INTERMITTENT CLAUDICATION

A Clinical Study

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Lameness in horses during exercise was recognised by veterinary surgeons in France and Germany about the middle of the nineteenth century and it was assumed that in some cases this was due to occlusion of the main arteries of the limb. In 1831, the syndrome was named "intermittent claudication" by the veterinarian Bouley. In 1846, Benjamin Brodie described a similar syndrome in a man "whose femoral artery was converted into a gristly cord, so as to be quite impervious from the origin of the profunda to the point at which it perforates the tendon of the great head of the triceps adductor muscle." He realised that lameness was due to the insufficiency of a reduced arterial supply for the added demands of muscle exercise, and claimed that the condition was to be found principally in "those who drink too much fermented liquor and do not take sufficient exercise" and "especially those who are overfed with animal food."

The description by Charcot (1858) of a patient with an aneurism of the common iliac artery was the first accurate and comprehensive survey of the clinical picture. Charcot (1887) and Marinesco (1896) added further clinico-pathological studies. Erb (1898) collected many examples; he reviewed the literature and emphasized that, when there was arterial disease, lameness or limping during exercise was due to pain and not to muscle weakness. Since that time many excellent studies of the symptomatology and etiology have been published.

THEORIES OF THE MECHANISM OF THE PAIN-SYNDROME IN VASCULAR DISEASE

1) Muscle spasm—Earlier clinicians were much impressed by the observation in patients with intermittent claudication that occasionally, after exercise, there was palpable change in the consistency of the calf muscles. They allowed that the primary cause of pain was arterial deficiency, but thought that the final mechanism initiating the pain was muscle spasm. This spasm was likened by Charcot (1858) and Marinesco (1896) to "cadaveric" rigidity, even although the pain of intermittent claudication is continuous and does not fluctuate with each muscle contraction. Erb (1898) gave support to this view; but it was abandoned by later writers because in the light of further investigations it proved obviously inadequate.

2) Arterial spasm—To estimate the rôle played by arteriospasm in intermittent claudication it must first be ascertained whether or not there is actually spasm in the vessels of the limb at the time that pain is felt. Evidence has been marshalled by various authors.

During exercise the arteriosclerotic foot pales. Pallor precedes the pain and it has been assumed that it is due to arterial spasm of the skin vessels, and that the muscle arteries might behave similarly, thus causing pain (Thomas 1922, Zak 1921). We too have observed this pallor during exercise but it is not always present. Moreover, pallor may be induced in a severely arteriosclerotic limb by briskly rubbing the skin over the tibia, or by passively flexing the ankle joint a few times. The deduction that muscle vessels and skin vessels behave in similar manner is not justified; nor is the assumption that pallor is due to arterial spasm (vide infra).

Lian (cit. Thomas 1929) believed that he had demonstrated spasm in the vessels of the lower limb of a patient with arteriosclerotic claudication by arteriography before and after

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exercise. Similar studies were repeated in greater detail by Veal and McFetridge (1936) and they showed that exercise-pain was in fact accompanied by dilatation of the vessels, especially those of smaller calibre.

After exercise of the arteriosclerotic limb, arterial pulsations at the level of the ankle joint are diminished, or they may disappear, and oscillometric readings taken on the lower leg are correspondingly decreased (Comroe 1923, Leary and Allen 1941). This observation was confirmed in great detail by Ejrup (1948). Both oscillometry and the palpation of vessels measure pulsation of the arterial wall and not flow of blood. A more reasonable explanation than that of arterial spasm is that inflow through the major vessels of the limb is limited by the arteriosclerotic process and that these vessels are unable to dilate normally in response to the demands of exercise. When structural changes are less advanced, the arterio-capillary bed of the muscles increases greatly in capacity and in the course of exercise it abstracts a much larger share of the normal blood supply than when the limb is at rest; and moreover, since the systolic stroke of the major vessels is distributed to a greater bed than before, pulsation in the medium-sized vessels which is measured by oscillometry is decreased.

Claudication has been described in limbs which in the resting state showed no arterial abnormality; yet, on exercise, there was characteristic pain and the pulses disappeared (Pearl 1937). Simmons (1936) stated that: "A small proportion of cases of intermittent claudication are due to pure spasm of the arteries in neuropathic individuals and no structural disease of the vessels is demonstrable either at the time or in later years." Such exceptional cases can be explained more satisfactorily by postulating a thrombotic lesion proximal to the femoral artery. The collateral circulation is good but not sufficient to supply the great needs of exercise. It is noteworthy that the only case of "spastic claudication" described in the literature that has been reviewed at a later date was proved to have organic constriction at the lower part of the aorta. (Lindquist 1948, cit. Ejrup.) Two of the authors of this article (Boyd and Jepson 1949) have seen two cases of thrombosis of the external iliac artery with a similar syndrome.

3) Muscle ischaemia as a cause of pain—In a series of analytical papers, Lewis and his co-workers demonstrated that intermittent claudication was due to inadequacy of blood flow through muscles during exercise causing accumulation in the tissue spaces of a pain factor—"factor P"—which stimulates the sensory nerve endings (Lewis, Pickering and Rothschild 1929; Lewis 1942). Katz (1935) suggested that factor P was a non-volatile acid. Lewis showed by occlusion-plhthysmography that the arterial bed was dilated at the time of the onset of pain.

An extension of Lewis's conception of muscle ischaemia causing pain—In the normal limb it is the muscles that are worked most strenuously in relation to their bulk that first give rise to pain. On the other hand, in limbs suffering from arterial disease, instead of all muscles having a blood supply that is approximately equal in relation to their bulk, the arteriosclerotic process alters the distributive pattern of the arterial supply either by general narrowing of the arteries or by local thrombosis. This redistribution differs from limb to limb according to the anatomical structure and the extent and topography of the arteriosclerotic lesions. It is the arterial inaccessibility of certain parts of the muscles that causes impaired nutrition and accounts for pain on exercise. Pain then arises in that muscle, or part of a muscle, that is worked most strenuously during the complex pattern of walking, and in which the arterial supply relative to the demands of exercise is most deficient. This pain on exercise disappears only when the collateral supply becomes adequate, or when the arteriosclerotic process increases and the affected muscle undergoes atrophy, or is replaced by fibrous tissue so that its potential as a focus of deep pain is destroyed.

In the arteriosclerotic limb, careful palpation of the musculature reveals patches that are tender to firm pressure. This tenderness is, of course, elicited only in superficial muscles and it is noted with particular frequency in the gastrocnemius and sometimes in the small
muscles of the sole of the foot such as the extensor digitorum brevis or in the dorsi-flexors of the foot. It may be obvious only after recent exercise, especially if this has been sufficient to give rise to the pain of claudication. The area of muscle tenderness varies. In limbs that are severely compromised by arterial insufficiency a single muscle or muscle group may be distinctly tender; but in better nourished limbs the patch often measures only a few centimetres in diameter. A site that is often tender is the upper part of the medial belly of gastrocnemius.

The pain suffered on pressure is likened by the patient to the pain of intermittent claudication. The cause of tenderness is not certain but it is probably metabolic in origin and depends upon the same factor that accounts for the pain of exercise. When pain is induced by exercising muscles under ischaemic conditions the accompanying muscle tenderness, and the pain itself, disappear rapidly when the blood flow is re-established although as Lewis (1942) pointed out "a little tenderness may remain for an hour or more if the test has been repeated several times." In arteriosclerotic limbs the involved muscles are continually on

the threshold of ischaemic pain and thus the tenderness, which may never entirely disappear, is always emphasized by exercise. More intelligent patients may notice a patch of deep tenderness while kneading the calf muscles, but usually no such story is elicited because surface representation of the deep pain may be distant from, and not overlying, its place of origin.

