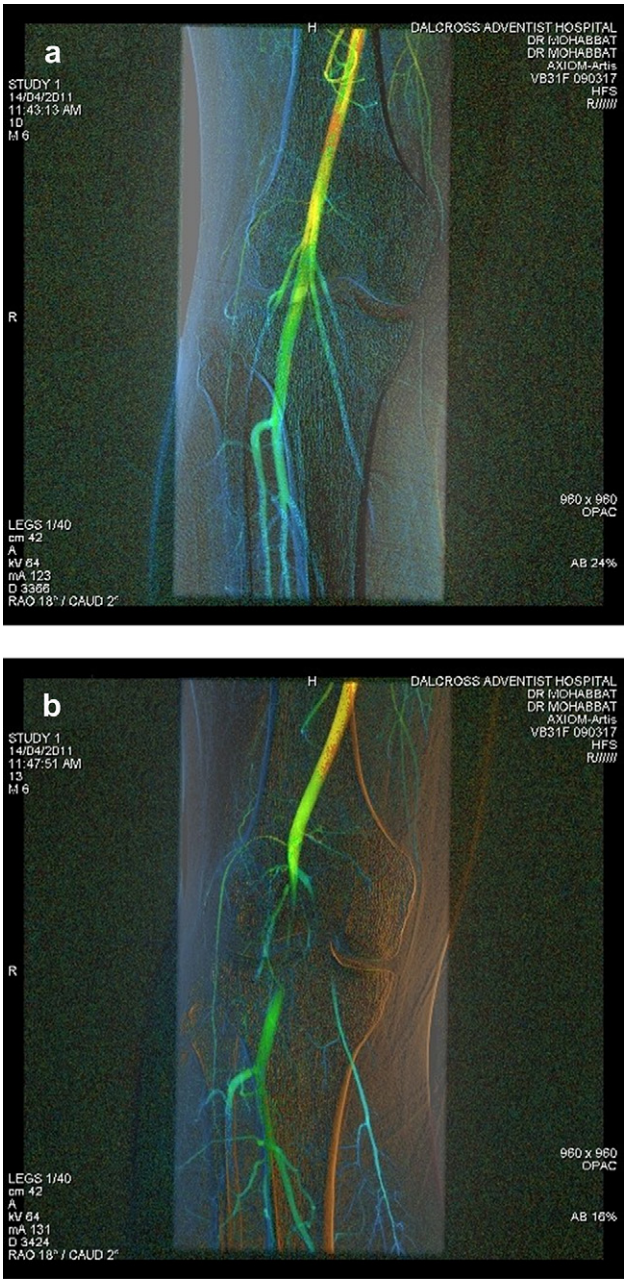

Figure 3. Angiographic colour rendering for F.P.E. syndrome. Decreasing flow code: red, yellow, green, blue. a) At rest: Popliteal artery open. Non-muscle collaterals small; collaterals through soleus and gastrocs open. Mild increase in flow (yellow) between the heads of the gastrocnemii. b) With active plantar flexion, there is complete occlusion of the popliteal artery and muscular collaterals with an associated increase in flow of non-muscular collaterals. Slow flow (blue) distally.

gastrocnemius were recorded. In 7 of these, the muscle arose from the intercondylar notch or lateral surface of the medial femoral condyle.

Discussion

The clinical ABI and duplex outcome after surgery confirms the pre-operative clinical approach is stringent enough to define the clinical syndrome of functional popliteal entrapment. A positive clinical outcome was associated with a reversal of the abnormal pre-operative vascular testing. Two patients with residual symptoms sustained an area of increased muscle bulk (localised mass of

