Chronic Occlusive Arterial Disease of the Extremities

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Chronic occlusive arterial diseases are the most common and the most disabling of all peripheral vascular disorders. Some of the causes of chronic arterial occlusion are arteriosclerosis obliterans, thromboangiitis obliterans (Buerger's disease), primary arterial thrombosis, which presumably results from hypercoagulable blood, embolism, which is almost always associated with heart disease or peripheral arterial aneurysms, trauma, and ergotism. Of these, arteriosclerosis obliterans is by far the most frequently encountered. Although differing clinically and pathologically, all of the diseases are similar in that they cause ischemia of tissues supplied by the occluded arteries. The degree of ischemia is directly proportional to the rapidity of development and the extent of occlusion and is inversely proportional to the extent and rapidity of development of collateral arterial anastomoses. The ischemia may be increased by arteriolar constriction and decreased by arteriolar dilatation from any cause. When blood flow diminishes to the point where tissue does not receive sufficient oxygen to sustain life, or when injury or infection increases the demand for oxygen that the occluded arteries are unable to supply, gangrene occurs.

It is beyond the scope of this presentation to discuss in detail the distinguishing features of the various chronic occlusive arterial diseases. Although much of what follows pertains to all or some of these diseases, most of it deals more specifically with thromboangiitis obliterans (TAO)* and with arteriosclerosis obliterans (ASO).* Recently the concept of TAO as a diagnostic entity has been vigorously challenged and defended on both clinical and pathologic grounds. It has been contended that TAO is due in fact to atherosclerosis, arterial embolism, or arterial thrombosis, or to a combination of all three conditions. Most experienced students of peripheral vascular diseases, however, agree with McKusick and associates that TAO is a distinct clinical and pathologic entity. Recent evidence strongly supports the usefulness of distinguishing between TAO and ASO because of the marked differences in prognosis of patients affected with either disease. Some of the distinguishing clinical features of these two diseases are set forth in Table 1.

Symptoms

The symptoms of chronic occlusive arterial disease result from impairment of blood flow to the extremities. The symptoms may appear gradually as a result of slowly progressive occlusion or suddenly as a result of acute arterial thrombosis or embolism. They may progress episodically or remain relatively static for many years. The outstanding symptom of chronic occlusive arterial disease is pain that may take several forms.

Intermittent claudication is the commonest and usually the earliest symptom. This is a pain, ache, cramp, or severe fatigue that affects muscles distal to the occluded artery when these muscles are exercised. Rest without a change of position or cessation of weight bearing promptly relieves the distress. In cases of ASO, intermittent claudication practically always involves one or both lower extremities, most commonly the calf, but in some patients it is noted in the foot, thigh, or hip alone or in association with the calf. During any stage of the disease intermittent claudication can usually be reproduced consistently after a certain distance of walking at a certain rate. A progressive or sudden decrease in the claudication distance, that is,
the distance that a person affected can walk before onset of claudication, indicates an increase in the arterial insufficiency of the affected limb. When intermittent claudication develops for the first time in a patient more than 40 years old, it is usually due to ASO.

Ischemic rest pain, located in the distal part of the extremity, may be severe and is usually worse at night. It indicates a serious degree of arterial insufficiency. Numbness and paresthesia of the toes or foot are often precursors of rest pain and indicate moderately severe arterial insufficiency. The patient may notice increased sensitivity of the affected extremity to cold.

The pain of ulceration and gangrene is similar to ischemic rest pain, although it is usually more localized and probably results from inflammatory changes associated with infarction, secondary infection, and ischemia of sensory nerve endings.

The pain of ischemic neuropathy is usually due to extensive chronic or sudden occlusion of an iliac or femoral artery. The pain extends over a large portion of the extremity and may follow the sensory distribution of a large nerve trunk. It is paroxysmal, shock-like, shooting or cutting and may be associated with burning sensations and paresthesia. The pain is frequently severe and difficult to relieve.