Infiltration of the tender area with a few cubic centimetres of 2 per cent. novocain makes it anaesthetic to deep pressure and, by blocking the deep pain trigger-point, it allows the patient to walk painlessly for a distance far exceeding that which was possible before injection. The increase in walking distance depends upon the degree of arteriosclerotic involvement in other muscles of the limb. The new pain that now limits progression is from a different site and, because of its distribution, it is often obvious that it arises from a different muscle. Infiltration of the skin or subcutaneous tissues overlying the tender patch does not alter the exercise-tolerance.

In order to deduce the muscle of origin from the history of surface-representation of pain, further experimental work was undertaken. A reliable method of producing experimental
muscle-pain is that described by Lewis and Kellgren (1939)—namely, the intramuscular injection of 6 per cent. hypertonic saline after infiltration of the skin and deep fascia with local anaesthetic. A few cubic centimetres is sufficient to cause deep aching, varying in intensity from discomfort to nauseating agony. The intensity depends not only on the susceptibility of individuals and the amount of solution injected, but also on the concentration of pain nerve-endings in the vicinity of the injection. These are probably sparse, so that the number of nerve endings that are stimulated varies by chance according to the site of the injected saline. It was our impression nevertheless that in patients with claudication, injection of tender areas, and mechanical stimulation by the needle-point, gave rise more readily to pain than similar procedures in "non-tender" areas and in normal muscles. Each muscle referred to in Figures 1 and 2 was injected in at least six different subjects, usually in both lower limbs. The areas of skin reference for the pain so produced were charted, and composite areas for many of the more important lower limb muscles were determined. The pain comes on almost immediately after the injection; it rises rapidly to maximal intensity; and it then falls gradually in four to six minutes. The quality is described characteristically as a "sickening ache" and is often compared to "cramp." There are ill-defined limits—a small centre of intense pain tending to shade off to mild aching at the periphery. Although limited in spread to the reference areas shown on the charts the pain may be felt far distant from the point of injection, and the site of maximal intensity may move within the area from minute to minute. When hypertonic saline is injected into muscles in the neighbourhood of joints (for example into the flexor hallucis longus or the extensor digitorum brevis near the ankle) the pain may be referred to the joint itself. When a peripheral mixed nerve such as the anterior or posterior tibial is stimulated by adjacent saline, paraesthesia is produced which is distinguished easily from the dull quality of "deep" muscle pain. The whole question of muscle pain is discussed admirably by Kellgren (1938).

CLASSIFICATION OF OBLITERATIVE VASCULAR DISEASE

It seems clear that the immediate cause of intermittent claudication is diminution of arterial supply to the muscles concerned; but the reason why the arterial supply is diminished is less apparent. There is much confusion in the terminology used in relation to obliterator arterial conditions causing circulatory deficiency in the lower limbs. Occlusive arterial disease beginning under the age of fifty years is usually classified as "Buerger's disease" or "thromboangiitis obliterans"; whereas over the age of fifty years the symptoms of arterial deficiency are usually grouped under the general term, arteriosclerosis. Leriche (1947), in a recent analysis of over 500 personal cases, limited the term thromboangiitis obliterans to patients under the age of thirty-five years; in patients over the age of fifty years he attributed the symptoms to arteriosclerosis; but he admitted that there was a third group of patients, with ages between thirty-five and fifty years, showing features of both conditions, that he was unable to classify.

Review of the arteriographic findings and clinical features in a large number of patients with deficiency of circulation in the lower limbs who were investigated between 1932 and 1949 show that they may be classified under three main headings: 1) primary thrombosis of the popliteal artery; 2) juvenile obliterative arteritis; and 3) senile obliterative arteritis—
a) diffuse obliterative arteritis, b) secondary popliteal thrombosis, c) secondary femoral thrombosis (Figs. 3-14).

Primary thrombosis of the popliteal artery and juvenile obliterative arteritis are usually included together under the heading "thromboangiitis obliterans." It seems, however, that in most patients under the age of thirty-five years the cause of claudication is traumatic thrombosis and not arterial disease, and this was certainly true in every case in the series now reported.
Intermittent Claudication

Juvenile obliterative arteritis comprises a group of cases corresponding to those described by Buerger and this condition may be a pathological entity. Buerger himself included under the heading of thromboangiitis obliterans patients who, from their symptoms, appeared to be suffering from degenerative arterial changes. In this report the term "juvenile obliterative arteritis" is restricted to obliterative arteritis beginning in the feet and following a characteristic clinical course. The condition seldom, if ever, begins after the age of thirty-five years.

Senile obliterative arteritis is the term used to include the degenerative arterial changes that are commonly associated with increasing age and are included under the general term "arteriosclerosis." Arteriosclerosis is usually considered to be a disease of old age, but careful examination of post-mortem and biopsy material in obliterative arteritis from patients under the age of thirty-five years sometimes reveals arteriosclerosis at an early age. Well-marked calcification of the vessels has been seen in the early twenties. Arteriosclerosis is undoubtedly the most frequent cause of arterial deficiency in the lower extremities in the fourth decade, and over the age of forty years it accounts for most cases of occlusive vascular disease.

In brief, almost all cases of obliterative arteritis in young people up to the age of about thirty-five years fall into one of the first two groups—primary thrombosis of the popliteal artery and juvenile obliterative arteritis; and in patients over the age of thirty-five years into the third group—senile obliterative arteritis. The nature of the pathological changes in the blocked vessels is largely speculative, but it is of no great importance from the point of view of treatment which cannot be directed towards cure of the underlying condition or restoration of the function of blocked vessels. Attention must be concentrated on the vessels that are still healthy with the object of assisting by every possible means the greatest and most rapid development of a collateral circulation.

### TABLE I

Analysis of 472 Cases of Obliterative Arteritis of the Lower Limbs

<table>
<thead>
<tr>
<th></th>
<th>Men</th>
<th>Women</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Traumatic thrombosis</td>
<td>4</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>Juvenile obliterative arteritis</td>
<td>8</td>
<td>—</td>
<td>8</td>
</tr>
<tr>
<td>Primary popliteal thrombosis</td>
<td>6</td>
<td>—</td>
<td>6</td>
</tr>
<tr>
<td>Senile obliterative arteritis</td>
<td>405</td>
<td>48</td>
<td>453</td>
</tr>
<tr>
<td>(a) Diffuse obliterative arteritis</td>
<td>192</td>
<td>26</td>
<td>218</td>
</tr>
<tr>
<td>(b) Secondary popliteal thrombosis</td>
<td>165</td>
<td>15</td>
<td>180</td>
</tr>
<tr>
<td>(c) Secondary femoral thrombosis</td>
<td>48</td>
<td>7</td>
<td>55</td>
</tr>
</tbody>
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**PRIMARY THROMBOSIS OF THE POPLITEAL ARTERY**

Nearly all patients under the age of thirty-five years who complain of intermittent claudication in the calf muscles have been found to have thrombosis of the popliteal artery. Arteriographic studies show that occlusion is confined to this artery, the other vessels appearing healthy. The extent of the occluded segment varies: it may extend from the level of the knee joint to the adductor opening; from the level of the knee joint to the termination of the artery; or throughout the whole length of the artery from the adductor opening to the bifurcation (Figs. 3–6). The fact that one end of the thrombosed segment is often found at the level of the knee joint suggests strongly that the causative process begins at this level. Several arteriograms obtained before there was actual thrombosis showed the earliest stage
of the lesion, namely, localised narrowing and irregularity of the lumen of the artery in the middle of the popliteal fossa (Fig. 3). Arteriograms of patients known to have sustained traumatic thrombosis of the popliteal artery from posterior dislocation of the knee joint (Fig. 7) are indistinguishable from primary thrombosis of the artery.

