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A current interpretation of popliteal vascular entrapment

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Functional popliteal artery entrapment is differentiated from anatomical entrapment by the absence of abnormal popliteal fossa anatomy. Although functional compression is a common entity in the general population, the precise etiology and natural history remains unknown. Magnetic resonance imaging clearly defines muscular variations within the popliteal fossa. In light of some of these variations, this article reviews embryological anatomy, diagnosis, classification, and treatment of the popliteal entrapment syndrome.

Abnormal anatomy of structures in the popliteal fossa may result in anatomic (embryological) entrapment of the popliteal artery, and less frequently of the vein. Patients with normal popliteal fossa anatomy, however, may also present with entrapment of the popliteal vessels, and the term functional entrapment has been introduced.

Anatomic popliteal entrapment

In anatomic popliteal entrapment, muscular or tendinous aberrations are responsible for compression and injury of neurovascular structures and the subsequent clinical entity (the anatomic entrapment syndrome).[1, 2, 3, 4 and 5] The embryological development of the popliteal fossa suggests that there is a “competition for space” between the primitive neurovascular bundle and various migrating muscles.[6, 7 and 8] The commonest anomalies involve the incomplete or delayed migration of the medial head of the gastrocnemius muscle (MHGM). The resulting abnormal lateral position of the MHGM may cause displacement of the popliteal artery.[4, 9, 10 and 11] Contraction of this bulky, powerful muscle within a confined space bordered by strong fascia and solid bone results in forceful compression of the adjacent neurovascular bundle.

The current classification of embryological entrapment (types I to V) includes abnormal development of the MHGM (types I and II); abnormal fibrous, muscular, or tendinous bands usually derived from remnants of the MHGM (type III), and a primitive position of the distal popliteal artery posterior to the popliteus (type IV; Fig 1)[4, 5, 9, 10 and 11] Popliteal

vein entrapment with any of the above anomalies is referred to as type V. This complex embryological picture, suggests that there may be subtle muscular variations of the known anomalies or other as yet unidentified rare malpositions.[5 and 12]