**Physical Signs**

Significant occlusive arterial disease of the extremities may be diagnosed before the onset of symptoms by routine determination of pulsations of peripheral arteries at every examination. The examination should include careful and unhurried palpation of the radial and ulnar arteries at the wrists, the abdominal aorta, and the femoral, popliteal, posterior tibial, and dorsalis pedis arteries. Absence or reduction in the amplitude of pulsation in any of these arteries except the dorsalis pedis or possibly the posterior tibial arteries usually indicates occlusive arterial disease. It should be kept in mind, however, that ana-
tonic anomalies may account for the symmetrical absence of pulsation in the posterior tibial arteries in as many as 8 per cent and in the dorsalis pedis arteries in as many as 15 per cent of normal people.9 The amplitude of pulsation in each artery may be graded from 0 (absent) to 4 (normal). Such systematic recording of pulsations allows a relatively accurate evaluation of the arterial circulation at a given time and is of value in assessing progression of the disease process.

Elevation and dependency tests allow an estimation of the cutaneous ischemia. With the patient in the supine position, the hips are flexed and the feet are elevated for approximately 30 to 60 seconds. This empties the visible superficial veins of the foot and causes varying degrees of pallor of the skin of the ischemic foot. The feet are then made dependent. The time required for normal color to return and for refilling of the emptied veins is noted. In occlusive arterial disease the return of color takes longer than 10 seconds and refilling of the veins takes longer than 15 seconds. These observations are invalid when venous incompetence with retrograde venous filling is associated. Persistent rubor of the skin of the distal portion of the foot and toes is usually present when the arterial circulation is greatly impaired. Other indications of arterial insufficiency are coolness and atrophy of the distal portion of the affected extremity. Occlusion of a smaller digital artery may be manifested by a cyanotic, cool digit and occasionally by cutaneous infaracts. In cases of ASO, ischemic ulceration and gangrene usually are confined to the toes or heel; in cases of TAO, ulceration and gangrene may affect the finger tips also. The typical ischemic ulcer has a grey-black base with little or no granulation tissue; concomitant local infection often is present; the most frequent organism is Staphylococcus aureus.

Laboratory and Special Studies

Relatively few laboratory data are needed to corroborate a clinical diagnosis of chronic occlusive arterial disease of the extremities. Plethysmography and skin-temperature studies are not necessary to establish a diagnosis or even to determine the best type of treatment. Although the oscillometer is still used by some physicians, it probably gives less reliable and less consistent information than careful assessment of the pulses by digital palpation.

All patients with occlusive arterial disease should be tested for hyperglycemia, since a large number of patients with ASO have latent or frank diabetes. Determinations of blood lipids may be advisable also, particularly for younger patients with occlusive arterial disease, since the concentration of cholesterol in the blood is elevated in about 50 per cent of men and 75 per cent of women with ASO who are less than 60 years of age.7 Diabetes mellitus or hyperlipidemia or both are rarely encountered in patients with TAO. Roentgenographic evidence of atheromatous calcification of the arteries of the pelvis or lower extremities occasionally may be useful in distinguishing ASO from TAO. Electrocardiograms are advisable in patients with ASO, whether or not symptoms of coronary heart disease exist, because of the frequency with which coronary sclerosis is associated with arteriosclerosis of the arteries of the extremities.

Arteriograms are almost never necessary in making a diagnosis of occlusive arterial disease of the extremities. Although arteriographic distinctions between ASO and TAO exist, the exact nature of the obstructing lesions cannot always be determined by this means. Arteriograms are frequently necessary, however, to determine the exact site and extent of the obstructing arterial lesions if arterial surgery is contemplated. Arteriography is rarely justifiable when the clinical findings indicate that the obstructing lesions are entirely distal to the popliteal arteries or when it is certain for other reasons that a direct surgical attack will not be made. In the absence of clinical evidence of involvement of the abdominal aorta or iliac arteries, the femoropopliteal system may be better visualized by percutaneous injection of contrast
medium into the common femoral artery on the affected side; otherwise an aortogram should be made. Both femoral arteriography and aortography carry a small but definite risk of serious local and systemic complications.