Histological examination of the occluded segment obtained by arterectomy from young men suffering from intermittent claudication of the calf muscles, usually thought to be due to "Buerger's disease," shows no evidence of inflammatory changes in the arterial wall (Boyd 1938). The lumen is found to be occluded by healthy clot in various stages of

Fig. 3
Primary popliteal thrombosis - pre-thrombotic lesion at the level of the upper border of the femoral condyles (from the Surgical Professorial Unit at St Bartholomew's Hospital by courtesy of Professor Sir James Paterson Ross).
Primary popliteal thrombosis—thrombosis beginning at the level of the upper border of the femoral condyles has extended proximally as far as the tendon of adductor magnus (by courtesy of Professor Sir James Paterson Ross).

The wall of the artery is normal. Patients with this condition have been traced for ten years and there has been no evidence of further arterial disease. This is in striking contrast with the after-histories of patients with proved arterial disease (“thromboangiitis obliterans” or arteriosclerosis).

The evidence in favour of primary popliteal thrombosis as a clinical entity may thus be summarised: 1) there is similarity of the arteriographic picture with that of traumatic thrombosis from dislocation of the knee joint; 2) there is no inflammatory or degenerative change in the walls of the artery; 3) the clinical course is more suggestive of injury than...
Primary popliteal thrombosis—the occlusion has extended distally from the level of the upper border of the femoral condyles; proximal extension is probably limited by a large sural artery.

constitutioinal disease. In further investigation of this possibility, special studies of the anatomy of the popliteal artery were made (Boyd and Wilde 1949).

**Anatomy of the popliteal artery**—After leaving the adductor hiatus the popliteal artery lies in loose fatty tissue and is freely mobile. Just above the level of the knee joint the artery enters a fibrous tunnel derived from the fascia on the deep surface of the gastrocnemius. The fascial covering narrows to form a definite fibrous band, \( \frac{1}{2} \) to \( \frac{3}{4} \) inch broad, attached to the capsule of the knee joint at the level of the joint. Arteriograms show that it is at this point that thrombosis of the popliteal artery usually occurs. It is suggested that injury, from
Primary popliteal thrombosis—thrombosis beginning at the level of the femoral condyles has extended both proximally and distally.

Normal knee movement or minor sprains and strains, may cause damage to the tunica intima and thrombosis of the vessel. The possibility that there may be traumatic thrombosis is obviously increased if there is abnormal fixation of the popliteal artery to the oblique ligament within the fibromuscular canal from inflammatory changes caused by minor infection of the toes or interdigital folds.

Care should be taken to distinguish primary thrombosis of the popliteal artery from secondary thrombosis occurring as a complication of senile obliterative arteritis. This distinction is fundamental in assessing the prognosis. Senile obliterative arteritis often occurs.
Traumatic thrombosis of the popliteal artery from posterior dislocation of the knee joint. Note that the thrombosis begins at the level of the upper border of the femoral condyles and that the arteriographic appearances are identical with those of primary popliteal thrombosis in which there has been no major bone or joint injury.

in the fourth decade. On the other hand degenerative arterial changes may be delayed until much later in life. Occlusion of the popliteal artery in a man aged thirty-five years may not, therefore, be due to primary thrombosis; it may be secondary to early senile obliterative arteritis, the prognosis then being poor. A similar finding in a man aged fifty years, most commonly secondary to degenerative change, could possibly be primary if there was no generalised arteriosclerotic change, and in this event the outlook for both life and limb would be good. It is clearly important to determine by careful examination of the cardio-vascular system whether the thrombosis is primary or secondary.
INTERMITTENT CLAUDICATION

JUVENILE OBLITERATIVE ARTERITIS

For the clinical syndrome in which obliterative arteritis begins distally in the arteries of the feet in young men, the general title "juvenile obliterative arteritis" is preferred to that of thromboangiitis obliterans, or Buerger's Disease. The first accurate study of "thromboangiitis obliterans," originally described by Winiwater in 1879, was published by Leo Buerger in 1908 and has since been known by his name. "Buerger's disease" like "Raynaud's disease" includes a number of conditions of differing pathology that have been separated from the general group of peripheral vascular disorders in which there is obliterative arteritis of distal distribution. The term "Raynaud's phenomena" is now used rather than "Raynaud's disease"; and similarly, for these conditions, some of which are due to arteriosclerosis, "Buerger's syndrome" would be more appropriate than "Buerger's disease."

After separating from this general group cases that are due to degenerative arteritis there remains an obliterative arteritis of obscure origin with characteristic clinical features that might be called "juvenile obliterative arteritis" or "thromboangiitis obliterans."

Juvenile obliterative arteritis seldom, if ever, begins after the age of thirty-five years. The obliterative process begins in the small arteries of the feet. Gradually, but relentlessly, it ascends the limb until the popliteal or even the femoral vessels are occluded (Figs. 8-10). There is often fungus infection of the interdigital folds. Patchy phlebitis in the superficial veins may precede the signs of arterial involvement—an occurrence that is seldom seen in arteriosclerosis. The pain usually becomes intolerable and by the time the obliterative process involves the popliteal artery amputation is demanded. The disease is often more advanced in one limb than the other but it is always bilateral. The more recently affected limb may need amputation even before the limb that was first affected.

In early cases, arteriography shows narrowing and finally obliteration of the small arteries of the feet. In more advanced cases arteriograms show relentless proximal spread of the disease; the tibial arteries, and eventually the popliteal arteries, become involved. A line can almost be drawn across the limb at the point to which the disease has reached. Collateral vessels show the corkscrew appearance that is associated characteristically with rapid hypertrophy and dilatation—a feature less marked in distal obliterative arteritis of arteriosclerotic origin.

Ischaemic pain and colour changes in the toes are often followed rapidly by ulceration and gangrene. Intermittent claudication in the small muscles of the sole of the foot, which unfortunately is often mistaken for the symptoms of chronic foot strain, may precede the more obvious phenomena of ischaemia. Ulceration occurs early, sometimes with gangrene, and not uncommonly in the presence of palpable pulses at the ankle. The course is progressive, though periods of apparent quiescence in one or other leg are often seen. One or both limbs are usually lost within three to five years of the onset of the disease.

It would appear that juvenile obliterative arteritis is confined to the lower limbs but this is an opinion which may need to be modified in the light of further experience. Involvement of the upper limbs is more typical of the distal type of senile obliterative arteritis.

