ORIGINAL CLINICAL SCIENCE STUDIES

Popliteal artery entrapment syndrome: More common than previously recognized

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Purpose: This report summarizes our experience with the popliteal entrapment syndrome in 88 limbs (48 patients) treated during a 10-year period.

Method: The study cohort consisted of a retrospective analysis of those patients who were seen with symptoms of claudication or severe ischemia by a single surgical group and in whom unequivocal evidence of popliteal entrapment was shown either with angiography or at the time of operation. The cases were collected prospectively in a private vascular surgical practice.

Results: Bilateral popliteal entrapment was found in 40 of the 48 patients. The mean age at the time of presentation was 35.0 years (SD, 11.6 years). Claudication was the most frequent presenting symptom (70 of 88 limbs). Types I, II, III, and IV popliteal entrapment were found in 58 limbs (15 arteries occluded), and 30 limbs (three occlusions) were seen with a "functional" popliteal artery entrapment (apparent absence of a developmental anatomic abnormality). Of the 18 limbs with severe ischemia and associated occlusion of the popliteal artery, 15 underwent bypass grafting with reversed saphenous vein grafts, all of which remained patent during the follow-up period (median follow-up, 4.2 years; range, 1 to 10 years). One popliteal artery occlusion that was treated with thrombectomy and vein patching occluded within 6 months and necessitated subsequent vein grafting. Two limbs with inoperable occluded popliteal arteries were not subjected to reconstruction (one necessitated amputation because of advanced ischemia, and the second had extensive thrombosis of the distal run-off). In two patients (four limbs), moderate presenting symptoms abated without surgery after the discontinuation of an extreme exercise program. The remaining limbs underwent surgical decompression (all popliteal arteries remained patent, with a median follow-up of 3.9 years).

Conclusion: The popliteal entrapment syndrome is more prevalent than has formerly been appreciated. On the basis of observations made in this series and in the surgical literature, we advise surgical correction in all cases of types I, II, III, and IV entrapment at the time of diagnosis to avoid occlusion as a result of continued arterial wall degeneration. In contrast, decompression is only advised in those patients with "functional entrapment" if they have discrete and typical symptoms because up to 50% of the normal population may display transient popliteal artery compression with extremes of plantar flexion or dorsiflexion. On the basis of the severe histologic changes found in those popliteal arteries that had undergone occlusion at the time of presentation, it is advised that the popliteal artery should be completely replaced, ideally with a vein graft, when significant degeneration or occlusion of the popliteal artery is noted at the time of operation. (J Vasc Surg 1999;30:587-98.)

First described by a medical student[1] who dissected an amputated leg in Edinburgh in 1879, popliteal artery entrapment syndrome was thought to be a rare phenomenon. After the first description of a clinical case by Hamming[2] in 1958, various isolated cases were reported.[3-19] In the mid 1960s, the term *popliteal artery entrapment syndrome* was introduced.[4,7] Servello[20] was the first to draw attention to the reduction in palpable distal pulses usually observed with forced plantar flexion or dorsiflexion in patients with this syndrome. Biemans and Van Bockel[21] focused attention on the clinical syndrome of popliteal artery entrapment in an extensive review of the literature in 1977.
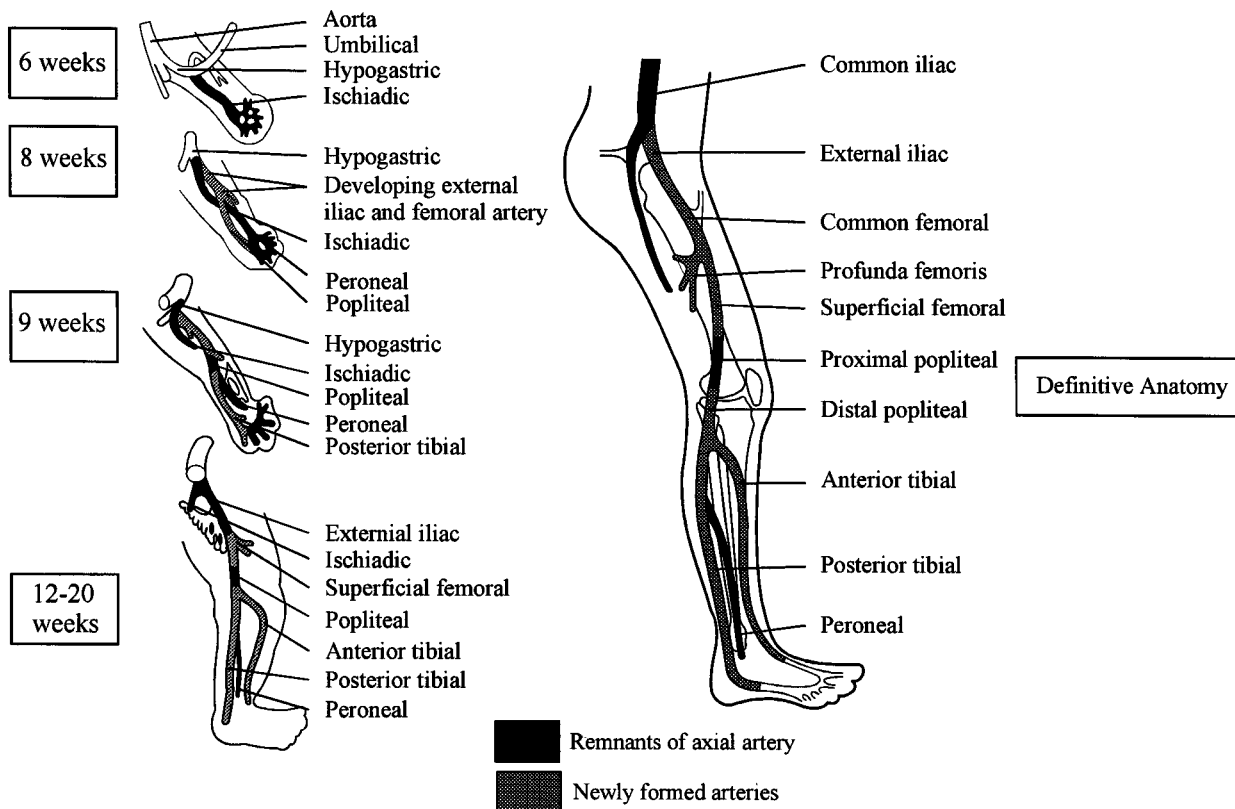
Fig 1. Embryologic derivation of popliteal and other lower limb arteries indicating which are derived from the axial artery and which nonaxial arteries develop with later differentiation.