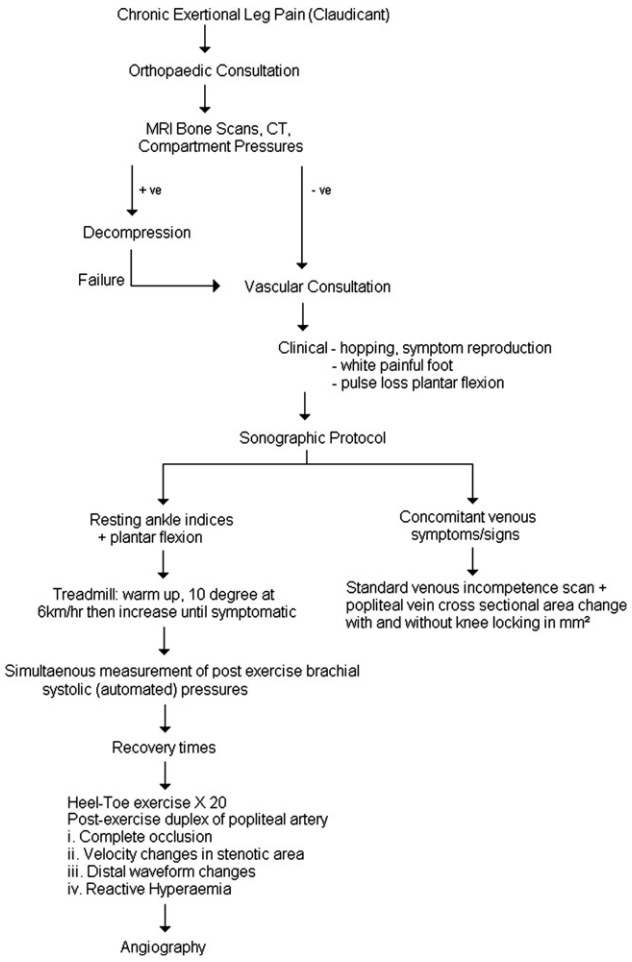

Figure 4. Suggested clinical and investigative protocol for functional popliteal entrapment.

shortened muscle fibres), as shown on post-operative MRI. There were three limbs that had no clinical improvement and one limb with significant residual symptoms, with abnormal duplexes post-operatively. The assumption is that the myotomy was inadequate. It is unclear why two limbs should develop anterior tibial compartment syndromes post-operatively when the pre-operative compartment pressures were normal.

There were 6 post-operative asymptomatic limbs with abnormal duplexes. It is possible that enough muscle was divided at operation to avoid symptomatology and yet these continued with some form of residual obstruction of the popliteal artery. In cases presenting with long-term recurrences requiring repeat surgical intervention ($n = 7$) the findings were of extensive fibrosis surrounding the neurovascular bundle ($n = 5$) rather than inadequate myotomy ($n = 2$).

Table 1
Pre and post-operative ABI results.

	Results		
	Pre-op: $n = 55$	Post-op: $n = 52$	$P(\chi^2)$
Resting > 1.0	53	51	NS
Exercise < 1.0	31	1	$<0.05\ \chi^2 > 10.8$
0.69 ± 0.19			
Exercise; no increase from resting baseline	19	5	$<0.05\ \chi^2 > 10.8$
Exercise increase	5	46	$<0.05\ \chi^2 > 10.8$

Table 2
Post-operative results: $n = 52$. Symptoms versus duplex outcome.

	Duplex		
	Normal	Abnormal	Total
No Symptoms	40	4	44
Symptoms	4	4	8
	44	8	52

Re-operated with normal outcome in both modalities.

Surgery was routinely performed posteriorly to allow better visualisation of the popliteal vessels and the relationship to the deep part of the medial head of the gastrocnemius, with removal of the origin of the muscle directly from the bone. Transection of only the deeper part of the gastrocnemius, which is immediately adjacent to the neurovascular bundle, is suggested to minimise the total power loss. Digital palpation of the neurovascular tunnel between the heads of the gastrocnemii with plantar flexion/extension is suggested to ensure unobstructed passage. Levien[3] confirms the post-operative approach gives better access.

Clinical presentation and investigations

The most important feature of the diagnosis is the reproduction of symptoms with verification by duplex ultrasonography with the help of the provocative tests. Clinically, observing the patient hopping at the same rate as the heart rate was very helpful. The physiological basis of this clinical finding relates to the increase in systolic pressure and the decrease in the diastolic pressure with exercise.[8] If the calf muscles are contracting at the same rate as the heart then only diastolic flow is supplying the muscle; similar to cardiac muscle blood flow. Fig. 5 shows the relationship of muscle contraction in a timeline with normal running.[9] The gastrocnemial/soleal group contracts 40% of the time. With popliteal arterial obstruction at the same rate as cardiac systole, the calf muscles are even more compromised hence the very rapid onset of symptoms. The standard clinical approach is shown in Fig. 4. Considering the invasive nature of pressure testing, perhaps an earlier non-invasive vascular workup would be appropriate if hopping produces an ischaemic limb. The ultrasonic findings are best performed after exercise with the patient repeatedly standing on tiptoe. If the duplex scan confirms partial or complete obstruction with provocative manoeuvres, then angiographic investigation is indicated.