Little help is gained from studying pathological changes in the vessels. Buerger, in his original study of nineteen amputated legs, described the macroscopic and microscopic changes which he considered to be specific. There was macroscopic evidence of perivascular fibrosis welding the artery, vein and nerve into a dense cord of fibrous tissue. Microscopy showed inflammatory infiltration of the vessel walls with fibrosis of the adventitia, atrophy of the media, and slight thickening of the intima from proliferation of endothelial cells. The lumen of the vessel was blocked by clots in various stages of organisation and, eventually, the thrombus became partly recanalised. These changes are seen also in arteriosclerosis. Any surgeon who has experience of amputating limbs for senile gangrene will have noticed
occasional difficulty in separating the components of the neurovascular bundle which are often bound inextricably by fibrous tissue. The change is most marked if there has been sepsis with ulceration and gangrene. When there has been recent infection the tissue planes around the neurovascular bundle are oedematous. In late cases, the inflammatory tissue is organised and converted into dense fibrous cords. These changes give rise to difficulty when
peripheral sympathectomy is attempted because isolation of the posterior tibial nerve from the artery and veins may present the greatest difficulty. The inflammatory change depends largely upon the degree of infection in the perivascular lymphatics. Specimens of the posterior tibial artery obtained by arterectomy in early cases, before there was infection and ulceration, do not show such marked perivascular fibrosis.
SENILE OBLITERATIVE ARTERITIS

This group includes the great majority of patients who complain of symptoms due to arterial deficiency. The degenerative arterial changes are most marked in the lower limbs. It is important to lay stress on the fact that senile obliterative arteritis is not confined to old age; it is seen in patients with wide variation of age. Marked atheroma has been recorded in young children, and it is by no means rare in the third decade; it is the most frequent cause of occlusive arterial disease between the ages of thirty and forty years; and it accounts for practically all cases of organic obliterative arterial change after the age of forty years.

a) Diffuse obliterative arteritis—Arteriography shows narrowing and irregularity of the main vessels with a beaded appearance of the larger branches (Fig. 11). Sometimes the pathological changes seem to fall principally on the main vessels; the smaller branches are abundant, and a collateral circulation is well developed. This is the most common group and there is often calcification of the arterial walls. In others, changes in the main vessels are less marked; the most noticeable features are the paucity of muscle branches which appear to end abruptly, the absence of collateral anastomoses, and the pronounced muscle wasting.

There is a third group, small but interesting, in which the most marked clinical feature is coldness of the feet and often of the hands. The toes, and sometimes the feet, are deeply cyanotic and cold but they become pink and warm when vasoconstriction is abolished by reflex heating or by paravertebral block of the lumbar sympathetic chain. There is little if any muscle wasting. Oscillometric readings are normal as far down as the ankle. In all three groups the blood pressure is usually high.

b) Secondary popliteal thrombosis—Thrombosis of the popliteal artery occurs in nearly half the patients with diffuse arterial disease. The arteriographic picture (Fig. 12) resembles that of primary popliteal thrombosis. Thrombosis begins in the segment of the popliteal artery behind the knee joint where the artery is attached to the capsule of the joint by a fibrous band. In diffuse obliterative arteritis arteriography shows the greatest changes in this segment. Repeated minor injuries caused by flexion and extension of the knee joint probably determine localisation of the disease to this part of the vessel. Thrombosis, beginning in the condylar region, usually extends upwards as far as the adductor opening (Fig. 13). Sometimes, however, distal extension occurs with occlusion of the lower half of the artery and the first few inches of the tibial vessels. Occasionally thrombosis extends in both directions, thus involving the entire length of the popliteal artery. The extent of thrombosis is probably determined by the calibre of branches leaving the artery, proximal spread being limited by the brisk flow through the anastomotic magna and distal spread by the flow through a large sural artery. The onset of thrombosis may be determined by injury or by lowering of the blood pressure with diminution of blood flow due to confinement to bed by reason of illness or operation. The effect of secondary popliteal thrombosis depends upon the degree to which the collateral circulation has already developed. If the calibre of the main vessels has been diminished gradually over a long period of time the collateral circulation is well developed and the effect of a new lesion on the peripheral circulation is negligible, but if thrombosis occurs early in the disease, severe ischaemia is bound to result.

c) Secondary femoral thrombosis—Thrombosis of the superficial femoral artery is much less frequent. In the series now reported it occurred in only fifty-five of 472 patients. Arteriography shows occlusion of the superficial femoral artery from the adductor opening to the point where the profunda femoris leaves the main trunk (Fig. 16). Thrombosis has been shown to begin in the region of the adductor opening, proximal extension being limited by the brisk flow of blood through the profunda femoris (Fig. 14). Arteriograms in diffuse obliterative arteritis often show gross deformity of the femoral vessel at the level of the adductor opening where the artery may be tethered to the margin of the tendinous opening.
The occurrence of femoral thrombosis is determined by much the same factors as those of popliteal thrombosis. The effect on the circulation in the limb depends, as in popliteal thrombosis, on the extent to which the collateral circulation has developed. Generally speaking the higher the block the less marked are the effects on the peripheral circulation.

Femoral thrombosis is to be suspected clinically if there is claudication in the calf of a limb which appears to be unusually healthy. Nutritional changes in the skin, nails and muscles—so commonly seen in secondary popliteal thrombosis—are seldom found when the femoral artery is occluded. Nevertheless, in femoral thrombosis, distal extension with gangrene of the limb occurs more commonly than in popliteal thrombosis.
Senile obliterative arteritis—b) Secondary politeal thrombosis. Early lesion beginning at the level of the upper border of the femoral condyles.

**CLINICAL INVESTIGATION**

Most patients in this series were first seen in the Neurovascular Out-patient Clinic of the Manchester Royal Infirmary. Investigation in the out-patient department was designed primarily to enable the surgeon to decide whether treatment could be given as an out-patient or whether it was advisable to admit the patient for further study. Peripheral vascular disease is of course part of a generalised degenerative change; the patient must be assessed as a whole, and it is not possible to carry out complete examination of the cardio-vascular system in a busy out-patient clinic; but before operative procedures were undertaken detailed...
in-patient investigation was always made. Salient features in the history and clinical findings were recorded on a printed proforma which recorded the location of pain, the approximate date when it was first noticed, the suddenness of onset, the distance walked at the time of onset and at the time of examination, and the presence or absence of coldness, numbness or rest pain. A record was made of history of injury, frostbite or phlebitis, of the amount of tobacco smoked, and of the age and cause of death of the parents. Clinical data included records of the general health, the state of the heart and blood pressure, the nutrition of the limb as estimated on an arbitrary scale—Grades I, II and III—the presence or absence of

Fig. 13

Senile obliterative arteritis—b) Secondary popliteal thrombosis (note extension of thrombosis upwards as far as the opening of adductor magnus).
muscle wasting, atrophy of the small muscles, atrophy of the nails, and colour changes. Records of walking-ability included the rate of walking, the site of pain after walking, the time of onset, the time of halting, the radiation of pain, and the relation of walking to diminution or stabilisation of pain. The pulses and oscillometric readings in both lower limbs were recorded on a chart. Oscillometric readings were taken above and below the knee, and above the ankle. The oscillometer is not an instrument of precision but it does allow assessment of changes in the pulsation of main vessels. Reduced oscillations throughout the limb are characteristic of diffuse obliterative arteritis; complete absence of oscillation is
Senile obliterative arteritis—illustrating the progress of a secondary femoral thrombosis.

usually found below a main vessel block, though it is to be noted that if the collateral circulation is well developed there may be slight excursion of the needle. One of the authors (A. M. B.) has used the oscillometer for fifteen years and during that time arteriography of doubtful cases has never revealed evidence of main vessel block that was not suspected by oscillometry.

Investigations thus outlined determine the underlying cause of claudication; and the clinical history of the localisation of pain indicates which muscles are involved. It remains to confirm the observations of the patient by examining his reactions to measured degrees
Secondary femoral thrombosis—fully developed secondary thrombosis of the superficial femoral artery.

of exercise, and also to assess the severity of the condition. The circulatory deficiency must be estimated in terms of the demands made by the individual upon his blood supply. The primary concern of a patient is the interference with his daily life caused by pain in the leg and, when the limb is not in danger, treatment must be directed to the relief of subjective manifestations. Estimates of the relative deficiency were therefore made by testing the ability of each patient to walk at his own rate under controlled conditions. This was done first by walking over a known circuit of the hospital corridors; but the test has now been replaced by walking on a machine specially constructed for the purpose (Fig. 17). An endless
belt, driven by an electric motor at a speed ranging from one to four and a half miles per hour, is fitted with a tachometer giving the speed in miles per hour and the distance travelled by a point on the belt. The patient steps forward from a stationary platform on to the belt which is moving towards him at the slowest speed; and the speed of the belt is then increased gradually until he believes that he is walking at a normal rate.