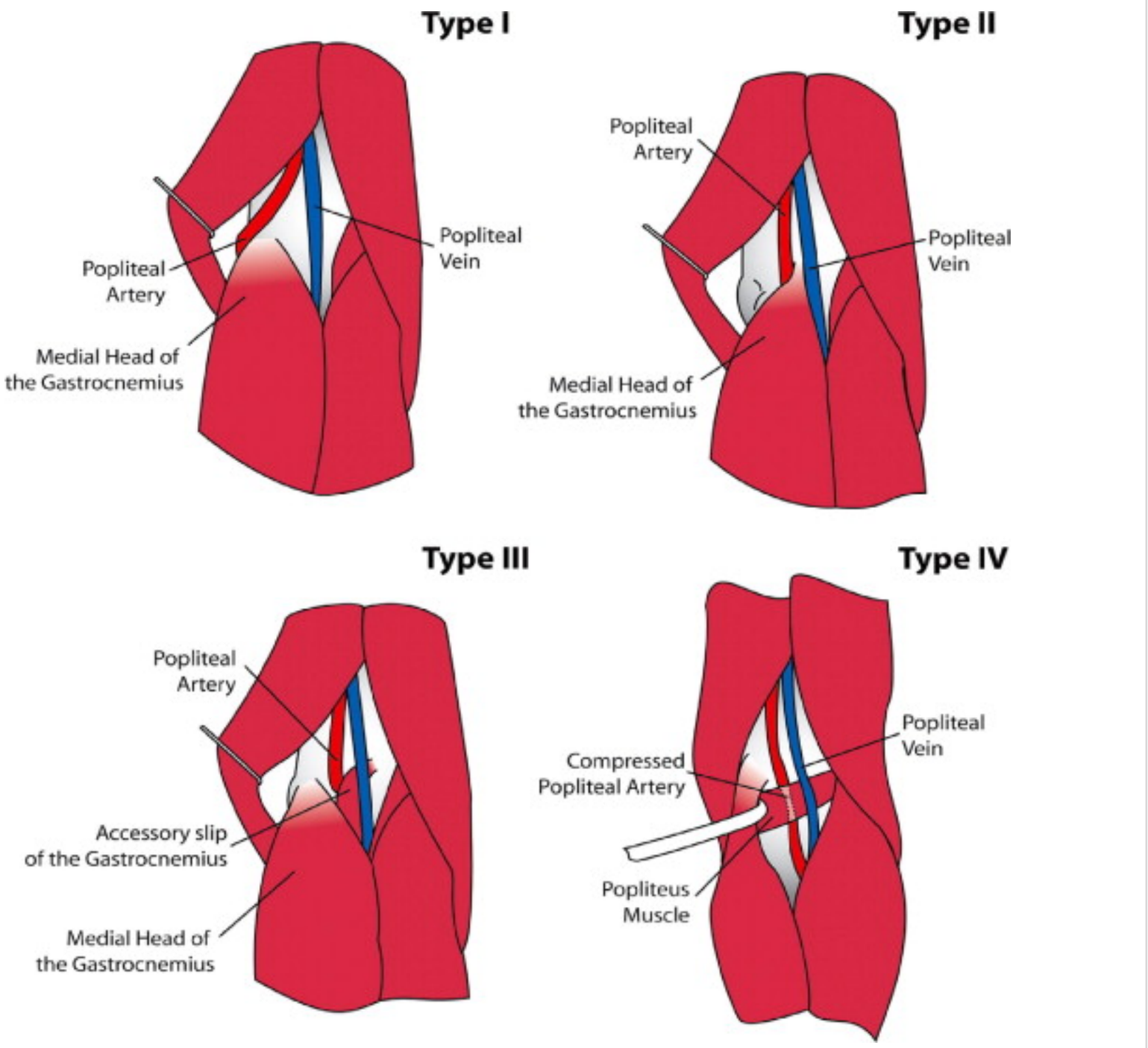

Fig 1.
Embryological popliteal entrapment, types I to IV.

Figure options

Functional popliteal entrapment

In functional popliteal compression, the popliteal fossa anatomy appears normal, and yet the popliteal arteries in a subset of patients will compress during leg movements. Rarely this compression may injure the artery, leading to symptoms (functional entrapment syndrome), which are frequently relieved by exploration of the popliteal fossa.[7, 10, 13 and 14] Early reports hypothesized that muscular hypertrophy within the popliteal fossa was responsible.[1, 10, 11 and 13] However, symptomatic popliteal artery occlusion has been reported in young untrained individuals, and some degree of popliteal arteries compression with forceful foot movements (functional compression) may occur in 30% to 50% of the general population.

Magnetic resonance imaging (MRI) suggests that compression occurs at the level of the soleal sling, where the soleus, gastrocnemius, popliteus, and plantaris muscles seem to be involved.[13] There is no clear explanation for why compression occurs at this level. Individuals who exercise regularly, especially those who are highly trained, will invariably form a large subset of all those who present with the entrapment syndrome.[2, 4, 8, 10, 13 and 14] In those at risk, repetitive and forceful movement of a hypertrophied muscle is more likely to compress and injure the adjacent artery, resulting in claudication. Hypertrophy has been implicated to some extent by biased sampling, and its precise role remains uncertain.[1, 13 and 14]

Evaluation with MRI

In a recent study we compared the MRI-visualized attachment of the MHGM with the

posterior surface of the femur in healthy untrained “occluders” and “nonoccluders.”[15] Subjects were identified as occluders if their popliteal arteries occluded with forceful plantar flexion (ie, functional compression). Our results indicated that in the occluders, there was more extensive MHGM attachment towards the midline of the bone just above the medial condyle as well as more extensive attachment within the intercondylar notch (Fig 2, Fig 3, Fig 4 and Fig 5).