The exercise ABIs give a good indication of the degree of functional abnormality (Table 1). However, because of the often rapid recovery times, the sonographic protocol has been modified to use an automated brachial pressure cuff to allow simultaneous measurement of the ankle pressures. The ABIs presented in the results underestimate the functional disability as the exercise ankle and brachial pressures were compared with the resting baseline brachial systolic pressures. In a few very fit individuals, although symptomatic, the ABIs were normal with the diagnosis confirmed on duplex and angiographically.

Colour rendered angiography (Fig. 3) may also be helpful particularly in defining the collateral pathways. With complete occlusions, the non-muscle collaterals dilate and the intramuscular pathways occlude. Where popliteal occlusion is incomplete, the relative increased flow (shown in colour) at the level of obstruction contrasts to the slow flow distally related to the increased peripheral resistance in the contracting muscles as well as the proximal obstruction.

Table 3
Analysis of all failures and complications.

Symptoms	Number	Aetiology
Progressive	2	Increase in divided muscle bulk
Unchanged	3	Inadequate myotomy
Residual	3	Shin splints, unknown
Haematoma	2	Early drain removal
Infection	1	Wound separation

Table 4
Pre and post-operative popliteal vein areas with and without knee locking (cm^2) – i.e. normal standing compared with knee slightly flexed.

Popliteal vein	Pre-operative	Post-operative
Unlocked	0.85 ± 0.37	0.72 ± 0.35
Locked	0.08 ± 0.05	0.69 ± 0.21

Associated venous compression

The venous component is also scanned ultrasonically with the patient standing with the knee locked or hyperextended and then with the knee unlocked i.e. slightly flexed, Fig. 2. Table 3 shows the changes post-operatively compared with the pre-operative findings.

Raju[10] documented a subset of patients presenting with the clinical features of chronic venous disease (C.V.I) with radiological features of obstruction of the popliteal vein with plantar flexion. He showed an increase in popliteal venous pressure with a simultaneous reduction in the ankle venous pressures. These findings are consistent with the concept of normal muscle pump function. The medical literature suggests that the pathological state may exist with the knee hyperextended (supine)[11,12] or when the knee is locked (standing).[13] The pathophysiology arises with the hypertrophy of the calf muscles required to power the increased body mass. Ultrasonically, the gastrocnemii are relaxed and crowded into the popliteal space with knee locking (extended).[13] There were differences in the clinical presentation with these ultrasonic findings. Only 5 limbs presented with popliteal vein obstruction while standing as well as arterial claudication. Patients with venous symptoms were older >30 years, had a BMI of >30, and who were more often more sedentary e.g. policemen, front row forwards, and had symptoms or signs typical of chronic venous insufficiency. They presented with swelling, aching, with restless legs while standing, and compulsion to shift the weight from one leg to the other and often searching for a place to sit down. Three limbs had early lipodermatosclerosis with sub malleolar flaring with minimal venous incompetence.

The duplex imaging of the veins is pathognomonic of an overall increase in pressure in the popliteal fossa rather than a side-to-side compression caused by two muscles acting as pincers (Fig. 2). Both the AP and lateral diameters are symmetrically reduced with the knee locked (extended) like the iris of a camera. In comparison, the popliteal arteries are compressed from one side to the other producing a reduction in one diameter only. The concept is confirmed at operation in the patients with venous obstruction having bulging of the hypertrophied popliteal fat pad and spontaneous wide separation of the edges of the popliteal fascia after incision. Removal of the popliteal fat pad and leaving the popliteal fascia unsutured improved the pre and post-operative duplex findings. In the much larger series,[13] the ultrasonic diameters changed from 11.7 ± 2.5 mm with the knee flexed to 1.0 ± 2.1 mm with the patient standing (knee locked (extended)). Post-operatively, the diameters were 10.2 ± 2.2 mm with the knee flexed to 9.0 ± 1.5 mm with the patients standing. This very significant improvement in diameters coincided with a significant improvement in symptoms. Similarly in these 5 patients, the venous symptoms, particularly swelling, diminished.