![Diagram](image)

**Fig. 17**
The claudicometer—exercise tolerance machine.

Such a machine permits direct observation of the limb throughout the test. It has been noted that sometimes there is pallor of the feet at the onset of claudication. Oscillometry after the exercise-period sometimes shows reduced readings, contrary to the usual finding of a marked increase. During the exercise-test the patient describes his sensations. From the data thus obtained it is possible to classify the type of arterial disorder.

**CLASSIFICATION INTO CLINICAL TYPES**

The hypothesis that there are three clinical types of claudication depends upon consideration of the events taking place in a muscle during exercise. The resting muscle has a scanty blood supply; but when it is exercised there is capillary vasodilatation (Krogh 1922). If the muscle is exercised under ischaemic conditions there will be pain (Lewis, Pickering and Rothschild 1929). It cannot be doubted that the protatic event in these phenomena is the accumulation of metabolites—both in the case of vasodilatation and of pain (Hamilton 1947, Lewis 1942). It is beyond the scope of this paper to discuss the mechanism involved, the nature of the various metabolites concerned, or their mode of action. The basic concept is not affected.

With the onset of muscle-exercise, metabolites accumulate until the threshold for the reaction of vasodilatation is reached, such increase in blood supply dissipating the metabolites by chemical or physical processes. Eventually equilibrium is reached—the metabolites being eliminated at the same rate that they are produced. The level at which this takes place depends, for a given rate of exercise, upon the available blood supply. In the normal individual equilibrium is reached before the metabolites accumulate sufficiently to produce pain. In the patient with claudication the threshold of pain-reaction is reached before equilibrium is attained. Figure 18 is an attempt to express this diagrammatically; the horizontal axis represents time from the beginning of exercise; the vertical axis represents accumulation of metabolites; curve (a) is that of a normal individual; curve (b) is that of a patient with claudication. The dotted lines show the levels of metabolites required to initiate the increase of blood supply ("vasodilatation") and the pain reaction.

Most patients with intermittent claudication find that when pain begins it is relieved quickly by a short period of rest and thereafter they can go on walking. In such cases,
metabolites were accumulated until the threshold was reached and they were dissipated during the rest period, only to rise again when walking was resumed (Fig. 19).

When a patient is asked to walk for the purposes of investigation he is not allowed to rest when pain first develops; he is urged to continue walking as long as possible. The events that occur after the onset of claudication vary according to the relationship of blood supply to the demand and three types of reaction may be distinguished. **Type 1**—In this type the
blood supply and demand are very nearly equal. When the patient is urged to continue walking he announces, usually in a surprised tone, that the pain has disappeared. Here the level of metabolites crosses the pain threshold before a position of equilibrium is reached; but when equilibrium is finally attained it is below the threshold (Fig. 20). The fact that equilibrium has been attained just below the threshold of pain can be demonstrated by making the patient walk more quickly than usual; pain then returns. Type 2—Most patients belong to this group. When the patient continues walking the pain attains a steady level and eventually he stops walking not because of the intensity of the pain but because of its persistence. Equilibrium is attained only above the threshold for pain-reaction (Fig. 21). Type 3—Here the blood supply is so far reduced that stability cannot be achieved; on being urged to walk further the patient complains that the intensity of pain is increasing; finally it becomes intolerable so that he is forced to halt. In this case the position of equilibrium is at so high a level that it cannot be reached because in attempting to achieve it there is unbearable pain (Fig. 22).

**TREATMENT OF INTERMITTENT CLAUDICATION**

**General considerations**—If traumatic thrombosis is excluded, the arterial condition that underlies intermittent claudication is widespread, progressive and ultimately fatal. Lack of knowledge of the etiology of the responsible arterial conditions limits the treatment of intermittent claudication to alleviation of the subjective manifestations that interfere with the daily life of the patient. The most rational method of relieving exercise-pain would be to increase the blood supply in order that the demand of exercising muscles might be met. When the deficit is small this can be done; but in severe cases the gap between supply and demand may be too great, or it may not be possible to increase the blood supply because there is severe occlusive change in a group of muscular vessels. Under these conditions relief can be gained only by reducing the function of the affected muscles.

In practice the problem is more complex than one of simple supply and demand. Each individual case must be considered carefully and many factors of varying importance must be assessed accurately, always making allowance for the fact that many patients are poor witnesses and that it is not always easy to determine the degree to which an individual is inconvenienced by claudication, or indeed whether he is claudicating at all.

**Factors influencing the choice of treatment**—1) Clinical type—In determining treatment, the clinical type of claudication is by far the most important factor and this cannot be over-emphasized. Failure to appreciate that there are different grades of claudication has led, on the one hand, to extravagant claims for the success of certain drugs or methods of treatment and, on the other, to disrepute of sound and logical procedures. The general principles of treatment that are indicated in each clinical group can be defined clearly. Clinical Type 1—In this type, supply and demand are almost equal. The very slightest increase in blood flow brings about complete relief of exercise-pain and there is a good response to any method of treatment that improves the blood supply. Suggestion is a powerful factor in any form of treatment and it may have particular application to patients who suffer from claudication. If the patient is a Type 1 claudicant he will respond to almost any method of treatment, provided only that his confidence is gained, and this accounts for the reputation that has been gained for Buerger's exercises, intermittent venous occlusion, and the administration of muscular extracts, hormones and anti-coagulants. Clinical Type 2—In this type of claudication the gap between supply and demand is much wider and relief can be gained only by bridging the gap. As a rule the supply is increased to meet the demand, but in certain cases the demand may be reduced to meet the supply. Sometimes a combination of both methods may be adopted. Clinical Type 3—In this type the gap between supply and demand can be narrowed but never closed—a fact that is of fundamental importance. Disappointment is
bound to result from operative treatment or from any measure that is designed solely to improve the blood flow. Relief from severe pain can be secured only by reducing the function of the muscles by neurectomy or tenotomy.

2) Age—The age of the patient has an important bearing on treatment; the younger he is the more necessary is it to secure the greatest possible increase in blood supply. Generally speaking, lumbar sympathectomy should be advocated in patients under the age of forty to fifty years regardless of the clinical category in which they may fall, not only because in younger patients it may be possible to improve the general nutrition of the limb, and by attention to general health to delay the progress of arteriosclerotic change, but also because the longer the patient has to live the more likely it is that amputation will be needed sooner or later.

3) Type of arterial disease—Estimation of the severity and type of the causative arterial condition is of great importance in planning treatment. In traumatic thrombosis the expectation of life is normal and the only danger is that of local failure of the collateral channels to undergo sufficient hypertrophy and hyperplasia. The most vigorous and dependable method of increasing blood supply and encouraging the collateral circulation is indicated. The questions that arise are: what is the risk to life, and what is the risk of losing the limb? The risk to life—Experience during the last eighteen years has shown that in diffuse obliterative arteritis, with calcification of the main vessels, loss of life is more likely than loss of the limb. These patients often suffer from hypertension, coronary thrombosis, left heart failure and cerebral complications; sometimes there are mesenteric vascular accidents. Calcified vessels associated with hypertension show little tendency to thrombosis, and peripheral gangrene is unusual. In general, angina of effort, past history of coronary thrombosis, dyspnoea on exertion, and hypertension are contra-indications to lumbar sympathectomy. Lumbar sympathectomy should be advised only in patients with diffuse obliterative arteritis and calcification of the main vessels when it is felt that the limb itself is in danger. The risk of losing the limb—Peripheral gangrene leading to loss of the limb is more likely to occur in obliterative arteritis complicated by secondary thrombosis of a main vessel, especially when the superficial femoral artery is involved. Patients with secondary thrombosis of the superficial femoral artery nearly always have a normal or low blood pressure and are specially prone to massive thrombosis followed by extensive gangrene. In Type 3 claudicants, where no more than a pain-relieving procedure is indicated, an effort to improve the blood flow should always be made. This applies also to patients with superficial femoral thrombosis.