The true incidence of the popliteal artery entrapment syndrome is unknown. Early authors believed the condition to be rare,[4,7,21-30] but it has become apparent that the condition is considerably more common than previously appreciated.[25-44] Bouhoutsas and Daskalakis,[34] in the first large series of cases described, reported an incidence of 0.165% in young males entering the Greek military service, and Gibson et al,[23] in a postmortem study, found a prevalence of 3.5%. The finding of compression or occlusion of the popliteal artery with forced plantar flexion or dorsiflexion occurring in a large proportion of healthy asymptomatic individuals has unfortunately precluded noninvasive tests or duplex Doppler scanning being used as a potential screening tool for accurately evaluating the occurrence of popliteal artery entrapment syndrome in the asymptomatic general population.[45-50] In the young athlete with claudication-like symptoms, popliteal artery entrapment syndrome may be the underlying cause of the symptoms in up to 60% of cases.[36,39] The entrapment mechanism has been documented to involve the popliteal vein in up to one third of cases.[6,8,21-25] The bilateral occurrence of the

condition was at first assumed to be rare, but recent reports have indicated a higher prevalence of bilateral disease than previously reported.[4,12,23,26-28] Popliteal artery entrapment syndrome has been reported to occur in more than one individual in a family.[29]

The objective of this study is to highlight the importance of the embryologic development of the vascular and muscular structures of the popliteal artery in the genesis of the various types of popliteal artery entrapment and to present our experience with this condition during a 10-year period up to December 1997.

Embryology and classification. Early attempts to classify the various types of popliteal artery entrapment syndrome were made on the basis of the anatomy observed at operation.[30,34] Subsequently, a better appreciation of the embryology of this condition led to a classification on the basis of the developmental anatomy, with five types of popliteal artery entrapment syndrome currently described.[26,38]

The anatomy observed in the various types of popliteal artery entrapment can be best appreciated by considering the developmental anatomy of the
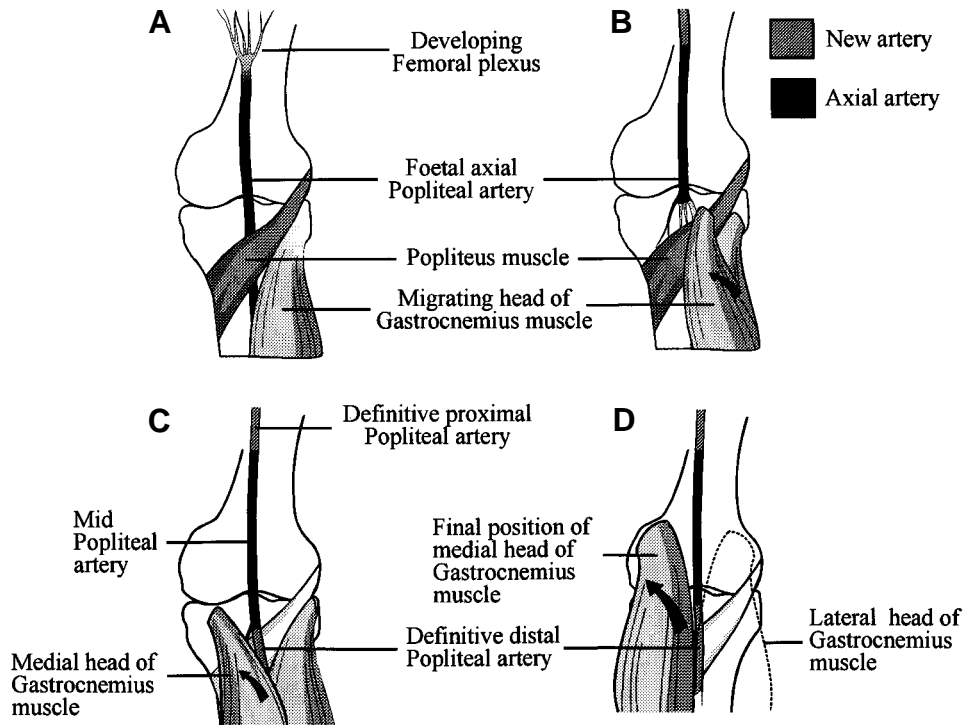
Fig 2. Pictorial representation of migration of the medial head of gastrocnemius muscle through the popliteal fossa during formation of the popliteal artery. **A,** Medial head of gastrocnemius muscle commences migration from region of fibula. At this stage, axial distal popliteal artery lies deep to popliteus muscle. **B,** Distal portion of popliteal artery involutes as medial head of gastrocnemius muscle passes from lateral to medial. Proximal popliteal artery is derived from fusion with developing femoral plexus, and mid portion of popliteal artery is formed from persistent axial artery remnant. **C,** New or nonaxial distal popliteal artery now forms superficial to popliteus muscle, after medial head has migrated through popliteal fossa. **D,** Normal definitive popliteal anatomy.

popliteal fossa. In lower order animals, the medial head of the gastrocnemius muscle arises proximally from the posterior aspect of the fibula and lateral tibia. During development in the human, with limb bud rotation medially and extension of the knee, the medial head of the gastrocnemius muscle migrates from its original lateral position,[51,52] across the popliteal fossa. With further development, the definitive attachment of the medial head of the gastrocnemius muscle is to the posterior surface of the medial femoral condyle.

