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Intermittent Claudication

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Intermittent claudication occurs most frequently in the lower extremities although other muscle groups are not immune. The diagnosis of intermittent claudication is acceptable when physical exertion produces pain or paresthesias in the foot, calf or thigh, promptly relieved by the cessation of the activity without change of position of the affected part. It never occurs in the legs as a result of standing, reclining or sitting and it has no relationship to the muscle cramps which patients have in bed.

Many diseases may affect the peripheral arteries and produce intermittent claudication. Thromboembolic obstruction, thromboangiitis obliterans, polyarteritis nodosa, scleroderma, and the arteritis associated with systemic infection may be encountered. Atheromatosis, with its predilection for the major arteries of the lower leg is the most common cause of intermittent claudication. The disturbance in function, however, is primarily dependent upon the location, extent and rate of development of the arterial obstruction rather than its specific etiology.

Mechanism

It is now generally accepted that intermittent claudication results from muscular ischemia secondary to arterial occlusive disease when blood supply is adequate for resting muscle but inadequate for a muscle which is actively contracting. The precise metabolic disturbance initiating the syndrome has, however, never been fully elucidated. The oxygen requirement of contracting muscle may increase 30 times above the resting level. This vastly increased requirement is met in part by extraction of more oxygen from each unit of blood passing through the capillaries and in part by an increase in arterial and capillary blood flow. It is the inability of obstructed arteries to deliver this additional blood that produces intermittent claudication.

Some investigators have maintained that arterial spasm alone can produce claudication. Support for this point of view has centered around the observation that there is often a diminution in the peripheral pulses after exercise in patients with intermittent claudication in whom pedal pulses were palpable at rest. Others have not found the evidence for spasm at all convincing. Pickering has shown how closely organic vascular narrowing and occlusion may mimic vascular spasm. The two known stimuli producing spasm are trauma and cold. While any artery adequately stimulated mechanically will probably go into spasm, there are no documented cases of intermittent claudication occurring from spasm alone. Thus, in Raynaud's phenomenon, although an intrinsic inherited peculiarity causes the digital arteries to respond to cold with intense constriction, intermittent claudication is not seen.

The neurogenic aspect of intermittent claudication has been extensively studied. The sensory and motor changes developing during a period of circulatory arrest to a limb have been described. Marked sensory impairment has always been noted long before any per-
ceptible deterioration in motor power. In a recent investigation of the effect of peripheral ischemia on nerve conduction, it was shown that among afferent fibers, the more rapid the conduction velocity of the fiber, the greater the depressant effect of ischemia. Motor fibers, however, are more resistant to ischemia than certain afferent fibers of slower conduction rate. The different sensitivities of nerve fibers to ischemia may explain why pain precedes loss of motor power and why, under some circumstances, intermittent claudication may be experienced as a pain and at other times as a paresthesia.

**Diagnosis**

In the vast majority of patients with claudication the pedal pulses are absent. So constant is this finding that one should be extremely hesitant, in the absence of anemia, to entertain a diagnosis of claudication if both the dorsalis pedis and posterior tibial pulses are full and bounding. Intermittent claudication has probably escaped recognition in severe anemia because it is usually a minor complaint and one that is likely to be grouped among the paresthesias commonly found in anemic patients.

Occasionally, normal peripheral arterial pulsations may not be palpable because the vessels pursue anomalous pathways. It is clear, however, that a considerable degree of narrowing and even occlusion can be present in the large arteries without producing detectable reduction in arterial pulsation. The brief course and large diameter of some collateral branches that bypass short occlusions explain the frequent clinical finding of the return of pulsatile flow distal to a complete occlusion (fig. 1). In such cases one does not expect, and one does not usually find, evidence of ischemia. If a patient complains of pain in the leg and the arterial pulsations are full and bounding, the symptom almost certainly is not due to arterial insufficiency.

Claudication in patients with normal or only partially impaired peripheral arterial pulsations has been attributed to occlusions of the branch arteries supplying important muscle groups and also to arteriolar obstruction. Patients in whom pedal pulses were present have been observed with claudication secondary to occlusion of an iliac artery or to coarctation of the aorta. In coarctation the velocity of blood flow in the large arteries of the leg is reduced, and the arterial-arteriolar difference in blood pressure greatly diminished. This small arterial-arteriolar difference represents sufficient encroachment on the local circulatory reserve to produce ischemic pain without obliterating the pulse. In the occasional patient with claudication and palpable pedal pulsations the occlusion will frequently be found proximal to the femoral artery. In view of the demonstrated occurrence in peripheral atherosclerosis of obstruction of the large arteries and the development of a rich collateral circulation, which could occasionally permit pulsatile flow distal to the occluded area, this explanation (i.e. occlusion proximal to the femoral artery) appears more reasonable than one that postulates small-vessel obstruction as the sole mechanism for claudication.