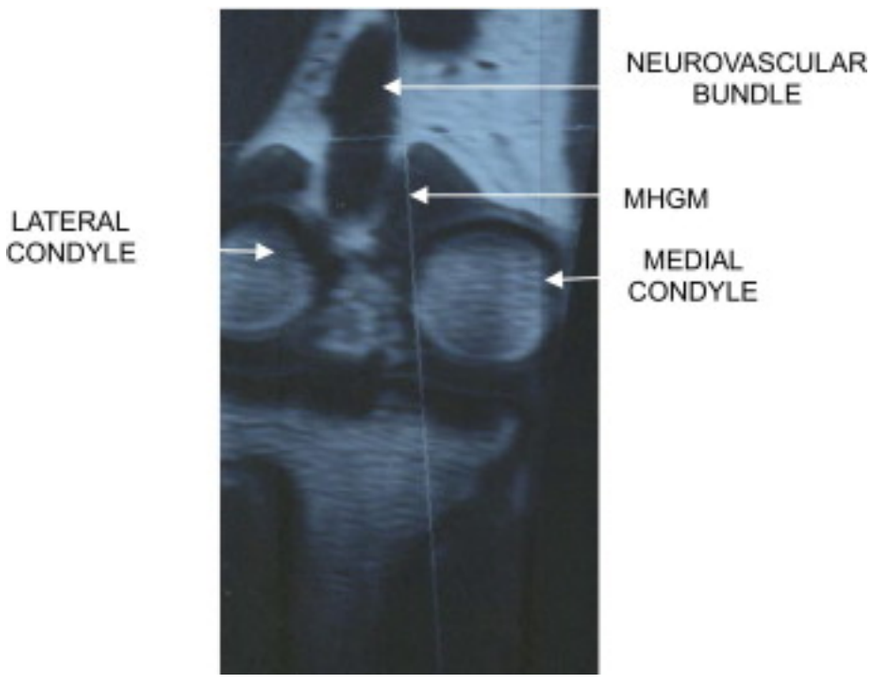

Fig 2.
Coronal scans of the right medial condyle in a nonoccluder. Eighteen percent of the inner circumference of the medial condyle has fibers attached from medial head of the gastrocnemius muscle *(MHGM)*.

Figure options

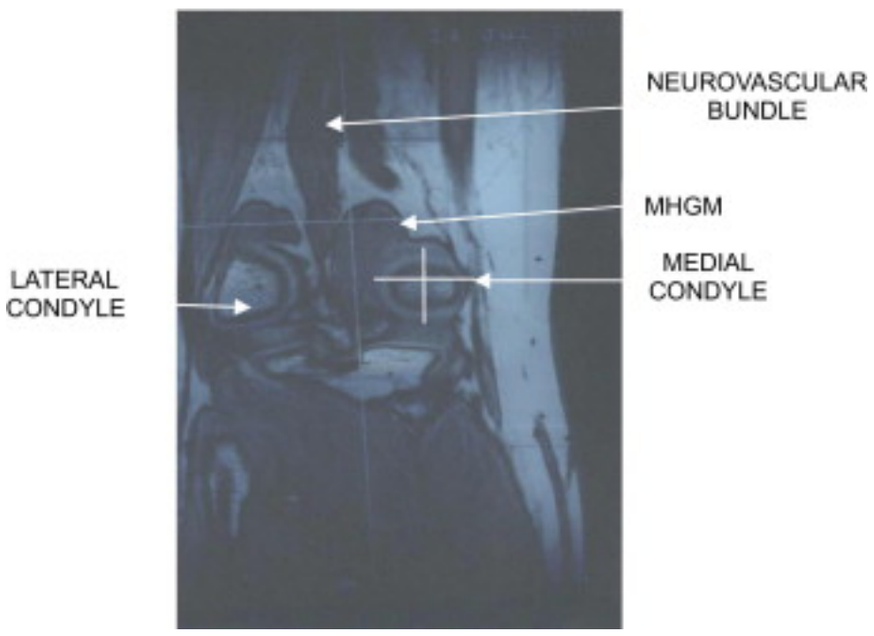

Fig 3.
Coronal scans of the right medial condyle in an occluder. Forty-five percent of the inner circumference of the medial condyle has medial head of the gastrocnemius muscle *(MHGM)* attached to it. The MHGM encroaches the neurovascular bundle within the intercondylar.