The embryologic popliteal artery in the developing limb bud is the continuation of the primitive axial or the ischiadic artery (Fig 1).[53,54] The proximal portion of the adult popliteal artery develops in continuity with the developing femoral artery[51] and is derived from fusion of the developing femoral arterial plexus and the popliteal remnant of the axial artery. The mid portion of the definitive popliteal artery is directly derived from the remnant of the axial artery. The

primitive distal popliteal axial artery, which lies deep to the forming popliteus muscle, disappears at about the 20-mm to 22-mm stage of the embryo, and the definitive distal popliteal artery forms superficial to the popliteus muscle by the fusion of two new vessels (the newly forming anterior and the posterior tibial vessels) after the medial head of the gastrocnemius muscle has migrated medially across the popliteal fossa, as illustrated in Fig 2. The medial head of gastrocnemius muscle therefore migrates through the popliteal fossa at about the same time as the rearrangement of the arterial structures.[51]

If the definitive distal popliteal artery forms before the migration of the medial head, the newly formed artery may be swept medially with the definitive artery now lying medial to the normally placed medial head of the gastrocnemius muscle. This results in the classical or type I popliteal entrapment and is associated with a marked medial deviation of the popliteal artery

Type I

Type II

Type III
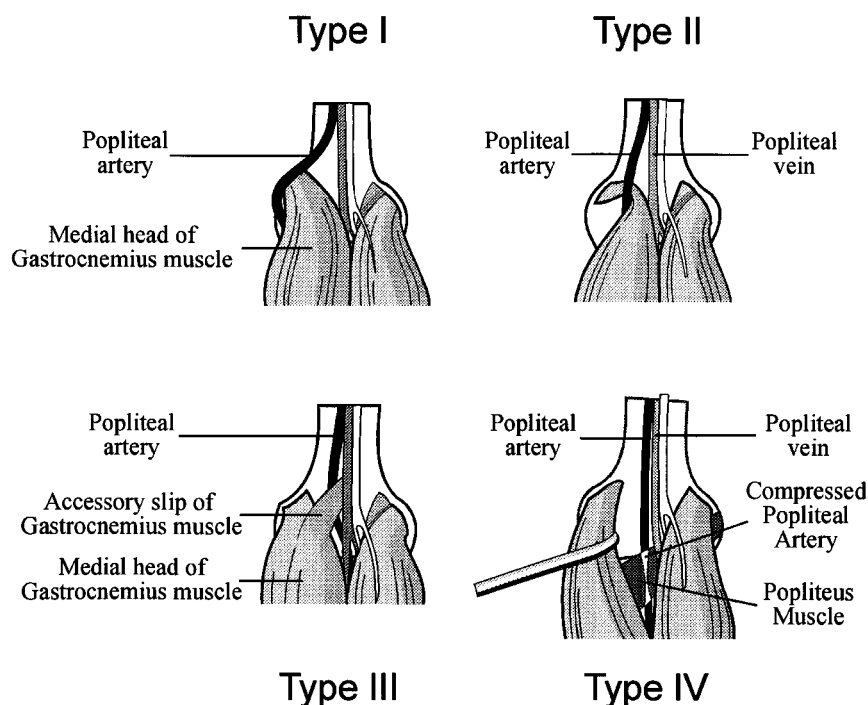
Type IV

Fig 3. Classification of types of popliteal artery entrapment syndrome.

in the popliteal fossa, both anatomically and on angiography, as depicted in Fig 3 (type I).

Alternatively, a prematurely formed definitive distal popliteal artery may partially arrest the migration of the medial head to a degree, resulting in a type II entrapment. In the type II entrapment, the popliteal artery is medially displaced to a lesser degree and lies deep and medial to a medial head of the gastrocnemius muscle, which has a variable attachment to the lateral aspect of the medial femoral condyle or intercondylar area. The artery therefore lies on the medial aspect of an abnormally placed medial head of gastrocnemius muscle, as shown in Fig 3 (type II).

If mesodermal remnants of the migrating medial head persist posterior to the popliteal artery or if the artery develops within the migrating muscle mass, a type III popliteal entrapment may result. Here the entrapment mechanism is formed either by an abnormal slip of mature skeletal muscle or by fibrous and tendinous bands derived from the remnants of the migrating medial head. These abnormal additional slips of muscle tissue may arise from either the medial or the lateral femoral condyles (Fig 3; type III). The definitive popliteal artery may even pass between a double origin of the medial head of gastrocnemius muscle.

If the axial artery persists as the definitive distal popliteal artery, it will lie in the primitive position, deep to the popliteus muscle or fibrous bands,[55] resulting in a type IV entrapment (Fig 3; type IV). It can therefore be appreciated that the entrapment types I to III are not discrete entities but rather a spectrum of anatomic abnormalities, with the adult anatomy being dependant on the time relationship between the stage of migration of the medial head of the gastrocnemius muscle and the evolving distal popliteal artery.

When any type of entrapment mechanism includes or surrounds the popliteal vein and the artery, Rich et al[8,26] term this event a type V entrapment.[6,21-23,56] Any of the types of entrapment (with the possible exception of the type I), may include the tibial nerves, resulting in neurologic paresthesia in addition to claudication as the presenting symptom.[32]

A type of popliteal artery entrapment occurs in individuals who have typical symptoms of popliteal entrapment and compression of the popliteal artery with stress maneuvers but in whom there is no apparent anatomic abnormality—this condition being termed "functional" entrapment.[46,47,50] In such individuals, the exact nature of the entrapment mechanism remains uncertain. It has been postulated that a hypertrophic medial head of gastrocnemius muscle impinges on the medial and posterior aspect

of the popliteal artery[45,46] and can cause physiologic occlusion of the artery in extreme plantar flexion or extension. Other investigators have postulated and advanced evidence for the entrapment occurring as a result of compression of the distal popliteal artery as it passes through the soleal muscle sling.[47] Up to half of apparently healthy, asymptomatic individuals may display the phenomenon of reduced or abolished popliteal artery blood flow with extremes of plantar flexion or dorsiflexion against resistance,[45-47,49] most of these individuals being totally free of any clinical symptoms. The theory has been advanced that a more lateral attachment of the muscular portion of the medial head of the gastrocnemius muscle to the posterior aspect of the medial femoral condyle, possibly as a consequence of incomplete embryologic migration, may predispose such individuals to a "functional" type of entrapment, which is more likely to manifest clinically should they undergo muscle hypertrophy consequent on regular lower limb exercise.[36] It has been proposed that this "functional" type of popliteal entrapment be termed type VI.[36] An acquired type of entrapment has been described after infragenicular bypass grafting surgery.[57,58]

The clinical picture. The clinical diagnosis of popliteal artery entrapment relies on recognition of the clinical picture of calf claudication in the young and often athletic individual,[4,7,8,31,32] with ankle pulses normal at rest if occlusion has not yet occurred. Untreated, the compression mechanism frequently results in the deterioration of the popliteal artery with the passage of time, resulting in eventual occlusion.[33,36] The sudden onset of severe disabling claudication and absent ankle pulses, usually in the absence of risk factors predisposing the individual to atheroma, characterize those patients in whom occlusion of the popliteal artery has taken place as a result of popliteal entrapment. The development of critical ischemia with the occlusion of the popliteal artery is rare.[36] Distal emboli[23,25,42,59] may result consequently on focal thrombus formation[30,36] at the site of entrapment or from popliteal aneurysm formation[25,26,34,36,60] caused by the entrapment.