4) Other painful conditions in the limb—It is not surprising that claudication in patients who are past middle-age, brought about by degenerative arterial changes, is often associated with other signs of wear and tear of the tissues. The clinical picture is often complicated by osteoarthritis, chronic foot strain, deep thrombo-phlebitis or varicosity of the veins. The contribution of each of these conditions to the disability must be estimated. It is clearly of no value to gain operative relief from claudication if, in fact, the pain was due to osteoarthritis of the knee joint. Much help can be gained in differentiating joint, muscle and vascular pains by infiltration of the popliteal or tibial nerves with novocain, and by paravertebral block of the lumbar ganglia. By repeating walking tests after the various nerve blocks it is usually possible to determine the relative importance of each source of pain.

5) Influence of associated disease—Chronic bronchitis and emphysema, angina of effort, and obesity, are often associated with arteriosclerosis and intermittent claudication; and operations for the relief of intermittent claudication should not be undertaken if, in fact, the halting of the patient is due to dyspnoea. The importance of these conditions can best be determined by direct observation under walking conditions. Diabetes, unless mild and well controlled, worsens the prognosis and accounts for rapid progress in the arteriosclerotic changes and the development of peripheral gangrene. Diabetes must be excluded before decisions are made as to treatment.
6) Economic and geographical factors—The necessity for immediate and complete relief of pain may arise from the inability of a patient to walk long distances to and from his work; and sometimes the contours of the country in which he lives may be a determining factor. A wealthy patient, with car and chauffeur, who is brought to a halt at 150 yards may have no need, and indeed no inclination, to walk more than 100 yards and he can be treated palliatively; whereas a patient able to walk 400 yards who is compelled to walk half a mile to his work requires direct attack on the affected muscle.

Details of treatment—Buerger’s exercises, contrast baths, intermittent venous occlusion, suction-pressure and similar measures of treatment had been prescribed in many cases included in this series before we first saw them but without any real evidence of improvement. We believe that they are of little value and have not used them. The methods of treatment that have been employed will be discussed under the headings: 1) methods of improving the blood supply; 2) methods of decreasing the vascular demands of muscles; 3) methods of relieving pain. The results, with brief comments, are tabulated at the end of each paragraph.

The classification of results calls for a word of explanation. Success claimed for 100 per cent. improvement might in practice mean no more than an increase in walking distance from one to two hundred yards. It was decided therefore to classify results under three headings: Good, meaning complete relief from pain or ability to walk one mile without pain; Improved, indicating that the patient was able to walk a useful distance, arbitrarily fixed at half a mile, without pain; Unrelieved, including patients in whom the walking distance was often much increased but was still not accepted as being of economic value.

METHODS OF IMPROVING THE BLOOD SUPPLY

Lumbar ganglionectomy—The quickest, the most radical, and in general the most satisfactory way of improving the blood supply is by sympathectomy. Evidence of calcification of the main vessels is no contra-indication to sympathetic denervation. Some of the most successful results of sympathectomy in senile obliterative arteritis have been gained in patients with radiographic evidence of calcification of the main vessels. Release of normal tone increases the blood flow even if there is no evidence of abnormal vasoconstriction. Generally speaking, lumbar ganglionectomy should be undertaken in all patients below the age of sixty years if the general condition does not contra-indicate operation. The first, second and third lumbar ganglia, with the intervening chain, should be removed in order to be certain that denervation is complete. The more usual operation, in which only the second and third ganglia are removed, is apt to result in incomplete denervation (Jepson and Ratcliffe 1949).

In patients over the age of sixty years lumbar ganglionectomy is not always advisable but if the general cardio-vascular condition is satisfactory there seems no reason why sympathectomy should not be undertaken even in these older patients. If it is believed that the risk of operation is not justified, chemical destruction of the sympathetic chain with 10 per cent. phenol is worth considering (Haxton 1947).

<table>
<thead>
<tr>
<th>Type 1</th>
<th>Good</th>
<th>Improved</th>
<th>Unrelieved</th>
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<tr>
<td>Type 2</td>
<td></td>
<td>11</td>
<td>5</td>
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<tr>
<td>Type 3</td>
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Comments on treatment by lumbar ganglionectomy—The results of lumbar ganglionectomy for arteriosclerosis confirm our previous views that this simple operation offers the best chance of rapid and certain improvement. The patient is allowed to get up on the fourth day and is discharged from hospital on the eighth or ninth day after operation. In many cases, walking
tests carried out on the day that they are discharged from hospital show great improvement. In others there is little immediate change but review one month later shows improvement. The distance that they can walk without pain steadily increases until most of them can walk a mile or more in comfort. The operative mortality is low. In this series, there was one death from coronary thrombosis on the ninth post-operative day. Altogether 632 lumbar ganglionectomies have been performed between January 1947 and March 1949 with one death. Two patients had cerebral thrombosis within forty-eight hours of operation but both recovered completely within a few weeks.

Paravertebral block with 10 per cent. phenol—Chemical destruction of the lumbar ganglia has proved to be of great value in patients in whom lumbar ganglionectomy was contra-indicated by reason of advanced age or impairment of general condition. The use of alcohol has been abandoned almost entirely on account of the not infrequent occurrence of muscular palsy and severe neuralgia due to unavoidable inclusion of the spinal nerves in the field of injection. Mandll (1948) showed in cats that 6 per cent. phenol had a selective action on the sympathetic ganglia causing complete destruction without damaging the spinal nerves.

H. A. Haxton (1947), working in the Neurovascular Unit at the Manchester Royal Infirmary, suggested 6 per cent. phenol for paravertebral block of the lumbar chain. Haxton's early cases, in which he used 5 cubic centimetres of 6 per cent. phenol, showed evidence of incomplete sympathetic denervation without undesirable sequelae. In order to accomplish more complete destruction of the lumbar ganglia he increased the strength of the phenol solution to 10 per cent., and the quantity injected to 10 or 15 cubic centimetres. Paravertebral block with 10 to 15 cubic centimetres of 10 per cent. aqueous solution of phenol has been used in this Unit in a large number of patients since May 1947, with gratifying results. So far no complications of any sort have been met.

Technique of paravertebral block—The equipment needed is a 10 c.c. Labat syringe, a fine needle for intradermal injection, 12 cm. and 16 cm. rustless steel needles 0·8–1 mm. diameter, 2 per cent. novocain, and 10 per cent. aqueous solution of phenol. The injection can be carried out easily in bed. The patient lies on his side with a pillow under the loin in order to separate the lumbar transverse processes widely. It is important that the back should be kept straight, avoiding either flexion or extension, and that the trunk should be in a strict lateral position at right-angles to the bed. The foot of the bed is raised on 10-inch blocks in order to encourage seepage of the phenol upwards along the tissue planes around the sympathetic chain, thus reaching the first lumbar or even the twelfth dorsal ganglia.