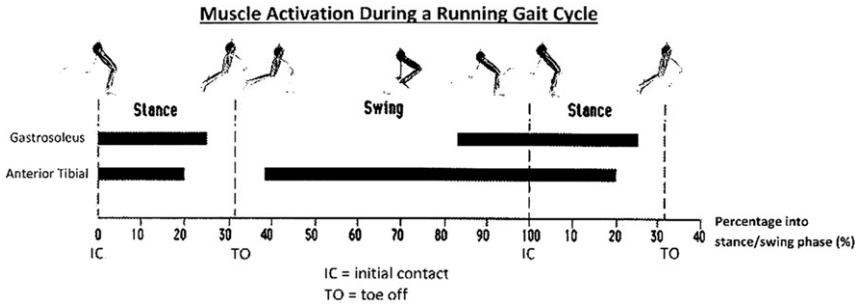

Figure 5. Muscle activation during a running gait cycle. Gastrosoleus group contracting 40% of the gait cycle. Anterior tibial contracting 80% of the gait cycle. The faster the run the less the perfusion time.

Associated limb compartment syndrome and recurrences

There were 13 limbs that previously had unsuccessful tibial compartment decompressions. Considering these patients had demonstrable arterial compromise and assuming ischaemia induces swelling, FPS may cause an increase in compartment pressures with exercise. It appears reasonable to suggest calf compartment decompression operation should be preceded by a vascular workup. As the contraction time is longer and therefore perfusion time less (Fig. 5) the anterior tibial compartment is much more liable to sustain ischaemia during exercise. The analysis of recurrences $n = 9$ (7 from the critical presentation and 2 from the present series) underpins the cause (popliteal entrapment) and effect (claudication), similar to a modern day Koch's postulates[14] (Fig. 6).

The possible anatomical abnormalities

Pillai[15] has defined a group of patients with functional popliteal syndrome where the medial head of the gastrocnemius arises more laterally. In seven of the last eleven cases in this series, the medial head arose in the intercondylar notch or attached laterally and inferiorly to the medial condyle. The overwhelming anatomical abnormality was the massive hypertrophy of the gastrocnemial muscles. Further pre-operative imaging analysis is in progress to confirm these observations.

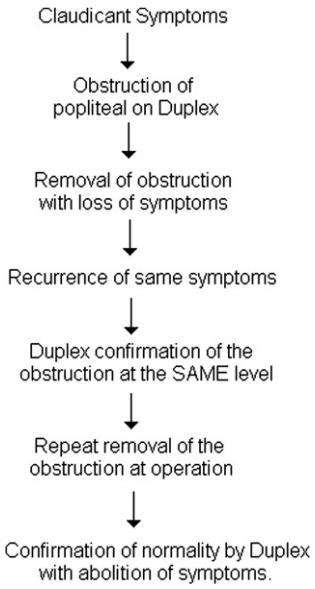

Figure 6. Cause and effect flow chart – justification of F.P.E is a pathological process.

Differential diagnosis

As these patients are often triathletes, Dutch bike rider's disease may affect the calf muscles primarily. However, in these cases, more often the thighs are affected, and on exercise ultrasonography, there are high velocities at the origin of the external iliac artery often confirmed by angiographic findings. The duplex scan of the popliteal fossa is normal under these circumstances. Occasionally early atheroma, dissection or embolisation may present a diagnostic problem but these alternate diagnoses are usually defined by standard duplex scanning. The other findings of muscle injury, stress fracture and tendonitis must be considered but are usually defined before presentation to the vascular surgeon.

Conclusion

Functional popliteal syndrome is related to hypertrophy of the medial head of the gastrocnemius muscle with popliteal artery occlusion during contraction. In approximately 10% of cases, the same hypertrophied musculature obstructs the popliteal vein during relaxation (standing). Radical transection of the medial head of the gastrocnemius muscle produces satisfactory results. The diagnosis is clinical with hopping induced symptomatic ischaemia being an important finding. Ultrasonic examination with provocative exercise manoeuvres and angiography confirm the diagnosis. Recurrences can occur but are best treated by further surgical intervention.

Ethical Approval

NA – Government already approved procedure.

Conflict of Interest/Funding

None.

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