The diagnosis of popliteal artery entrapment syndrome may be confirmed with Doppler scan ankle pressures,[26,28,34,61,62] pulse volume recordings,[25] duplex Doppler scanning,[28,49,63-65] computerized axial scanning,[66-68] magnetic resonance imaging,[45,47,69,70] and magnetic resonance angiography. All of these methods rely on the demonstration of popliteal artery compression, with reduced or abolished popliteal artery blood flow occurring with forced active plantar flexion or dorsiflexion of the foot against resistance. However, the most widely used diagnostic method continues to be contrast angiography, particularly to plan surgery when degeneration, aneurysm, or occlusion of the popliteal artery is suspected.[38,44,71] As with the noninvasive tests, angiographic results may show an apparently healthy popliteal artery at rest and usually necessitate forced active plantar flexion or dorsiflexion of the foot against resistance to show the abnormality, provided the artery has not yet undergone degenerative changes.[47,50] In contrast, cases of popliteal adventitial cystic disease will usually show the characteristic angiographic abnormality, both at rest and on stress views.

The object of the current study is to present our experience with the popliteal artery entrapment syndrome during a 10-year period up to December 1997.

PATIENTS AND METHODS

Patients with claudication-like symptoms of the legs causing severe and debilitating symptoms, who were seen by a single group of surgeons, all underwent clinical examination followed by Doppler scan ankle/brachial pressure index measurement. In those patients in whom the distal pulses and ankle/brachial pressure index were normal in the symptomatic leg, the patients underwent screening with both popliteal artery duplex Doppler scanning and ankle Doppler scan recording during active plantar flexion and dorsiflexion against resistance. When these test results were found to be positive with reduced or abolished popliteal or distal flow with this maneuver (an alteration of > 0.2 in the ankle/brachial index), the patients were subjected to conventional contrast arteriography, both in the resting and in the forced plantar flexion and dorsiflexion positions for confirmation of the diagnosis.

In those patients in whom popliteal occlusion was suspected on the basis of reduced or absent ankle pulses at rest and in those patients in whom the ankle/brachial Doppler scan pressure index was less than normal at rest, popliteal artery entrapment syndrome was considered if this condition was shown in the contralateral leg. If subsequent angiography showed local popliteal artery occlusion with no evidence of proximal or distal atheroma, popliteal artery entrapment syndrome then was confirmed with exposure and demonstration of the entrapment mechanism at operation.

Only the patients with unequivocal evidence of popliteal artery entrapment, either on angiography or at operation, were included in this study. Only the patients with symptoms that were sufficiently severe

Fig 4. Examples of angiography of popliteal artery entrapment syndrome. **A,** Demonstrates medial and then lateral deviation of artery at rest as result of type III additional muscular head causing entrapment. **B,** Demonstrates localized entrapment of artery with plantar flexion as result of localized fibrous band causing type III entrapment. **C,** Demonstrates occlusion of distal popliteal artery with plantar flexion as result of type IV popliteal entrapment confirmed at operation. **D,** Demonstrates localized popliteal artery aneurysm formation as result of popliteal artery entrapment syndrome.
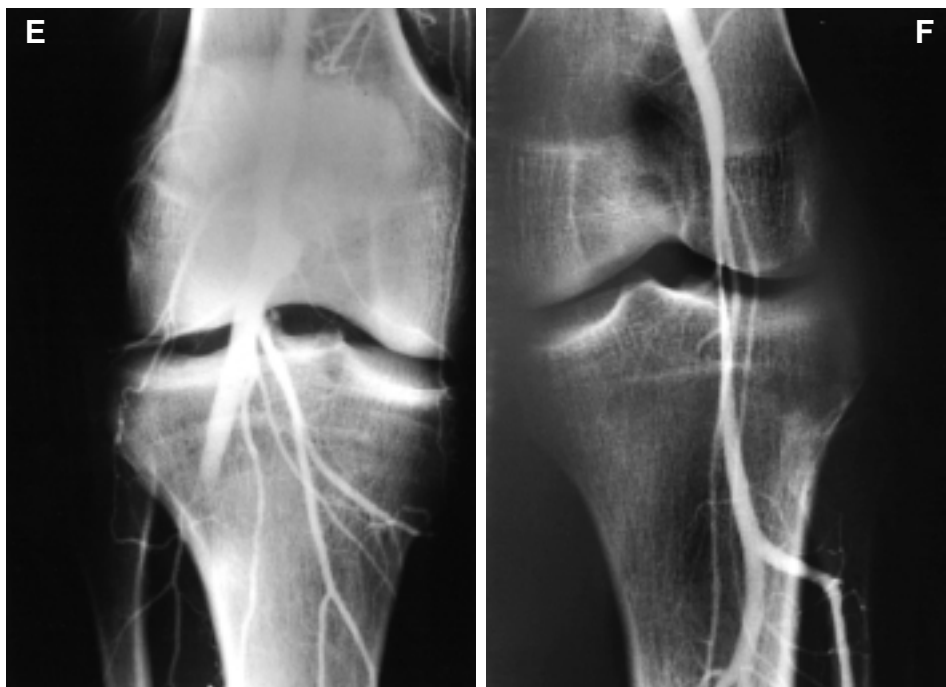
Fig 4. Cont'd. E, Demonstrates embolic occlusion of distal popliteal artery, embolus originating from aneurysmal change because of popliteal entrapment. **F,** Demonstrates longer smooth entrapment often seen with "functional" or type VI entrapment.

enough to cause them to discontinue their sporting activities were considered for surgery if the preoperative picture suggested "functional" popliteal vascular entrapment. Patients were not included if their symptoms were not typical of popliteal artery entrapment syndrome, and no patient was included in the study on the basis of positive noninvasive tests alone.

RESULTS

A total of 88 limbs in 48 individual patients was included in this study during a 10-year period from January 1988 to December 1997. The types of entrapment, presenting features, and treatments are summarized in Tables I and II. In 40 patients, the symptoms and entrapment were present bilaterally, and in eight patients, the condition was either totally asymptomatic or not present in the contralateral leg. The mean age for all the patients was 34.9 years (SD, 11.64 years; range, 16 to 55 years). There were 53 limbs in the male patients (mean age, 36.8 years; SD, 11.36 years; range, 16 to 55 years), and 35 limbs in the female patients (mean age, 32.0 years; SD, 11.46 years; range, 16 to 52 years).