The clinical picture of intermittent claudication is frequently complicated by the coexistence of other pathologic conditions, such as neuritis or arthritis, that may cause pain in

**Fig. 1.** Roentgenogram of an amputated limb after injecting the arteries with radiopaque medium. “A” indicates an anastomotic vessel between the calcified but patent anterior tibial artery and the peroneal artery. The internal diameter of the collateral channel exceeds that of the main peroneal artery.
an extremity. It is important routinely to consider the existence of these other lesions since the symptoms they produce may be more disabling than the claudication itself. In most instances a careful history and physical examination will clarify the problem.

Value of Oscillometry, Arterial Calcification and Arteriography

The oscillometer remains overrated as an aid in estimating the degree of arterial insufficiency. The impression that it is more accurate than palpation undoubtedly derives from its graphic representation of the arterial pulse. The normal range of values is extremely broad and is influenced by the diameter of the extremity as well as by the manner of application of the pneumatic cuff. The limitations of the instrument are emphasized by situations in which the arterial circulation is excellent and yet the oscillometric excursion small. Occasionally, careful palpation reveals pulsations which are not recorded by the oscillometer. The instrument has its main value in confirming the absence of palpable pulses and in demonstrating a difference in pulsatile flow in the two extremities when peripheral pulses cannot be discerned. It is, however, essential to realize that oscillometric readings do not accurately reflect the effectiveness of the collateral blood supply.

In the past, undue significance has been attached to the presence of calcification in the walls of atherosclerotic vessels. The clinical effects of atherosclerosis are not dependent so much on hardening of the vessels as on narrowing or obliteration of the lumen. Calcification is not only inconstant but also is a relatively benign process, causing little if any interference with the flow of blood. Of particular interest are several reports of the lack of correlation between arterial calcification and occlusion and even of an inverse relation between the two. A recent study has clearly demonstrated the absence of any correlation between calcification and occlusion in patients in whom

Fig. 2. Roentgenogram of amputated leg showing inverse relation between arterial calcification and occlusion. A: Lateral roentgenogram prior to arterial injection of radiopaque medium. Visible calcification of entire popliteal and anterior tibial arteries and upper fourth of posterior tibial artery. B: Lateral roentgenogram after injection. Popliteal, anterior tibial and upper fourth of posterior tibial arteries patent. Occlusion of remainder of posterior tibial and all of peroneal arteries. C: Roentgenogram of leg after it has been "unrolled" so that major vessels lay in one plane. Sites of patency and occlusion shown more clearly than in B.
the clinical picture of atherosclerosis and gangrene was sufficiently severe to necessitate amputation\(^2\) (fig. 2). These observations indicate that the taking of a roentgenogram merely to determine whether or not arterial calcification is present does not contribute materially to the diagnosis of intermittent claudication.

Since it is easy to demonstrate collateral vessels by the injection of radiopaque mediums during life it was natural that arteriography should be employed in the evaluation of patients with intermittent claudication. It has not been possible, however, to translate these objective anatomic evidences of collateral flow into functional terms. Arteriography, thus, cannot be used as a prognostic guide to therapy or as an indication of the paucity, richness or effectiveness of these auxiliary vessels. One does not need an arteriogram to appreciate that collateral circulation must be extensive if major arterial pulsations in the limb are absent and the tissues of the toes are viable.

It should be realized that, although occlusions are not rare in the femoral and iliac arteries, the most frequent site of obstruction in atherosclerosis is between the knee and the ankle.\(^2\) It is precisely this bottleneck area that is most difficult to visualize completely even with a good contrast medium. It is much easier both during life and at necropsy to demonstrate the femoral artery, the popliteal artery or the terminations of the anterior and posterior tibial arteries in the foot than to visualize the tibial arteries in the lower leg. The intra-arterial injection of radiopaque mediums, moreover, is not performed without discomfort or risk. Arteriography is a valuable diagnostic aid in reconstructive surgical procedures on obstructed arteries. Knowledge of the localization of the occlusive process and the limits of arteriography should, however, temper any surgical plan to relieve intermittent claudication in the atherosclerotic limb by restoring the integrity of the arterial lumen in the thigh.

**Therapy**

To provide effective therapy for intermittent claudication, the physician must be cognizant of certain facts about the syndrome and of the patient’s reaction to his disability. Spontaneous improvement in intermittent claudication shortly after its onset is frequently observed. Significant variations in exercise tolerance occur naturally in the course of the disease. After occlusion of a major artery in an extremity, one may expect an increase in collateral circulation during the first year: this increase may continue for two or three years, independent of therapy.

The initial distress of the patient with intermittent claudication is often out of all proportion to the actual disability. This discrepancy is due, in some cases, to the fact that the syndrome represents the first serious physical impairment encountered in a vigorous, active life and one that is envisaged as becoming inexorably more confining as time passes. Others interpret “hardening” of the leg arteries to mean the inevitable spread of vascular disease to other vital organs. Finally, there are those who equate intermittent claudication with gangrene and amputation. The physician who fully appreciates such fears is in an excellent position to help these patients.