Prognosis

A recent study \(^4\) of patients who were 45 years of age or less when a diagnosis of chronic occlusive arterial disease of the extremities was first made indicated a practically normal life expectancy for those with TAO and a decreased life expectancy for those with ASO (fig. 1). Patients with ASO do not die because of the occlusive lesions in their peripheral arteries but rather because of the frequently associated arteriosclerotic lesions in their coronary and cerebral arteries. Since these patients do not die of ASO per se, surgical restoration of arterial blood flow to the extremities cannot be expected to alter their life expectancy.

The same study showed that the incidence of ulceration and the necessity for amputation of a lower extremity before or at the time of diagnosis and for 10 years thereafter was significantly lower among patients with ASO than among patients with TAO. None of the patients with ASO required an amputation of a finger, whereas 6.3 per cent of the patients with TAO required amputation of one or more fingers within the 10-year follow-up period. In another study \(^7\) on nondiabetic patients with ASO who were less than 60 years of age at the time of diagnosis, only 4 per cent required amputation of a leg shortly after the original examination and an additional 5 per cent required similar amputation during the follow-up period, which was a minimum of 5 years. If the patient discontinued smoking, the subsequent amputation rate was much lower than when use of tobacco was continued. The subsequent amputation rate among diabetic patients with ASO involving the femoral artery was approximately four to five times that of nondiabetic patients, and the diabetic patient who has ischemic gangrene has only about a 50 per cent chance of avoiding amputation of the involved leg.\(^8\)

Medical Measures

The general principles of medical treatment are the same for all types of chronic occlusive peripheral arterial disease, but treatment of the patient with a specific occlusive disease must be individualized. Control of associated conditions such as diabetes mellitus or polycythemia vera should not be neglected. Although there is no definite proof that the course of ASO is altered by decreasing elevated concentrations of lipids in the blood, an attempt should be made to control hyperlipidemia, preferably by dietary means.\(^9\)

The precipitating cause of many of the ulcerative or gangrenous lesions is trauma from mechanical, chemical, or thermal sources. All patients, therefore, should be given detailed instructions concerning the care of extremities and the avoidance of trauma. Minor wounds, contusions, scratches, application of heat, fungous infections, or surgical procedures around the nails may lead to local necrosis and secondary pyogenic infection. Strong antiseptic solutions or ointments should not be applied to the skin of ischemic extremities. Sloughs and crusts may be drained or loosened with soaks or warm wet dressings of normal saline or boric acid solution. Since most ulcerative or gangrenous

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**Figure 1**

Survival curves for patients with thromboangiitis obliterans (TAO) and arteriosclerosis obliterans (ASO) compared with that of a normal population (Adapted from McPherson, Juergens, and Gifford).\(^4\)

**Treatment**

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lesions are secondarily infected, appropriate systemic antibiotic therapy may be helpful in healing the lesions. Occasionally, careful surgical debridement of gangrenous tissue will facilitate healing, but this should be done only after conservative local and antibiotic treatment.

Repeated use of analgesics may be necessary for temporary relief of ischemic rest pain or the pain of ischemic neuropathy. Salicylates, d-propoxyphene hydrochloride (Darvon), or one of the narcotic alkaloids such as levorphanol tartrate (Levo-Dromoran) may be needed to control the pain, which is often severe. One of the phenothiazine tranquilizers may be helpful in alleviating anxiety often associated with severe pain. Narcotics should be used with caution because the chronicity of ischemic pain may lead to addiction.

Long-term anticoagulant treatment with one of the coumarin compounds is usually indicated for patients who have had more than one episode of sudden arterial occlusion whether this be due to embolism from chronic atrial fibrillation, primary arterial thrombosis, or thrombosis associated with ASO. These drugs should not be used, however, unless the patient is cooperative and facilities are available for accurate prothrombin-time determinations.

Tobacco smoking is very likely an important factor in the etiology of TAO and may be an important etiologic factor in ASO. Amputation is much less frequent among patients with either disease who discontinue smoking than among those who continue to smoke. In addition, smoking causes peripheral vasoconstriction in almost all persons. For these reasons the physician should urge complete and permanent abstinence from tobacco for all patients with chronic occlusive peripheral arterial disease.