In 70 limbs, angiographic results confirmed the presence of popliteal artery entrapment syndrome and showed an apparently undamaged and patent popliteal artery. In 66 limbs, at the time of surgical exploration, a healthy popliteal artery was confirmed with intraoperative inspection and palpation of the entrapped area, followed by the release of the entrapment mechanism, usually by myotomy of the medical head of the gastrocnemius muscle, abnormal muscle slips, or tendinous bands responsible for the entrapment mechanism. All the patients who underwent treatment in this manner have remained with healthy and patent popliteal arteries during the follow-up period (median follow-up, 3.9 years; range, 1 to 10 years). Almost without exception, those patients who had previously been compelled to stop their sporting activities as a result of the symptoms of popliteal artery entrapment were able to resume normal sporting activities after postoperative recovery. In two patients, each with bilateral disease, popliteal artery entrapment syndrome of moderate severity was associated with extreme and unusual physical exercise (ballet dancing and competitive cycling). In these two patients, the angiographic results suggested moderate "functional" popliteal artery entrapment syndrome bilaterally with a long, diffuse narrowing of the popliteal artery on plantar flexion (Fig 4*F*) but an otherwise angiographically normal artery at rest. Both the patients elected to discontinue the extreme

Table I. Presenting features of 88 limbs with popliteal artery entrapment syndrome

Type	I	II	III	IV	Functional or type VI	Total
Total in series	5	12	33	8	30	88
Occlusion with severe ischemia	4	5	3	3	3	18
Entrapment causing typical claudication symptoms	1	7	30	5	27	70
Venous entrapment	1	3	6	0	0	10

Table II. Analysis of treatment of different types of popliteal entrapment syndrome

Type	I	II	III	IV	Functional or type VI	Total
Total	5	12	33	8	30	88
Occlusion	4	5	3	3	3	18
Myotomy and VG	3	4	3	3	3	16
Myotomy only	1	7	30	5	23	66
No operation	1	1	0	0	4	6

VG, Vein graft.

physical activity with consequent progressive and total resolution of their symptoms. Both patients remain well and asymptomatic with normal popliteal arteries on duplex scanning after 2 and 3 years follow-up, respectively.

Eighteen limbs were seen with occlusion of the popliteal artery or distal embolization as a result of aneurysmal change at the entrapment site (13 in male patients: mean age, 33.9 years; SD, 11.6 years; and five in females: mean age, 35.4 years; SD, 14.4 years). Of the 18 limbs with occlusion (Table II), 15 were treated with the replacement of the occluded segment of the popliteal artery with reversed saphenous vein grafts. Eight instances of aneurysmal change were noted in this group of patients, all associated with type I to IV entrapments. All the patients with aneurysmal change had popliteal arteries replaced with saphenous vein grafting. All 15 remain well and patent for the follow-up period (median follow-up, 4.2 years; range, 1 to 10 years), with 14 of the 15 returning to normal sporting activities. One 52-year-old man, who was seen early in our experience with right popliteal artery occlusion as the result of popliteal artery entrapment, underwent treatment with thrombectomy and vein patch of the popliteal artery. This patient returned with recurrent ipsilateral popliteal artery occlusion within 6 months. A saphenous vein graft replacement of the popliteal artery then was successfully performed. Although the patient did not return to sporting activities, the graft remains patent and healthy on duplex ultrasound scanning 9 years later. Three of the 18 arteries that had progressed to focal occlusion of the popliteal artery were found at exploration to have a type VI or "functional" type of entrapment, with focal degeneration of the artery (confirmed histologically) that had progressed to occlusion.

One 48-year-old woman was seen late at another center after occlusion of the popliteal artery (type I) and, after repeated attempts at catheter thrombectomy, she eventually required amputation for advanced ischemia of the limb. A 46-year-old woman (with type II occlusion) was seen with severe ischemia after considerable delay. Angiographic investigation showed extensive propagated thrombus in the distal circulation that precluded revascularization, and the patient underwent treatment with anticoagulation therapy. Each of these patients was shown to have popliteal artery entrapment syndrome in the contralateral limb that necessitated release with myotomy, and both of these patients were shown to have hypercoaguable states. On investigation, the first patient demonstrated increased platelet aggregation and the second patient was shown to have both anti-thrombin III deficiency and increased platelet aggregation. No other patients in this series with occlusion had critical ischemia develop.

One 42-year-old woman had previously undergone treatment with femoropoliteal vein grafting for undiagnosed local popliteal artery occlusion at another center. The patient then was seen by us with distal emboli arising from a type III popliteal artery entrapment syndrome that had not been recognized at the time of the original surgery, the vein graft having been placed deep to the entrapment mechanism. The patient underwent successful treatment with the release of the entrapment mechanism by myotomy with preservation of the original vein graft and was well with the graft remaining patent 2 years later.

Details of surgical procedures. Of the 66 limbs subjected to primary exploration in this study, 58 procedures were performed with the medial approach. Five primary and three revision procedures were performed with the posterior approach. All 16 of the vein graft operations were performed with a medial approach to facilitate the harvesting of the saphenous vein.

In those limbs in which a healthy artery was found to be associated with a type I, II, or III entrapment, the entire medial head of gastrocnemius muscle, both muscular and tendinous portions, were divided in addition to the division of any abnormal muscle slips, heads, and tendinous bands. In the 23 limbs in which investigation and exploration showed only an abnormally large medial head of gastrocnemius muscle ("functional entrapment"), surgery was uniformly performed with the medial approach. After the careful dissection of the popliteal fossa to exclude a type I to IV entrapment, the entire muscular portion of the medial head of the gastrocnemius muscle was divided at the level of the tibial plateaux, followed by the careful mobilization and lysis of the distal half of the popliteal artery. Only the tendon of the medial head of the gastrocnemius muscle was left intact.