In practice, the disability in most patients with intermittent claudication is not profound; only a small percentage have to change their employment because of a limited exercise tolerance. Furthermore, intermittent claudication does not have the same prognostic relation to gangrene and amputation that angina pectoris has to death from myocardial infarction, cardiac arrhythmia or congestive heart failure.

In this clinical setting therapeutic attempts to alleviate claudication should be essentially devoid of risk. In addition, all measures aimed at relieving intermittent claudication should have as their goal a decrease in the disparity between the supply and demand for blood by ischemic tissue. Any procedure diverging from this aim may aggravate the disability.

Today there is no single, effective measure for the treatment of intermittent claudication that has gained wide acceptance. This is not for any lack of ingenuity on the part of workers in the field. Within the last half-century many diverse therapies have been suggested. Drugs investigated have included calcium, vitamin E, theobromine, methyl testosterone, depropanex,
cytochrome C, intravenous hypertonic saline solution, thiouracil, histamine and various sympatholytic agents.

Mechanical aids, some of them based on demonstrably favorable physiologic alterations in peripheral blood flow, have included intermittent venous occlusion, passive vascular exercise (Pavaex) and Buerger’s exercises.

Surgical procedures have also covered a wide spectrum. It has been recommended that the femoral vein be ligated, and that the femoral artery be anastomosed to the femoral vein. More recently, accessible occluded femoral and popliteal segments have been resected and replaced by grafts. In addition to this direct attack on the blood vessels themselves, the sympathetic innervation of the lower extremity has been interrupted by peripheral stripping, paravertebral blockade with procaine, alcohol and phenol and extirpation of the lumbar ganglia. Finally, division of the common peroneal and posterior tibial nerves and tenotomy of the Achilles tendon have been attempted.

Much interest exists in the therapeutic value of interrupting sympathetic tone either medically or surgically in intermittent claudication. Although there is little evidence that the arteriolar vessels are in spasm in intermittent claudication, it has been assumed that dilatation of the arteriolar and postarteriolar bed will improve the nutrition of ischemic tissue. Unfortunately, methods such as measurement of skin temperature and limb volume changes do not record effective capillary blood flow but rather increments in the total amount of blood or the rate at which blood passes through a portion of the extremity. There is evidence that sympathetic denervation produces a relaxation of the direct arteriovenous communications as well as of the arteriole-metarteriole-capillary network. Whether it is possible, therefore, to improve capillary blood flow by removing normal tone is not at all clear. The observation that interruption of sympathetic activity may increase skin temperatures does not necessarily imply a similar vasodilatation in deep structures. The reverse has in fact been claimed. Some observers believe that interruption of normal sympathetic tone (even without producing systemic hypotension) may occasionally aggravate ischemia. Although secondary arterial and venous thrombosis has been implicated, the mechanism whereby such a catastrophe could occur might be from extensive diversion of blood from ischemic tissue through dilated arteriovenous shunts.

Systemically administered vasodilators may cause vasodilatation throughout the body, thereby defeating their purpose. It is clear that there is a relatively greater “resistance” to the interruption of sympathetic tone in the lower as compared to the upper extremity. This resistance may require the use of relatively large dosages of sympatholytic agents with resulting hypotension and tachycardia. Among patients with coronary artery disease such disturbances in hemodynamics may precipitate myocardial ischemia and even necrosis. In this regard it is essential to recall the high incidence of coronary artery disease in patients past 50 years of age as well as the fact that many patients may have coronary artery obstruction without clinical manifestations. In the great majority of patients with intermittent claudication, therefore, the systemic use of vasodilator drugs is probably contraindicated.

There have been several reports of the value of intra-arterial vasodilators in the management of intermittent claudication. This route of administration, it has been claimed, will minimize the systemic effects of the drugs. Recently, however, the intra-arterial injection of Priscoline has also been reported to cause myocardial infarction. Intra-arterial therapy requiring repeated arterial puncture invites, of course, the risk of thrombosis in sclerotic arteries. The value of such treatment in patients with advanced obliterator disease remains to be established.

Surgical sympathetic denervation has been claimed to produce a “local” vasodilatation and a relief from the pain of claudication. Yet, lumbar sympathectomy causes a response not only in the ischemic area but throughout the entire extremity. As mentioned above, there is inadequate evidence that effective nutritional blood flow is increased by this pro-
procedure. Finally, experimental and clinical data indicate that in a large majority of patients, return of sympathetic tone occurs within months of the operative procedure, even though the foot may remain warm. These observations tend to invalidate the claim that sympathectomy has any "prophylactic" value in the prevention of intermittent claudication.

In patients with intermittent claudication and arteriographic evidence of a segmental occlusion in the femoral artery, resection of the occluded zone and its replacement with a venous or arterial graft has been recommended. Such a procedure, it has been argued with justification, would deliver blood to the tibial arteries at a more effective pressure. This potential advantage, however, must be balanced against the limited disability produced by the claudication and the multiple risks of the operative procedure itself. Segmental arterial resection for intermittent claudication remains an experimental procedure: it is our opinion that the underlying pathologic disturbance and the clinical course of the disability will ultimately cause this therapeutic approach to be discarded.