Figure options

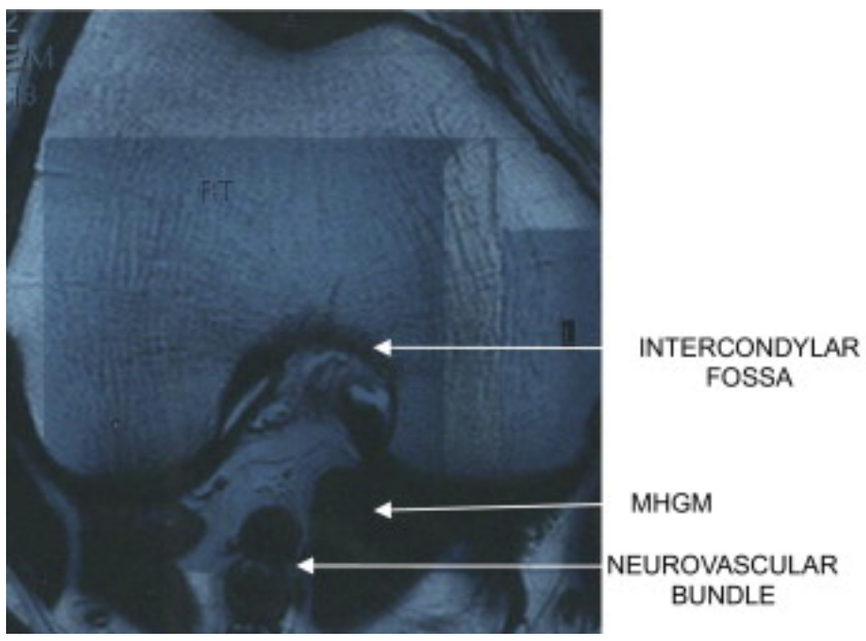

Fig 4.
Axial scans of the right intercondylar notch in a nonoccluder. Twenty percent attachment of the medial head of the gastrocnemius muscle *(MHGM)* fibers within the notch.

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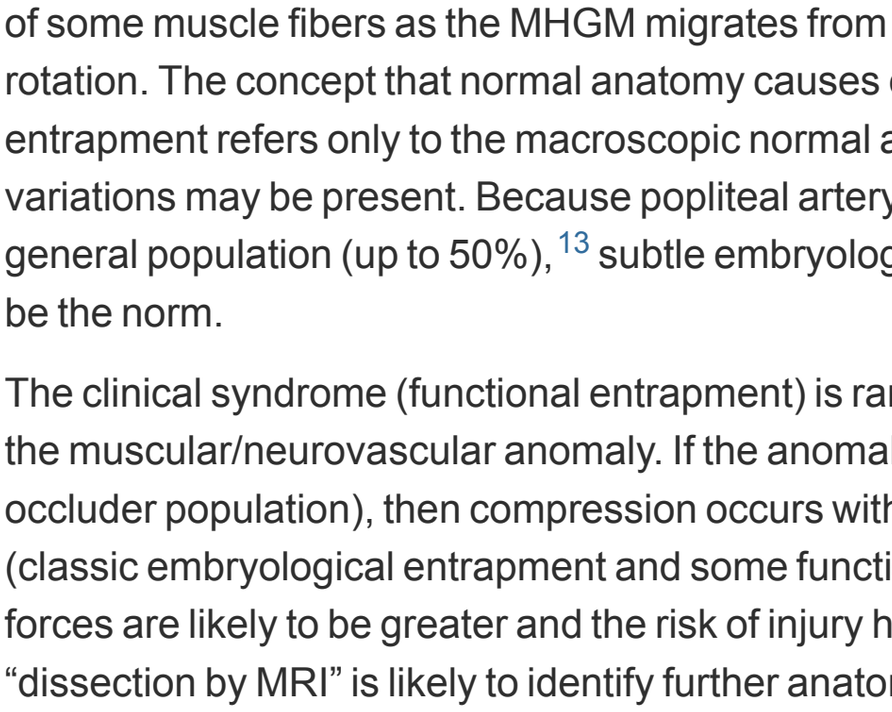

Fig 5.
Axial scans of the right intercondylar notch in an occluder. Fifty percent attachment of medial head of the gastrocnemius muscle *(MHGM)* in the notch.