DISCUSSION

A greater awareness of popliteal artery entrapment syndrome as a possible diagnosis in young adults who are seen by the sports medicine specialist probably accounts for the high prevalence of this condition seen at our center. Certainly, better investigation and screening of these patients has led to more accurate and frequent diagnosis and treatment of popliteal artery entrapment syndrome.[32] The increasing frequency with which this condition is reported[33,34,38] strongly suggests a greater awareness of the syndrome.[72-76] Better evaluation by sports medicine specialists of the problem of the athlete with calf pain[32,77-79] has improved the diagnostic yield in the young patient with unexplained calf pain. More than half the patients under the age of 50 years who are seen with claudication symptoms of the lower limbs in this and other series were subsequently shown to have popliteal artery entrapment syndrome as a cause of their symptoms.[6,36]

Most of the early reports of popliteal artery entrapment syndrome described patients whose disease had progressed to total occlusion of the artery. The natural history of the popliteal artery with unrelieved compression was thought to be an aggressive one, and on this basis, surgery was advised whenever

the diagnosis was confirmed.[44] The description of progressive fibrosis of the entrapped vessel wall leading to aneurysm formation and thrombosis[9,25,26,32,33] supports this recommendation. A classification of the histologic changes seen with popliteal artery entrapment syndrome has been proposed.[36] This classification is based on the severity and extent of the histologic changes of neovascularization and subsequent progressive fibrosis occurring first in the adventitia (stage 1) and progressing with time to involve the media (stage 2). Thrombosis is thought to result when there is replacement of the intimal region by fibrosis (stage 3), thus rendering the flow surface thrombogenic. Aneurysmal formation, as found in eight cases in this study, implies extensive stage 2 disease.

The implication of such a finding is that the degree of arterial degeneration normally encountered when thrombosis has occurred is so severe that the arterial wall cannot be salvaged. This would explain the poor medium term patency results obtained after popliteal artery occlusion treated with a lesser procedure, such as thrombolysis, angioplasty, or thrombectomy with patching, as seen in one of our early cases. On the other hand, we and others report excellent long-term patency after aneurysm repair or occlusion treated with saphenous vein grafting. This argues strongly in favor of the complete replacement of the popliteal artery, preferably with saphenous vein, when significant degeneration of the artery is noted with preoperative angiography or if thickening, irregularity, or early aneurysm formation is noted at the time of operation.

We have tended to use the medial approach for most patients with popliteal artery entrapment who have required operation. Although the anatomy is less well demonstrated with the medial approach, these young athletic individuals appear to recover and return to normal sporting activities more rapidly after a medial approach. It is admitted that the posterior approach does provide superior visualization of the popliteal fossa anatomy, and we have used this approach for all revision operations.

Of interest in the present series, only two patients had severe limb-threatening critical ischemia develop at the time of occlusion of the popliteal artery. In each instance, this was shown to be associated with a hypercoaguable state. The other patients with normal distal vessels did not have critical ischemia develop, which suggests that in these 16 patients the occlusion occurred slowly and permitted satisfactory development of collateral vessels. With patent and normal distal vessels beyond the localized occlusion, these

patients had limiting and often severe claudication symptoms develop after popliteal artery occlusion but did not have critical ischemia.

Although our data show no significant difference between the ages of those patients with popliteal artery occlusion and of those in whom a myotomy only was required, the youngest patients in the series were in most cases athletes with type I or II entrapments or with tight localized tendinous bands of the type III and IV entrapments who had undergone treatment for popliteal artery occlusion. The patients with occlusion at an older age invariably had muscular entrapment mechanisms of the type III or type VI. This finding suggests that the rate of arterial wall degeneration in popliteal artery entrapment syndrome may depend on the degree of compression and the magnitude of the forces exerted on the popliteal artery by the compression mechanism.

Most cases of type I and II entrapment are easy to diagnose with angiography and other methods of imaging. In addition, the more localized types of entrapment seen with type III and IV are, in our experience, frequently distinguishable from the more diffuse narrowing of the artery found at angiography with the "functional" or type VI entrapment, as shown in Fig 4. On the basis of these observations, we would strongly support the advice to offer surgical correction in all cases of type I to IV at the time of diagnosis[44] and not to wait until arterial degeneration has supervened.

The demonstration that the popliteal artery will undergo some transitory compression or even temporary occlusion with extremes of plantar flexion or dorsiflexion in up to half of the normal population cannot be ignored. The simple demonstration of popliteal artery compression with such stress positions cannot justify operation in patients with otherwise normal anatomy and minor or no symptoms.[45,50,80] On the other hand, we have in the present series documented three popliteal arteries that have undergone occlusion in two patients with a "functional" or type VI symptomatic entrapment and normal anatomy apart from a large muscular medial head of gastrocnemius muscle. The demonstration that a functional popliteal artery entrapment syndrome may progress to occlusion with the histologic picture of chronic compression[36] and with the degeneration not as a result of atheroma justifies a more aggressive surgical approach to symptomatic patients who are shown to have a type VI entrapment. Until further research elucidates the clinical significance and natural history of degeneration of the popliteal artery in the functional type of entrap-

ment in both the asymptomatic and symptomatic patient, the correct management of this condition must remain controversial.

Finally, we believe that the various manifestations and types of popliteal artery entrapment syndrome are much more prevalent than originally appreciated. This diagnosis should be considered in any patient under the age of 50 years with typical calf and foot claudication symptoms on exercise, particularly if the symptoms occur in an athletic individual and if the normal risk factors for atheroma are absent. The finding of an isolated popliteal artery aneurysm or isolated popliteal artery occlusion in the young physically active individual without evidence of systemic atherosclerotic disease should be considered to be caused by popliteal artery entrapment syndrome, unless proven otherwise.

The evidence suggests that all patients in whom the type I to IV entrapment is diagnosed before occlusion of the artery should undergo surgical release of the entrapment mechanism before deterioration of the popliteal artery by repetitive compression. On the other hand, only patients with significant and typical symptoms should be offered surgical treatment for the "functional" or type VI popliteal artery entrapment. Once the popliteal artery has undergone occlusion, the evidence suggests that the artery is beyond repair and it is recommended that the artery be replaced, preferably with saphenous vein graft, to ensure optimum long-term popliteal artery patency in these often young and physically active individuals.