Tenotomy of the Achilles tendon is another procedure recommended for the relief of intermittent claudication. It is well established that uncomplicated claudication can invariably be relieved by a slowing of the rate at which the patient walks; tenotomy effectively achieves such a reduction in speed. However, it is usually possible to induce patients to walk more slowly without resorting to psychotherapy.

Despite the apparent therapeutic nihilism expressed in the preceding paragraphs, a detailed, concrete and positive program can be offered the patient suffering from intermittent claudication. On the basis of the pathologic physiology and the emotional responses referred to earlier in this presentation, every physician can effectively treat these patients without intricate diagnostic or therapeutic technics. The following description summarizes the approach currently used at the Beth Israel Hospital. Several illustrative cases are also included.

**Practical Management of Intermittent Claudication**

I. Establishing the Diagnosis

Many patients are referred with an incorrect diagnosis of claudication. It is important to remember that the diagnosis of claudication is made entirely by history; since the diagnosis carries considerable significance to the patient, it should not be accepted lightly. Fortunately, claudication is one of the most specific of symptoms. It is brought on only by continuous exercise and is relieved promptly by discontinuance of the exercise without change of position of the affected part. Furthermore, it is most unusual to be unable to demonstrate some impairment of the peripheral pulsations in these patients. Attention to these simple details will eliminate errors in diagnosis.

II. Determining the Ancillary Factors

Having established that intermittent claudication is present, one must next search for ancillary factors important in increasing the disparity between blood supply and demand. The most common of these are congestive failure, anemia, diabetes, and fresh arterial occlusion.

a. Congestive Failure and Anemia. The role of congestive heart failure and anemia in aggravating arterial insufficiency deserves special emphasis. Marked reduction in peripheral blood flow and vasoconstriction occur in advanced congestive heart failure. Reversal of these altered hemodynamics by successful treatment of the congestive heart failure is frequently accompanied by a decrease in the ischemia. In anemia the tissues are supplied with oxygen adequate for basal requirements, but the low tissue oxygen tensions result in a diminution of the margin of safety. Thus, in the presence of advanced occlusive disease, even a mild anemia may decrease this margin of safety sufficiently to tip the delicate balance between adequate nutrition and ischemia. Although it has been demonstrated that anemia may stimulate interarterial anastomoses; once clinical manifestations of claudication have developed in an extremity, ischemia will be decreased by cor-
rection rather than maintenance of the anemia. It is important, in this regard, to realize that the formed elements of the peripheral blood and bone marrow do not change strikingly as people grow older and that a mild anemia in elderly patients is not a “normal” finding.

b. Diabetes. The prominence of diabetes among patients with arterial occlusive disease and gangrene is impressive. If glucose tolerance curves are obtained on all patients with arterial occlusive disease and gangrene, the incidence of diabetes becomes even greater. The infrequency with which gangrene has developed in patients with normal glucose tolerance curves has been so striking among our patients with peripheral arterial obstruction that we have found this simple test to be of considerable value in predicting the clinical course of arterial insufficiency. The severity of the diabetic state is not related to the development of gangrene and in many patients the metabolic defect can be recognized only after examination of the blood sugar. Diabetes, therefore, should be suspected in every patient with peripheral atherosclerosis.

It is not difficult to understand why diabetes alters unfavorably the prognosis in intermittent claudication. The metabolic disorder probably accelerates the atherosclerotic process itself. It is, thus, associated not only with peripheral atherosclerosis but also with coronary atherosclerosis and resultant congestive heart failure. Diabetes produces retinal and peripheral neuropathy and accelerates cerebral atherosclerosis: the former impairs the patient’s awareness of local trauma; the latter, with its attendant mental changes, interferes with the patient’s ability to insure good care of his feet. Finally, resistance to infection may be impaired in diabetic patients.

c. Fresh Arterial Occlusion. A fresh embolic or thrombotic arterial occlusion may be associated with the onset of intermittent claudication or may cause a sudden decreased walking tolerance in patients with established claudication. Factors such as hypotension and polycythemia which favor the development of intravascular thrombosis therefore require prompt and effective correction. In every patient in whom a fresh arterial occlusion led to gangrene at the Beth Israel Hospital during the past five years, extensive propagation of the clot was found. Since one cannot predict in which patient extension of a fresh occlusion will occur, we have considered as medical emergencies all patients with fresh arterial occlusion and have treated them promptly with anticoagulant drugs.