The bedclothes should be removed so that the lower extremities are exposed to room temperature, about 20° C. being ideal. The skin temperature of the feet, preferably on the inner side of the heel, is recorded, readings being taken every few minutes until the skin temperature reaches a steady level. After skin preparation and isolation of the lumbar region with sterile towels, an intradermal wheal is raised with 2 per cent. novocain at a point near the outer border of the erector spinae, four fingers breadth lateral to the spine of the second lumbar vertebra. A 16 centimetre needle is most commonly used but a 12 centimetre needle is adequate in small and thin individuals. The needle is passed obliquely through the selected point, directed medially at an angle of about 30 degrees from the horizontal plane. The operator should make a mental picture of the relations of the erector spinae, psoas muscle and vertebral bodies, drawing an imaginary line from the point of injection to the front of the body of the second lumbar vertebra.

By this technique the needle often passes lateral to the tip of the transverse process. If possible the transverse process should be felt, the needle be withdrawn a little, inclined slightly upwards or downwards in order to pass above or below the bone, and then advanced a further 4 or 5 centimetres through the psoas muscle until the antero-lateral aspect of the vertebral body is reached. Occasionally the needle passes in front of the vertebral body and pieces the aorta or vena cava. Puncture of the great vessels is harmless and is, in fact, a useful indication of the position of the needle point. If this occurs the needle should be withdrawn and reinserted at a greater angle until bone is felt. When the needle is judged to be placed satisfactorily in close proximity to the lumbar chain, after careful aspiration in order to be quite certain that the spinal theca or a blood vessel has not been entered, 2 cubic centimetres of buffered 2 per cent. novocain solution is injected.

If the needle has been placed correctly a rise of skin temperature, usually first detectable in the skin over the medial side of the heel below the medial malleolus, will be recorded within two or three minutes. A large rise in the skin temperature cannot be expected in patients with advanced arteriosclerosis. In
any patient, however, in whom phenol block is indicated, there will be some elevation of skin temperature. A rise of even one degree, occurring within two or three minutes, shows that the needle is correctly placed.

If there is no alteration in skin temperature within five minutes, the needle should be withdrawn and reinserted at a different angle and the novocain injection repeated. It is unwise to inject the phenol solution unless the point of the needle is proved to be correctly placed. Only when the operator is satisfied with the position of the needle should 10 to 15 cubic centimetres of 10 per cent. aqueous solution of phenol be injected. The phenol may not be in complete solution at room temperature. If the fluid is cloudy, the bottle should be warmed by standing it in hot water for a few minutes until the phenol is completely dissolved. The syringe also should be warm.

The patient should remain in the lateral position for twenty minutes in order to keep the pool of phenol in contact with the lumbar ganglia. He may then be turned on his back and the blocks removed from the foot of the bed. He should remain flat on his back for one hour, after which he can return home with instructions to lie down for the rest of the day.

**TABLE III**

<table>
<thead>
<tr>
<th>Type</th>
<th>Good</th>
<th>Improved</th>
<th>Unrelieved</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 1</td>
<td>3</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Type 2</td>
<td>5</td>
<td>9</td>
<td>4</td>
</tr>
<tr>
<td>Type 3</td>
<td>—</td>
<td>—</td>
<td>6</td>
</tr>
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</table>

Comments on treatment by paravertebral block with phenol—Chemical sympathectomy with 10 per cent. phenol has a definite though limited place. The method has been used in patients over the age of sixty years and in those in whom lumbar ganglionectomy was contra-indicated by reason of their general condition. Paravertebral block with phenol has been used also in advanced Type 3 cases where the patient complained of a cold foot or where the feet showed severe nutritional changes or incipient gangrene.

**Vitamin E (α-tocopherol) therapy**—It is unfortunate that the use of α-tocopherol in cardio-vascular conditions has been exploited by the popular press as a new “miracle drug.” Critics have applied the principle “Falsus in uno; falsus in omne” and thereby discredited favourable observations. The treatment was supported by Vogelsang and Shute (1947, 1948) and criticised by Baer and Heine (1948, 1949). The authors did not feel convinced by the arguments presented on either side and decided that observations should be made on selected cases. Vitamin E was given to Type 2 cases who were awaiting admission to hospital. Ten patients were given daily doses of 200 milligrammes of α-tocopherol (Ephynal, Roche). Their progress was reviewed after one month. They stated that they felt better but assessment of walking-ability showed little if any change. In every case, however, there was noticeable objective improvement after two months. The dose of “Ephynal” was increased to 400 milligrammes daily with further improvement. Larger doses have been used since, but it would appear that a daily dosage of 400 milligrammes is optimal. In view of these results it was considered that further observations were warranted. Vitamin E has also been given with benefit to patients with severe nutritional changes in the feet.

**TABLE IV**

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<thead>
<tr>
<th>Type</th>
<th>Good</th>
<th>Improved</th>
<th>Unrelieved</th>
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<tbody>
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<td>Type 1</td>
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<tr>
<td>Type 2</td>
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<td>32</td>
<td>13</td>
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<tr>
<td>Type 3</td>
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Analysis of the cases treated with α-tocopherol is shown in Table IV. It must be emphasized that these figures are derived only from clinical observation. The number of variables is such that strictly controlled experiments must of necessity involve long term studies; these are being undertaken and the results will be reported in due course.
Comments on treatment by \( \alpha \)-tocopherol therapy—\( \alpha \)-tocopherol is the only substance that has given consistently good results in cases other than those grouped in Type 1. Patients with vascular disorders of Type 2 and 3 who had previously been treated with other drugs without benefit showed definite improvement when treated with "Ephynal." The consistency with which there was a lag period of four to six weeks before improvement was noted was most striking. After a few months there was much improvement in the appearance of the feet in patients who showed nutritional changes.

METHODS OF DECREASING THE VASCULAR DEMAND BY MUSCLES

Thiouracil—A trial of thiouracil in the treatment of intermittent claudication was initiated by a chance observation. A patient attended the Vascular Clinic of the Manchester Royal Infirmary who had suffered for many months with Type 3 claudication and was also severely thyrotoxic (B.M.R. + 50 per cent.). It was decided that the thyrotoxicosis should be controlled with thiouracil before treatment of the claudication was undertaken. When a normal B.M.R. had been reached, and the clinical manifestations of hyperthyroidism were controlled, it was discovered that the pain of claudication was greatly relieved.

<table>
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<tr>
<th>TABLE V</th>
<th>Results of Treatment by Thiouracil in Thirty-one Cases</th>
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Thirty-one patients with intermittent claudiation of mixed types, whose B.M.R.'s and blood cholesterols were within normal limits, were then treated by a prolonged course of thiouracil until a state of early myxoedema, confirmed by B.M.R. and blood cholesterol readings, was reached.

Comments on treatment by thiouracil—No significant improvement was noted in this group as a whole, even in cases that developed severe myxoedema. Our conclusion is that thiouracil is not warranted as a form of treatment for intermittent claudication.

METHODS OF RELIEVING PAIN

Antistin—The work of Barsoum and Gaddum (1935) suggested that in the ischaemic limb the histamine content of venous blood was increased. This statement has not been accepted generally but it seemed wise to try the effects of anti-histamine drugs to see if relief could be afforded to patients with claudication. Antistin was selected for trial because it appeared to

<table>
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<th>TABLE VI</th>
<th>Results of Antistin Therapy in Twenty-six Cases</th>
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<td>Good</td>
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<td>Type 3</td>
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have the least undesirable side-effects; and it was given to twenty-six patients in a daily dosage ranging from 300 milligrammes to 900 milligrammes according to tolerance. There were many unpleasant side-effects including headache, nausea and vomiting. One patient lost his sense of smell but recovered it when administration of the drug was stopped. There were two cases of cerebral thrombosis but these may have been coincidental. In view of the variable tolerance and the high incidence of side-effects, the treatment was discontinued.
Comments on antistin therapy—The mixed properties of the anti-histamine compounds at present available make it very difficult to assess their mode of action in intermittent claudication. The unpleasant side-effects make their use undesirable.