REFERENCES

1. Stuart TP. Note on a variation in the course of the popliteal artery. J Anat Physiol 1879;13:162-5.
2. Hamming JJ. Intermittent claudication at an early age, due to an anomalous course of the popliteal artery. Angiology 1959;10:369-70.
3. Gedge SW, Spittel JA Jr, Ivins JC. Aneurysm of the distal popliteal artery in its relationship to the arcuate popliteal ligament. Circulation 1961;24:270-3.
4. Carter AE, Eban RA. A case of bilateral developmental abnormality of the popliteal arteries and gastrocnemius muscles. Br J Surg 1964;51:518-22.
5. Turner GR, Gosney WG, Ellingson W, et al. Popliteal artery entrapment syndrome. JAMA 1964;208:692-3.
6. Hamming JJ, Vink U. Obstruction of the popliteal artery at an early age. J Cardiovasc Surg 1965;6:516-24.
7. Love JW, Whelan TJ. Popliteal artery entrapment syndrome. Am J Surg 1965;109:620-4.
8. Rich NM, Hughes CW. Popliteal artery and vein entrapment. Am J Surg 1967;113:696-8.
9. Abbott WM, Darling RC. Axillary artery aneurysm secondary to crutch trauma. Am J Surg 1973;125:515-20.
10. Albertazzi VJ, Elliot TE, Kennedy JA. Popliteal artery entrapment. Angiology 1969;20:119-28.

11. Husni EA, Ryu CK. Entrapment of the popliteal artery and its management. Angiology 1971;22:380-6.
12. Ezzet F, Yettiz M. Bilateral popliteal artery entrapment: case report and observations. Cardiovasc Surg 1971;12:71-4.
13. Brightmere JGJ, Smellie WAB. Popliteal artery entrapment. Br J Surg 1971;58:481-5.
14. Delaney TA, Gonzalez LL. Occlusion of the popliteal artery due to muscular entrapment. Surgery 1971;69:97-101.
15. Harris JD, Jepson RP. Entrapment of the popliteal artery. Surgery 1971;69:246-50.
16. Edmondson HT, Crowe JA. Popliteal arterial and venous entrapment. Am Surg 1972;38:657-9.
17. Chavatzas D, Barabas A, Martin P. Popliteal artery entrapment. Lancet 1973;2:181-2.
18. Gaylis H, Rosenberg B. The popliteal artery entrapment syndrome—a bilateral case. S Afr J Surg 1973;11:51-4.
19. Gallagher EG, Hudson TL. Popliteal artery entrapment. Am J Surg 1974;128:88-90.
20. Servello M. Clinical syndrome of anomalous position of the popliteal artery. Circulation 1962;26:885-90.
21. Biemans RGH, Van Bockel JH. Popliteal artery entrapment syndrome. Surg Gynaecol Obstet 1977;144:604-9.
22. Gerkin T, Beebe HG, Williams DM, Bloom JR, Wakefield TW. Popliteal vein entrapment presenting as deep venous thrombosis and chronic venous insufficiency. J Vasc Surg 1993;18:760-6.
23. Gibson MHL, Mills JG, Johnson GE, Downs AR. Popliteal entrapment syndrome. Ann Surg 1977;185:341-8.
24. Turner EH, Grove JA. Popliteal arterial and venous entrapment. Am Surg 1972;38:657-9.
25. Darling RC, Buckley CJ, Abbot WM, Raines JK. Intermittent claudication in young athletes: popliteal artery entrapment syndrome. J Trauma 1974;14:543-52.
26. Rich NM, Collins GJ, McDonald PT, Kozloff L, Clagett GP, Collins JT. Popliteal vascular entrapment—its increasing interest. Arch Surg 1979;114:1377-84.
27. Collins PS, McDonald PT, Lim RC. Popliteal artery entrapment: an evolving syndrome. J Vasc Surg 1989;10:484-90.
28. di Marzo L, Cavallaro A, Sciacca V, et al. Surgical treatment of popliteal artery entrapment syndrome: a ten year experience. Eur J Vasc Surg 1991;5:59-64.
29. Soyka P, Dunart JH. Popliteal artery entrapment syndrome: familial occurrence. Vasa 1993;22:178-81.
30. Insua JA, Houng JR, Humphries AW. Popliteal artery entrapment syndrome. Arch Surg 1970;101:771-5.
31. Cummings RJ, Webb HW, Lovell WW, Kay D. The popliteal artery entrapment syndrome in children. J Pediatr Orthop 1992;12:539-41.
32. Clanton TO, Solcher BW. Chronic leg pain in the athlete. Clin Sports Med 1994;13:743-59.
33. di Marzo L, Cavallaro A, Sciacca V, Mingoli A, Stipa S. Natural history of entrapment of the popliteal artery. J Am Coll Surg 1994;178:553-6.
34. Bouhoutsos J, Daskalakis E. Muscular abnormalities affecting the popliteal vessels. Br J Surg 1981;68:501-6.
35. Ikeda M, Iwase T, Ashida K, Tarkawa H. Popliteal artery entrapment syndrome—report of a case and study of 18 cases in Japan. Am J Surg 1981;141:726-30.
36. Levien LJ. Popliteal artery thrombosis caused by popliteal entrapment syndrome. In: Greenhalgh RM, Powell JT, editors. Inflammatory and thrombotic problems in vascular surgery. London: W. B. Saunders Co Ltd; 1997. p. 159-68.
37. Ferro R, Barile C, Bretto P, Buzzachino A, Ponsio F. Popliteal artery entrapment syndrome: report on seven cases. J Cardiovasc Surg 1980;21:45-52.
38. Persky JM, Kempczinski RF, Fowl RJ. Entrapment of the popliteal artery. Surg Gynecol Obstet 1991;173:84-90.
39. Murray A, Halliday M, Croft RJ. Popliteal artery entrapment syndrome. Br J Surg 1991;78:1414-9.
40. McDonald PT, Easterbrook JA, Rich NM, Collins GJ, Kozloff L, Clagett GP, et al. Popliteal artery entrapment syndrome. Clinical, noninvasive and angiographic diagnosis. Am J Surg 1980;139:318-25.
41. Rich NM. Popliteal entrapment and adventitial cystic diseases. Surg Clin North Am 1982;6:449-65.
42. Schurmann G, Mattfeldt T. The popliteal artery entrapment syndrome. Eur J Vasc Surg 1990;4:223-31.
43. Rudo WD, Noble HB, Conn JJ, et al. Popliteal artery entrapment syndrome in athletes. Physician Sports Med 1982;10:105-14.
44. Fowl RJ, Kempczinski RF, Whelan TJ. Popliteal artery entrapment. In: Rutherford RB, editor. Vascular surgery. 4th ed. Philadelphia: W. B. Saunders Co; 1995. p. 889-94.
45. Chernoff DM, Walker AT, Khorasani R, et al. Asymptomatic functional popliteal entrapment: demonstration at MR imaging. Radiology 1995;195:176-80.
46. Rignault DP, Pailler JL, Lunel F. The "functional" popliteal artery entrapment syndrome. Int Angiol 1985;4:341-3.
47. Turnipseed WD, Pozniak M. Popliteal entrapment as a result of neurovascular compression by the soleus and plantaris muscles. J Vasc Surg 1992;15:285-94.
48. Di Cesare E, Marsili L, Marino E, et al. Stress MR imaging for evaluation of popliteal artery entrapment. J Magn Reson Imaging 1994;4:617-22.
49. Akkersdijk WL, de Ruyter JW, Lapham R, Mali W, Eikelboom BC. Colour duplex ultrasonographic and provocation of popliteal artery compression. Euro J Vasc Endovasc Surg 1995;10:342-5.
50. Erdoes LS, Devine JJ, Berhard VM, Baker MR, Berman SS, Hunter GC. Popliteal vascular compression in a normal population. J Vasc Surg 1994;20:978-86.
51. Colborn GL, Lumsden AB, Taylor BS, Skandalakis JE. The surgical anatomy of the popliteal artery. Am Surg 1994;60:238-46.
52. Bardeen CR. Development and variation of the nerves and the musculature of the inferior extremity and of the neighbouring regions of the trunk in man. Am J Anat 1907;6:259-390.
53. Senior HD. The development of the arteries of the human lower extremities. Am J Anat 1919;25:55-95.
54. Senior HD. The development of the human femoral artery, a correction. Am J Anat 1920;17:271-9.
55. Haimovici H, Sprayregen S, Johnson F. Popliteal artery entrapment by fibrous band. Surgery 1972;72:789-92.
56. Leon M, Volteas N, Labropoulas N, et al. Popliteal vein entrapment in the normal population. Eur J Vasc Surg 1992;6:623-7.
57. Baker WH, Stoney RJ. Acquired popliteal entrapment syndrome. Arch Surg 1972;105:780-2.
58. Carpenter JP, Lieberman MD, Shlansky-Goldberg R, Braverman SE, Soulen M, Holland GA, et al. Infrageniculate bypass graft entrapment. J Vasc Surg 1993;18:81-9.
59. Fong H, Downs AR. Popliteal artery entrapment syndrome with digital embolisation—a report of two cases. J Cardiovasc Surg 1989;30:85-8.
60. Haddad M, Barral X, et al. The embolic type of popliteal entrapment syndrome. Vasa 1990;19(1):63-7.