At the present time an ideal anticoagulant is not available. There is some experimental and clinical evidence to indicate that substances such as Dicumarol which depress prothrombin conversion may not provide optimal anticoagulant activity. At present heparin is considered by us the drug of choice in the short-term treatment of acute arterial occlusion. Recently, a new method has been described for the intermittent intravenous administration of heparin without repeated venipunctures. The technique is based on the use of inlying polyethylene catheters attached to rubber-capped Tuohy-Borst adapters. Catheters have remained in veins for as long as four weeks without the development of ascending thrombophlebitis or systemic reactions. The method has been used successfully in the prophylaxis and treatment of thromboembolism in patients with a variety of underlying pathologic states. Patients with acute myocardial infarction were treated with heparin without greater inconvenience than is incurred with Dicumarol controlled by a daily prothrombin time.

d. Additional Factors. With surprising frequency, patients with claudication are more seriously disabled by an unrecognized coexisting disability than by their arterial insufficiency. Night cramps abolished by quinine or pyribenzamine, flat feet corrected by arch supports, the postphlebitic syndrome ameliorated by elastic bandages, chronic gouty arthritis relieved by uricosuric agents may so improve a patient’s walking tolerance that the discomfort from the claudication alone is no longer disabling. If some such ancillary disability is diligently searched for in each patient with claudication, the therapeutic yield will be very gratifying.

A positive program also requires a re-evaluation of the medical profession’s attitude concerning smoking. The continued use of
tobacco by patients with intermittent claudication is still strongly interdicted by many authorities. The fact that tobacco smoking increases the tone of peripheral vessels is adequate justification for urging all patients with acute arterial insufficiency to stop smoking, at least until the imbalance between obstruction and collateral flow has been improved. It is equally justifiable on the basis of a substantial body of clinical data to urge all patients with thromboangiitis obliterans to abstain from tobacco. However, for the large group of ambulatory patients with intermittent claudication from atherosclerosis the need for such restriction is not so clear. There is no evidence that tobacco, any more than mildly cold weather, adversely affects the course of the disease. Some investigators have been unable to demonstrate any deleterious effects of tobacco on intermittent claudication due to atherosclerosis. Others have permitted patients to smoke a few cigarettes daily without apparent harm. Tobacco smoking in many of these individuals is a well entrenched habit. For them abstinence may be neither necessary from the organic point of view nor desirable for emotional reasons.

Although there are cogent nonvascular reasons for discouraging the use of cigarettes, we no longer insist that patients with stable intermittent claudication secondary to atherosclerosis discontinue tobacco as part of the therapy of their peripheral arterial insufficiency.

III. Assessing the Disability

Having established the accuracy of the diagnosis and having satisfied oneself that all potentially related disorders have been adequately explored and treated, one comes to the third aspect of management: evaluation of the disability. At the outset, one must recognize and deal with the psychological hazards induced in the patient by the diagnosis itself. The significance of the disability to the patient is perhaps the most important limitation imposed by the syndrome. Failure to meet this problem squarely, particularly since a quick cure cannot be affected by drugs or surgery, will result in failure of treatment, loss of the patient or both. The fact that claudication will usually improve, or at least remain stationary and will not bar him from gainful employment should be clearly explained to the patient. This assurance will be reinforced when the patient observes that if he reduces his speed, he can walk further before experiencing pain. Many patients have developed a pattern of rapid walking and it may be necessary to reeducate them to walk more slowly. Finally, if the patient has a normal glucose tolerance test, one can use this finding to reassure him with regard to his fears concerning amputation.

IV. Walking Program

Almost routinely a walking program is recommended to each patient. The patient is urged to walk to pain tolerance at least four times daily. To present such therapy to a patient effectively, the physician must be convinced that the virtues of the plan are real. Walking to pain tolerance provides the most physiologic stimulus yet devised for the development of a collateral circulation in those areas where it is most needed, namely, the ischemic zone. Second, it offers the patient a specific program which is simple and completely safe. Gangrene of a toe cannot be induced by excessive walking, per se. Third, walking to pain tolerance has a most salutary effect on that large group of patients who, prior to seeing a physician, had voluntarily limited their activity to avoid pain. Invariably, these patients had assumed that the pain itself was in some way harmful or undesirable. Finally, the walking routine promptly acquaints the patient with improvement as it develops: a blood count or an oscillometric reading are not needed to convince him or to suggest to him that he is better.

V. Foot Care

In approximately half of the patients developing gangrene, a history is obtained of avoidable trauma, unnecessary minor surgery of the toes or the injudicious use of various local applications. Experience with ischemic extremities has repeatedly demonstrated that patients with extensive occlusive disease may be protected from gangrene by diligent care of
the feet. Conversely, in the presence of proved minimal occlusive disease a severe neglected insult to the leg may result in extensive gangrene even in the presence of pedal pulsations. It is this knowledge which fully justifies the effort involved in acquainting each patient with the minutiae of good foot care. This is particularly important among patients with diabetic neuropathy or impaired vision.

The following type of instructions may be profitably given to each patient with intermittent claudication.

(a) Avoid Temperature Extremes. Do not expose the feet to cold water or low temperatures that may produce frostbite. Do not bathe feet in hot water and avoid exposure of the legs to hot water bottles, electric pads, sun or ultraviolet light. The complaint of cold feet at night can frequently be relieved with warm socks and blankets rather than by the direct application of heat.