Myoneurectomy—Internal popliteal neurectomy—The possibility of relieving the pain of intermittent claudication by division of branches of the internal popliteal nerve to the gastrocnemius occurred to Sir James Learmonth and ourselves independently and at about the same time. While one of us (A. M. Boyd) was in charge of a Centre for Vascular Injuries in Egypt during the recent war it was observed that whereas ligation of the popliteal vessels usually gave rise to intermittent claudication, some patients were still able to walk without pain after excision of an aneurism in the popliteal fossa. It was noted that patients who did not complain of the pain of claudication had paralysis of the gastrocnemii, doubtless through division or stretching of branches of the internal popliteal nerve during exposure of the aneurism. The significance of this observation was not appreciated until 1947 when an arteriovenous aneurism of the popliteal artery was operated upon and the gastrocnemius was paralysed. Thereafter it was decided to divide the nerves to both heads of the gastrocnemius in patients with severe claudication.

In practice, the operation proved disappointing, although much interesting information was obtained. Patients who were relieved of the pain of claudication in the gastrocnemius nevertheless suffered pain in the soleus after walking a further hundred yards or so. The necessity of knowing the skin reference of pain from various muscles of the limb was thus revealed and for this reason studies of the cutaneous reference of pain from these muscles were undertaken (vide supra).

Division of the external popliteal nerve—Three patients complained of severe exercise pain in the extensors of the leg which halted them after walking 100 yards or less. The external popliteal nerve was infiltrated with 2 per cent. novocain and walking tests were then repeated. All three were relieved of pain and were able to walk more than half a mile in comfort. They did not mind the foot-drop and all agreed that the external popliteal nerve should be divided. The nerve was exposed through a half-inch incision over the neck of the fibula; it was crushed and divided. All three patients were able to walk painlessly on the second day after operation. So far they have remained satisfied.

Division of the posterior tibial nerve—A lady aged sixty years, weighing 18 stone (252 pounds) complained of severe pain in the left calf and in the sole of the left foot under the heads of all the metatarsals; it was difficult to determine which pain was the more crippling. There was diffuse obliterative arteritis and early nutritional changes in the foot. Paravertebral block with 10 per cent. phenol relieved the exercise-pain in the left calf, but the patient was halted at seventy yards by pain under the heads of the metatarsals. The posterior tibial nerve at the level of the ankle joint was infiltrated with 2 per cent. novocain, after which she was able to walk a quarter of a mile in comfort, being halted only by dyspnoea. Division of the nerve just above the ankle joint gave complete relief.

Tenotomy of the tendo Achillis—Sir Heneage Ogilvie (1949), in his Beyer Memorial Lecture, drew attention to the value of occasional idleness. In this erudite address, Ogilvie stressed the necessity of having time to think. One of us (A. M. B.) enjoyed such a period of idleness in an inn in Somerset, listening to local gossip, and in that time the possible value of tenotomy of the tendo Achillis occurred to him. He learned that in the latter part of the nineteenth century a general practitioner in that vicinity did all his rounds on horse-back and kept his horses until they were too lame to work, whereupon he divided a tendon near the hoof and thereby gained from them another five years of work. Horses are prone to claudication and it seemed that relief was obtained by dividing the tendon of the affected muscles.

After consultation with our orthopaedic colleagues we decided that division of the tendo Achillis might relieve exercise-pain in the calf muscles. The first patient upon whom this operation was performed was one with Type 3 claudication with exercise-pain in the right
calf which halted him at seventy yards. After division of the tendo Achillis he was able to walk two miles without pain on the third day after operation. He is now walking five miles daily in a hilly district in Wales carrying on his job as a postman. The operation of subcutaneous tenotomy of the tendo Achillis can be carried out in a few seconds under pentothal anaesthesia. The tendon is divided with a fine sharp tenotome just above the heel. The tenotome knife is inserted on the medial side and the tendon is divided from within outwards. There is no bleeding. The patient is able to get up the next day and most of them are able to walk half to one mile; others complain of pain at the site of operation and defer walking until the second or third day. By the fourth day after operation all patients are able to walk more than one mile without pain. There have been no complications and no serious disabilities after operation. The gait is almost normal and not noticeably worse than before operation. Plantar-flexion is of course weakened but most patients are able to rise on their toes. After a few months the severed ends of the tendon become united with lengthening, but the patients are still free from exercise-pain.

**TABLE VII**

<table>
<thead>
<tr>
<th>Results of Tenotomy of the Tendo Achillis in Twenty-four Patients</th>
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<td><strong>Type</strong></td>
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Comments on treatment by tenotomy of the tendo Achillis—This simple but eminently satisfactory operation has replaced popliteal myoneurectomy in this clinic. The operation was originally restricted to Type 3 cases. It was felt, however, that the results were so satisfactory that late Type 2 patients, whose improvement by other methods had been inadequate, might be included.

**GENERAL COMMENTS**

The most successful treatment for the Type 3 case of intermittent claudication, in which the blood supply is so far reduced that stability cannot be achieved by any attempt to improve the vascular supply, is undoubtedly tenotomy of the tendo Achillis. All except two patients have been able to walk more than a mile thereafter and the two who halted at less than that distance did so because of anginal pain. The operation, although trivial in itself, does cause slight disability and certainly will not permit elderly and shuffling patients with claudication to walk with the brisk gait of young men whose pain has been relieved. It does, however, relieve them of the pain which has made their lives a misery and it enables them to spend their remaining days in reasonable activity. For the Type 2 case of claudication, when age and general condition permit, lumbar ganglionectomy must be rated as the most successful treatment. In more elderly patients chemical sympathectomy has its place. Further comment on the results of treatment with a-tocopherol must be withheld until the results of controlled experiments are known. Type 1 cases can be cured by any form of therapy in which the patient has faith. The one failure in this series was a patient who could not be persuaded that his condition was not incurable.

**SUMMARY**

1. A description is given of historical discoveries relating to intermittent claudication. Various theories that have been advanced are discussed. A hypothesis, based on the work of Lewis, is elaborated.
2. A classification of obliterative arterial disease is outlined. The three groups that are distinguished are: primary thrombosis of the popliteal artery; juvenile obliterative arteritis; and senile obliterative arteritis.

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3. The methods adopted for assessment of the severity of disease, including study of the clinical features, arteriographic findings, results of novocain infiltration and examination of the patient on a walking machine, are reported.

4. Methods of treatment by Buerger's exercises, contrast baths, intermittent venous occlusion and suction pressure; by lumbar ganglionectomy and paravertebral block with phenol; by vitamin E (α-tocopherol) therapy; by treatment with thiouracil and antistin; by internal popliteal myoneurectomy and division of the external popliteal and posterior tibial nerves; and by tenotomy of the tendo Achillis, are discussed.

5. It is concluded that tenotomy of the tendo Achillis should replace myoneurectomy in Type 3 cases where the blood supply is so far reduced that vascular stability cannot be achieved, and that it might apply in Type 2 cases in which there is persistent pain at a steady level.

6. The results of treatment in 276 patients with intermittent claudication are recorded.

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