Figure options

The occluders appeared to have greater muscle bulk adjacent to the neurovascular bundle. These anatomic variations are likely to be due to “embryological lagging behind” of some muscle fibers as the MHGM migrates from lateral to medial during limb bud rotation. The concept that normal anatomy causes either functional compression or entrapment refers only to the macroscopic normal anatomy, but subtle MRI-detected variations may be present. Because popliteal artery compression is common in the general population (up to 50%),[13] subtle embryological variations detected by MRI may be the norm.

The clinical syndrome (functional entrapment) is rare and may be related to the degree of the muscular/neurovascular anomaly. If the anomaly is not extreme (most of the normal occluder population), then compression occurs with little risk of injury, and if extreme (classic embryological entrapment and some functional occluders), then entrapment forces are likely to be greater and the risk of injury higher. In the future, popliteal fossa “dissection by MRI” is likely to identify further anatomic variations (explained by embryologic differences) and separation into functional/anatomic entities may become less important.

Clinical presentation

A common clinical picture is that of a young athletic individual presenting with calf or foot claudication.[7, 8, 11, 13 and 14] Intense physical activity may precipitate other symptoms such as coldness, paraesthesia, or numbness.[7 and 8] More severe symptoms are rare, but rest pain and tissue loss have been described.[1, 2, 7 and 8]

Arterial wall degeneration depends on the degree of compression, the magnitude of forces exerted on the popliteal artery, and the time during which this occurs. In turn, the severity of the anatomic abnormality, muscle hypertrophy, and the type of physical activity also need to be considered. It is probable that the severity of the muscular abnormality is the most important variable. Severe anatomic abnormalities (anatomic occluders with obvious embryological abnormality) may provide greater compressive forces that increase the rate of arterial injury, and these individuals are likely to present with symptoms at a young age.

Conversely, the rate of arterial injury may be lower in functional occluders, and it is possible that arterial degeneration may only manifest at a much later age. For example, atherosclerotic risk factors also promote arterial injury, inflammation, and degeneration. Is it possible that functional compression, which is reported in up to 50% of the general population, represents an additional risk factor? Encouraging exercise therapy in

individuals with claudication with predominant popliteal stenoses thus becomes a moot point. Similarly, evaluating functional popliteal neurovascular compression as a risk in chronic venous hypertension, acute deep venous thrombosis, and ill-defined nerve symptoms needs further assessment.

Diagnostic evaluations

Current diagnostic modalities include handheld Doppler examination, duplex ultrasound scanning, MRI and MR angiography (MRA), and catheter-directed contrast angiography.[4, 7, 10, 13 and 15] Each test may be performed at rest or with the foot being forcefully plantar flexed.[7, 8 and 15]

We have found certain limitations with provocative testing. When posterior tibial pulses are assessed during forceful plantar flexion, a change occurs in the anatomic relationship between the artery and muscle at the ankle. Slight displacement of the handheld probe results in loss of signal and gives the impression of arterial occlusion. Similarly during ultrasound evaluation of the popliteal fossa, muscle movement may obscure, but not totally compress, the adjacent artery. Velocity changes are therefore difficult to interpret. To increase the diagnostic accuracy, measurements have to be repeated, with realignment of the transducer probe together with multiple, slow, deliberate movements of the foot against resistance.[15]

Ankle-brachial indices (ABI) with forceful plantar flexion are also difficult to interpret. The ABIs vary depending on the degree of contraction, the size of the calf muscle, size of the cuff, and position of the calf in relation to the muscle bulk.[15] ABIs are therefore only useful where the artery is completely occluded and may be less helpful when assessing graded compression of a patent artery, such as when screening populations.

MRI is the current gold standard to identify popliteal fossa anatomy at rest.[13, 15 and 16] Reports suggest that the suspected level of arterial occlusion with forceful plantar flexion may also be determined by MRI.[13] Considerable straining is required to create forceful planter flexion. It is difficult for some individuals to maintain forceful contraction for the duration of the imaging. Significant movement artifacts limit the accuracy during diagnostic testing. Similarly, when a specific muscular abnormality impinges on or close to the artery, movement-related artifacts prevent identification of the precise point of arterial occlusion. In this regard, it is hoped that evolving MR techniques will help to establish the syndrome of subtle anatomic variations on arterial compression. The anatomically variant MHGM that has been identified in asymptomatic occluders may form an important control to which variants from symptomatic occluders can be compared. Catheter-directed angiography is therefore the current gold standard to identify arterial occlusion during plantar flexion.[7]

Treatment

Therapy for popliteal entrapment syndromes depends on the extent of arterial injury, the severity of the muscular anomaly, the dynamic relationship between contracting muscles and the adjacent artery, the clinical presentation, and the level of physical activity.