61. Miles S, Roediger W, Cooke P, Mieny CJ. Doppler ultrasound in the diagnosis of the popliteal artery entrapment syndrome. Br J Surg 1977;64:883-4.

62. Jeffery PG, Immelmenn EJ, Harries-Jones P. Popliteal artery entry syndrome: a report of two cases. S Afr Med J 1985;67:692-4.

63. MacSweeny STR, Cumming R, Greenhalgh RM. Colour doppler ultrasonographic imaging in the diagnosis of popliteal artery entrapment syndrome. Br J Surg 1994;81:822-3.

64. Allen MJ, Barnes MR, Bell PR, Bolia A, Hartshorne TC. Popliteal artery entrapment syndrome. Eur J Vasc Surg 1993;7:342-5.

65. di Marzo L, Cavallaro A, Sciacca V, Lepidi S, Marmorale A, Tamburelli A, et al. Diagnosis of popliteal artery entrapment syndrome: the role of duplex scanning. J Vasc Surg 1991;13:434-8.

66. Rizzo RJ, Flinn WR, Yao JST, McCarthy WJ, Vogelzang RL, Pearce WH. Computed tomography for evaluation of arterial disease in the popliteal fossa. J Vasc Surg 1990;11:112-9.

67. Muller J, Morris DC, Nichols DM. Popliteal artery entrapment demonstrated by CT. Radiology 1984;151:157-8.

68. Williams LR, Flinn WR, McCarthy WJ, Yao JST, Bergan JJ. Popliteal artery entrapment: diagnosis by computed tomography. J Vasc Surg 1986;3:360-3.

69. Fujiwara H, Sugano T, Fujii N. Popliteal artery entrapment syndrome: accurate morphological diagnosis utilizing MRI. J Cardiovasc Surg 1992;33:160-2.

70. McGuinnes G, Durham JD, Rutherford RB, Thickham D, Kumpe DA. Popliteal artery entrapment: findings at MR imaging. J Vasc Interv Radiol 1991;2:241-5.

71. Greenwood LH, Yrizanny JM, Hallett JW. Popliteal artery entrapment: importance of the stress runoff for diagnosis. J Cardiovasc Interv Radiol 1986;9:93-9.

72. Bouhoutsos J, Goulios A. Popliteal artery entrapment: report of a case. J Cardiovasc Surg 1977;18:481-4.

73. Cairols MA, Blanes I, Gimenez A, Miralles M, Sieyro F, Latorre E. An exceptional case of popliteal entrapment syndrome. Eur J Vasc Surg 1994;8:754-6.

74. Duwelius PJ, Kelbel JM, Jardon OM, et al. Popliteal artery entrapment in a high school athlete: a case report. Am J Sports Med 1987;15:371-3.

75. Inada K, Kirose M, Iwashima Y, et al. Popliteal artery entrapment syndrome: a case report. Br J Surg 1978;65:613-5.

76. Mark LK, Kiselow MC, Wagner M, Goodman JJ. Popliteal artery entrapment syndrome. JAMA 1978;240:465-6.

77. Iwai T, Konno S, Soga K, Hatano R, Yamada T, Menjo M. Diagnostic and pathological considerations in the popliteal artery entrapment syndrome. J Cardiovasc Surg 1983;24:243-9.

78. Lysens RJ, Rensen LM, Ostyn MS, et al. Intermittent claudication in young athletes: popliteal artery entrapment syndrome. Am J Sports Med 1983;11:177-9.

79. Turnipseed W, Detmer DE, Gridley F. Chronic compartment syndrome. Am J Surg 1989;210:557-63.

80. Verhoeven ELG, Lucarotti ME, Campbell WB. Vanishing popliteal entrapment. Eur J Vasc Endovasc Surg 1995;9:244-6.