In addition to abstaining from tobacco, patients with occlusive arterial disease should avoid vasoconstricting drugs and exposure to cold. A warm environmental temperature may be of value in producing reflex vasodilatation. An ounce of whiskey or brandy given three or four times daily is a simple and relatively effective type of medication when ischemic rest pain is present and the need for vasodilatation is critical. Although there is physiologic evidence that the numerous vasodilating drugs currently available cause vasodilatation in normal extremities, the evidence for significant vasodilatation in extremities affected by occlusive arterial disease is not impressive. Papaverine hydrochloride and tolazoline hydrochloride (Priscoline) may be of value when given intravenously or intra-arterially in the treatment of an acute arterial occlusion, but these and other drugs appear to be of little value in chronic occlusive arterial disease.

**Surgical Measures**

Regional sympathetic ganglionectomy is probably the most effective method of producing maximal and persistent vasodilatation. This procedure is indicated when an increase of the circulation to an ischemic hand or foot is needed, in the latter case particularly if a direct surgical attack on the arteries is technically impossible or contraindicated. It does not cure the basic disease process and after such operations there is still need for avoidance of trauma and tobacco. It is doubtful that intermittent claudication is ever much improved by sympathectomy but the persistent vasodilatation probably offers some protection against further ischemic skin complications. Sympathectomy gives the best results in patients with moderate ischemia of the feet or hands with minimal or no skin lesions, or with only minor degrees of ischemic neuropathy. In some patients with extremely severe ischemia, sympathectomy may precipitate gangrene, the so-called paradoxical effect. Destruction of the sympathetic ganglia by percutaneous injection of absolute alcohol may be of value when ganglionectomy seems indicated but the risks of such a procedure are deemed prohibitive. An alcohol block is less permanent than surgical ganglionectomy; it should not be attempted by anyone who is not skilled in this type of therapy.

Surgical restoration of arterial continuity may be accomplished by resection of a chronically obstructed segment and insertion of
a graft, insertion of a prosthetic tube to bypass an occluded segment, or thromboendarterectomy. The last procedure is being used with increasing frequency and occasionally a patch graft of plastic material or autogenous vein is sutured in at the same time to enlarge the lumen. Bypassing procedures are used when the occlusive lesion is too extensive for thromboendarterectomy. Reconstructive arterial surgery is applicable for occlusions that are proximal to the trifurcation of the popliteal artery, and operability depends on the occlusion being segmental, that is, there must be an adequate arterial lumen above and below the occlusion. Reconstructive arterial surgery for chronic occlusive peripheral arterial disease is practically limited to ASO involving the abdominal aorta, iliac, and femoral arteries. Arteriosclerotic occlusion of the subclavian or brachial arteries rarely produces severe ischemic symptoms and TAO almost always involves the smaller distal arteries, which are inoperable for technical reasons.

Direct arterial operation in nondiabetic patients is chiefly indicated for disabling intermittent claudication. As mentioned previously, this type of surgical procedure is not lifesaving because patients rarely die as a result of ischemia of tissue distal to the arterial obstruction. If the disease is progressing rapidly or if amputation seems inevitable, an arterial operation is justified in the hope that amputation can be avoided or can be made at a lower level. This type of procedure should not be employed merely because an arterial lesion is present, and it cannot be recommended as a routine prophylactic measure for preservation of an affected limb, since the incidence of gangrene is relatively low in the usual uncomplicated case of ASO. In selecting patients for arterial reconstructive surgical procedures, consideration must be given to the fact that postoperative occlusion of an inserted graft or an endarterectomized segment, particularly in the femoropopliteal region, may occur even if the surgery has been performed by experienced vascular surgeons.

Unfortunately, amputation of a finger is sometimes necessary in cases of TAO and amputation of a lower extremity is sometimes necessary in cases to TAO and of ASO. If gangrene is extensive and there is evidence of toxemia, amputation should not be delayed. When there is severe and intractable ischemic rest pain without gangrene, amputation may be necessary if a thorough trial (at least 6 weeks) of medical or of other surgical treatment fails to control the pain. It is frequently advisable to administer antibiotics before and after the amputation.

References
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