(b) Avoid Mechanical Trauma. Toenails must be trimmed carefully to avoid nicking the skin and cut straight across to prevent ingrowth of the nail. Rough nail edges or nail splitting can be avoided by soaking the toes in warm (not hot) water prior to cutting. If vision is poor or for any other reason difficulty is encountered in cutting the nails, they should be trimmed by a member of the family or a qualified chiropodist. Patients are advised that it is extravagant to save money on shoes. Shoes must fit well, be made of relatively soft leather, and be broken in slowly to avoid blisters. Coarse socks are undesirable because they tend to wrinkle. Corns and callouses can usually be traced to inadequate shoes or poor metatarsal arches. If these defects are corrected the thickened layers of skin will gradually soften and can be gently removed. If chiropody is necessary, the chiropodist should be informed of the vascular problem. The patient is instructed never to go about the house without slippers or walk barefoot at a beach, lest injury be incurred from some unsuspected object on the ground. Crowded places where the feet may be injured also demand special care on the part of the patient.

(c) Local Hygiene. The feet must be kept clean by frequent bathing (at least every other day). The skin must be thoroughly dried with a towel after bathing and then powdered with plain, nonmedicated talc. If feet perspire freely, a change of socks preceded by powdering of the feet should be carried out twice daily. In addition to cleanliness and dryness, it is imperative that the skin be kept soft and pliable so that it remains free of cracks which can lead to gangrene. The application of a very thin layer of lanolin to the feet nightly is very effective in this regard. The feet may be covered with cotton or wool socks to avoid soiling the bed sheets. Excess lanolin should always be removed with cotton before applying the sock. Prevention of fungus infection can be achieved by keeping the feet dry and avoiding sources of contamination such as public baths, showers and swimming pools. Finally, no medicine or chemical should be applied to the foot unless prescribed by a physician.

In general, bland powders, lanolin, and certain nonsensitizing antibiotic ointments (for the treatment of local infections on advice of a physician) are the only substances that may need to be applied to the skin of the ischemic extremity.

(d) Injury. Minor injuries, particularly those involving a break in the skin, justify a call to a physician. When in doubt, bed rest and exposure of the foot to room temperature without the weight of bed clothes is the safest treatment until the doctor arrives.

Gangrene rarely appears de novo even in the limb with extensive old arterial obstruction. It usually develops because of the additional burdens produced by fresh arterial occlusion, diabetes, congestive heart failure, anemia, trauma and infection. At the present time, diagnostic and therapeutic measures are readily available to meet these major threats to the viability of the ischemic limb.

Case Reports

Case 1. Fresh Femoral Artery Occlusion, Producing Intermittent Claudication, Treated Effectively with Anticoagulants. Gradual Spontaneous Decrease in Claudication During Subsequent Twelve Months

J. L., a 46 year old stock room clerk, was hospitalized because of the recent onset of disabling leg
pain. Five days before entry, while walking down a corridor, the patient noticed a sudden, severe, dull ache in the right calf which disappeared with cessation of walking. Until admission, symptoms of aching, numbness and tingling persisted in the right leg. Fifteen to 20 steps at his normal rate of speed precipitated the pain in the right calf. Pressure of the sole of the right foot against the car accelerator produced numbness of the foot and calf.

Angina pectoris, dramatically improved by radioactive iodine (I$^{131}$) therapy, had been present for six years. There was knowledge of allergy to various foods and drugs manifested by urticaria but no history of hypertension, myocardial infarction or diabetes.

Significant findings on examination were restricted to the lower extremities. The right foot was cooler to palpation than the left to a level of six inches above the malleoli. Only the femoral pulse was palpable on the right; all pulses were palpable in the left lower extremity. There was also, in the right leg, definite calf tenderness and a positive Homans' sign but no edema.

Urinalysis, blood count, electrocardiogram, chest x-ray and a glucose tolerance test were all within normal limits.

The patient was put to bed and received intravenous heparin every six hours for nine days. At this time, he developed urticaria immediately following each of two intravenous doses of heparin and the drug was discontinued. During the first week the temperature demarcation receded from a level six inches above the malleoli down to the toes. The patient was ambulated without further anticoagulant therapy and gradually permitted to increase his activity. Following the first day of heparin therapy calf tenderness and the positive Homans' sign disappeared.

Prior to discharge from the hospital a walking program was outlined for the patient and he was instructed concerning foot care. During the 12 months following discharge the patient's walking limitation from his claudication, though still present, had receded to a point where it no longer interfered with his work. None of the absent pulses returned.

Comment. In this patient the deposition of a fresh femoral artery occlusion manifested itself as intermittent claudication. In view of the cardiac history and the excellent pulses in the contralateral extremity, it is likely that the occlusion was embolic in origin. Immediate anticoagulant therapy was instituted to prevent the growth of the arterial clot, to retard the deposition of thrombi on atheromatous plaques distal to the fresh occlusion and finally to prevent the development of a pulmonary embolus secondary to the venous thrombosis which was obviously present in the patient's right leg on entry. The rather dramatic and progressive improvement in the claudication simply reflects the growth of an effective collateral circulation within several months unaided by any special therapeutic procedure other than repeated walking to the limit of pain tolerance. This case is typical of the spontaneous improvement in walking tolerance that continues to occur for many months after the onset of claudication. If this patient had had a sympathectomy, an embolecctomy or a segmental arterial resection, the favorable result might readily have been credited to the surgical intervention.