When the artery is occluded, symptoms are likely to be severe and the muscle anomaly extreme. In these cases, MRI (and MRA) confirms the diagnosis and indicates the need for muscle detachment during saphenous vein bypass.[7, 13 and 16] In patients with critical ischemia and young active individuals with claudication who wish to persist with a lifestyle that involves sports, reverse saphenous vein bypass and release of the entrapment mechanism is the preferred treatment.[7 and 8] The perceived entrapment mechanism is carefully identified by palpation along the neurovascular bundle and released by performing a myotomy of the MHGM or excising an abnormal muscular or tendinous band.[7] The newly created tunnel through which the reversed saphenous vein graft passes is thus widened.

In symptomatic individuals with normal distal pulses, MRI and catheter-directed contrast angiography at rest and after forceful plantar flexion will demonstrate anatomy and the

dynamic relationship between muscle and artery.[7, 8 and 15] The natural history of popliteal artery compression appears to be an aggressive one. Gross embryological abnormalities tend to cause progressive degeneration and destruction of the vessel wall.[7] Exploration of the popliteal fossa is therefore recommended in types I to V entrapment. Myotomy of the MHGM and excising abnormal tendinous bands is all that is required if the artery appears normal.[7, 8 and 14]

In symptomatic individuals with macroscopic normal anatomy (functional entrapment), the natural history of the compressed popliteal artery remains uncertain, but occlusion and degeneration have been described.[7 and 8] Surgery is therefore recommended in functional occluders with significant repetitive and typical symptoms.[7 and 8] The concept of functional entrapment remains controversial because the etiology and precise entrapment site is unknown. Symptoms associated with functional entrapment are nonspecific and include claudication and paraesthesia. As a result, neurogenic compression has been suggested.[7]

A variety of operative procedures have been described to relieve symptoms. These include MHGM myotomy, fasciotomy, excision of the plantaris muscle, release of the soleal sling, and excision of the popliteal muscle.[4, 5, 7, 8 and 11] Symptoms have been relieved in most individuals even though intraoperative anatomic abnormalities have not been found. Popliteal fossa “decompression” and extreme fasciotomy are probably the most important common components of surgery. Subtle anatomic or embryologic muscular abnormalities, muscle hypertrophy, repetitive or forceful activity, compartment syndrome, and neurovascular irritation or entrapment are all likely variables in functional compression/entrapment.

Results of popliteal fossa exploration, bypass, or muscle detachment, or a combination of these, and fossa decompression generally appear to be good. Most series report a small number of patients, but >90% appear to return to activities in sports ≤3 months with resolution of all previous symptoms.[2, 7 and 10] Most saphenous vein bypass grafts have been reported to be patent at 5 years.[7]

The manifestations and types of popliteal artery compression range from mild to severe. Further experience with MRI is likely to differentiate these types, based on anatomic and embryological variations. An awareness of the more severe anomalies, particularly in the young athlete, appears to be mandatory if good clinical outcomes are to be achieved.

Conclusions

Embryological anatomic variations are probably responsible for both functional and anatomic entrapment. A spectrum of anomalies from mild (macroscopically normal anatomy) to severe (macroscopically abnormal anatomy) is likely to exist. Evidence suggests that severe anomalies cause an aggressive and early degeneration of the popliteal artery. Compression of the popliteal artery with plantar flexion is common in the general population. This is probably due to a mild embryologic anomaly. The hypothesis that these mild anomalies may exponentially injure the neurovascular bundle with time cannot be ignored. MRI-based research involving symptomatic occluders and older individuals with popliteal artery stenoses, venous hypertension, and paraesthesia is warranted.