Case 2. Intermittent Claudication, Two Years. Marked Improvement with Walking Exercises. Patient Never Stopped Smoking

F. S., a 42-year-old businessman, came for advice because a sympathectomy had been recommended for the treatment of his intermittent claudication. Four months earlier, the patient noted the onset of tingling of the toes of the right foot while running for a train. Tingling and numbness in the toes of the right foot recurred whenever he ran but subsided promptly when the activity was stopped. One month later, on walking to work, the patient noticed that at the completion of a quarter mile distance he would develop a numbness in the right foot. The paresthesia was relieved by cessation of walking without sitting down; it never occurred while sitting or reclining.

Because of these symptoms the patient consulted an orthopedist who placed pads in the patient's shoes. This resulted only in a shift in the paresthesias from the toes to the longitudinal arch of the foot.

Examination of the feet revealed that only the dorsalis pedis pulsation was absent on the left. On the right the femoral and popliteal pulsations were full and bounding, the posterior tibial pulsation was present but markedly diminished and the dorsalis pedis pulsation was absent. Oscillometric readings were: left forearm, 6 units; left calf, 2 units, right calf, 1½ unit. There was no true pallor on elevation or delayed rubor on dependency. Hair was present on the lower leg; there was no edema or nutritional change except that the skin over the dorsum of the right foot was dry and thickened.

Urine, blood count, electrocardiogram and a glucose tolerance test were all normal.

Because of considerable anxiety the prognosis and significance of intermittent claudication was carefully explained to the patient. The patient was instructed in foot care and in walking exercises. The nightly use of lanolin promptly softened the ker-
totically areas on the right foot. The patient who used one pack of cigarettes daily, was permitted to continue smoking. During the next 18 months, despite no change in the arterial pulsations or oscillometric readings, there had been a more than fourfold increase in the patient's walking tolerance.

**Comment.** This patient showed marked improvement in his claudication within an 18 month period despite continuance of cigarette smoking and with no therapy other than walking exercises. This case again illustrates the spontaneous improvement usually continuing for many months after the onset of claudication. The oscillometric findings added no information not already available from palpation of the pulses. The mechanical alterations in posture produced by foot pads were of interest in that they changed the muscles which developed the greatest ischemia so that the patient's symptoms were shifted from one area of his foot to another.

The extent of the arterial obstruction and the degree of the disability from claudication in this patient were very mild. The major problem was not the physical limitation per se but rather the fear of amputation and the concern over spread of the occlusive process to other vital areas. Permission to smoke, insistence on walking to tolerance, discussion of the significance of the normal glucose tolerance test, the negative electrocardiogram and the normal fundoscopic findings when coupled with improved walking tolerance without benefit of sympathectomy all contributed toward giving this patient considerable reassurance.

**Case 3. Intermittent Claudication Five Years. Nocturnal Paresthesias Four Years, Incapacitating for One Month. Paresthesias Completely Relieved by Pyribenzamine With Return to Gainful Employment. No Change in Intermittent Claudication. Striking Therapeutic Result For Seventeen Months**

J. B., a 50 year old chauffeur, was referred for advice concerning the use of sympatholytic agents in the treatment of his arterial insufficiency. Five years previously, the patient first noted pain in the right calf after walking five city blocks. The pain was relieved after five minutes by cessation of walking without sitting down. Beginning four years previously, the patient found that he was frequently awakened from sleep by a “burning” or a “pins-and-needles” sensation in his feet. He tried unsuccessfully to relieve these paresthesias by a number of maneuvers, including placing the leg in hot water. The paresthesias were diminished only by hanging the foot over the side of the bed. These sensory disturbances, which had been interpreted as reflecting advanced arterial insufficiency, had become so severe and frequent during the four weeks prior to referral that the patient was fatigued, irritable from lack of sleep and unable to work.

The patient had smoked 30 cigarettes daily for years. There was no history of diabetes or hypertension.

The significant findings on physical examination were restricted to the lower extremities. No pulses were palpable in either leg below the femoral triangle. The oscillometric reading at the right calf was zero and at the left calf was 14 unit. There was marked pallor on elevation bilaterally; on dependency, rubor was delayed 25 seconds in the right leg compared with 15 seconds in the left extremity (normal less than 10 seconds) and the right foot was of a deeper hue than the left after two minutes of dependency. There was no hair below the knees bilaterally, but skin nutrition was otherwise good. In addition, varicose veins, slight ankle edema and pruritic scaly lesions between the toes were present in the right lower extremity.

Laboratory studies, including a urinalysis, blood count and a glucose tolerance test, were all within normal limits.

The disability from claudication was explained, a walking program outlined, and the patient instructed in proper foot care, including the avoidance of hot water. Potassium permanganate soaks were prescribed for the trichophytosis and quinine was given for the nocturnal paresthesias. The pruritic interdigital lesions disappeared with therapy. When no improvement resulted from the quinine, Pyribenzamine, 50 mg. at bedtime, was substituted with prompt relief of the paresthesias to the point where sleep was no longer interrupted. Temporary cessation of Pyribenzamine resulted in recurrence of paresthesias. This patient has now been followed for 17 months. He has continued uninterrupted at work and is free of paresthesias at night. He still has claudication on walking 5 to 10 blocks, depending on his rate of speed but no longer finds this a significant disability.

**Comment.** In this patient with severe arterial insufficiency, nocturnal paresthesias, rather than claudication, was the limiting disability. Relief of the night pains by Pyribenzamine restored this patient to gainful employment.
No change in claudication was observed over a 17-month period during which time the patient abstained completely from tobacco. This case is typical of the lack of improvement in exercise tolerance after claudication has been present for several years. The patient did learn, however, that he could control the onset of claudication by his speed of walking. It was important in this instance to realize that the paresthesias did not represent an ischemic neuritis for which, at present, there is no satisfactory treatment. No further therapy for the intermittent claudication is contemplated. There is certainly no indication for the use of sympatholytic agents. Elastic stockings were not prescribed for the venous insufficiency because of the presence of advanced arterial occlusive disease.

Case 4. Intermittent Claudication Four Years. Infection in Weight Bearing Area of Congenitally Clubbed Foot. Amputation Avoided by Conservative Measures. Previously Unsuspected Diabetes Mellitus Recognized by Glucose Tolerance Test

M. G., a 70 year old shoe repairman, was referred for a possible amputation of the left leg. The patient had had crampy pain in the right calf on walking 5 to 10 blocks for four years. He had a left congenital clubfoot for which he had never received treatment. The extremity had been essentially asymptomatic until two weeks previously when a painful swelling followed by a small ulcer developed over the weight bearing inferior aspect of the left foot. Because of the vascular disease, the severe deformity of the foot and the presence of infection a supracondylar amputation had been recommended to the patient.

On two occasions in the past two years the patient had been hospitalized for episodes of acute myocardial infarction. During these illnesses both femoral and popliteal pulsations were readily palpable but the pedal pulses in both legs were absent. There had been no evidence of congestive failure, arrhythmia, anemia or glycosuria. A fasting blood sugar had been normal. Claudication had become more severe since the second myocardial infarction one year ago.

Examination revealed a left club foot. The femoral artery pulsations were palpable bilaterally. The popliteal and pedal pulses were absent. A painful tender swelling surrounding a small superficial ulcer was present over the lateral aspect of the cuboid bone. There was minimal surrounding cellulitis. The infection was treated locally with bacitracin ointment and bed rest. Subsequently, crutches were used to eliminate weight bearing on the left foot. The ulcer healed gradually over a four-week period. After several days of a high carbohydrate diet, a modified glucose tolerance test revealed that the venous blood sugars (Folin-Wu) one hour and two and one-half hours after the ingestion of 100 Gm. of glucose were 235 and 200 mg. per 100 cc., respectively. On the basis of this finding a diagnosis of diabetes mellitus was made. The patient was carefully instructed as to his diet and proper foot care. A special shoe was obtained and adjusted to prevent weight bearing on the lateral aspect of the foot. Subsequently, the patient was given walking exercises and the past 12 months has been ambulatory and free of any difficulty with the left leg. The patient has adhered to his diabetic diet and the blood sugar levels have returned to normal values. Claudication in the right foot has remained unchanged.

Comment. Many factors favoring a decision to amputate were present in this patient: advanced age, two myocardial infarcts, absence of pulses below the femoral triangle, marked deformity of the foot, infection and diabetes. The infection, however, had been precipitated by a specific type of trauma which it was believed could be remedied by the use of a special shoe. The notorious risks of prolonged bed rest were avoided—once the lesion responded to antibiotics—by ambulation with the aid of crutches. Progression of the peripheral arterial insufficiency since the second myocardial infarct was manifested by loss of the popliteal pulsation, by aggravation of the claudication and by the inability of the skin to withstand an amount of trauma previously tolerated. Yet collateral circulation was adequate to permit healing of the ulcer when infection was controlled and trauma removed. The claudication in the contralateral extremity was not a disabling factor. The glucose tolerance test permitted the recognition and treatment of previously unrecognized diabetes mellitus. This case emphasizes that a negative urinalysis and a normal fasting blood sugar do not rule out a diagnosis of diabetes. Although this patient was saved from a major amputation, many threats to the viability of the extremities remain. Constant attention to the small details of good foot care will always be necessary.
SUMMARY

The diagnosis and therapy of intermittent claudication are critically reviewed. A detailed, concrete and positive program is described whereby every physician can effectively treat patients with intermittent claudication without recourse to intricate diagnostic or therapeutic techniques. The value of this program is illustrated with four case reports.

REFERENCES
