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John Homans

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CIRCULATORY DEFICIENCY IN THE EXTREMITIES IN RELATION TO MEDICO-LEGAL PROBLEMS*

DR. JOHN HOMANS†

INTRODUCTION

The diseases to be described are able to reach their full development in the absence of injury, and often do so. As a rule, injury is an aggravating factor, hastening the development of the disorder—precipitating it in some instances—bringing out a latent condition. But occasionally, as in the case of thrombosis of blood within a blood vessel, a blow, fall or strain may appear, in any one instance, to be the sole recognizable cause of the disease. From a medical point of view, the distinction between an aggravation and a basic cause is significant. From the medico-legal standpoint, it is less so. In workman's compensation cases, for example, the insurer is equally liable, whether a circulatory disorder is aggravated or the individual breaks a bone. So far as possible, in the following account of various circulatory diseases, it will be pointed out how injury may affect them and under what circumstances it is most likely to do so. But medical conceptions are far from fixed and, very often, authoritative pronouncements are not available.

CIRCULATORY DEFICIENCY IN THE EXTREMITIES

Arteriosclerotic Arterial Deficiency

The two varieties of chronic circulatory deficiency, due respectively to arteriosclerosis (hardening of the arteries) and thrombo-angiitis obliterans (Buerger's Disease) have much in common. Arteriosclerosis occurs in the elderly as a result of the wear and tear of life. Thrombo-angiitis obliterans, which is of unknown origin, attacks a far younger group. But both diseases cause a serious deficiency in the supply of arterial blood in the affected limbs. The upper extremities, though actually subject to both diseases, are rarely so changed as to offer a clinical or medico-legal problem. The lower limbs are the ones seriously affected, and the descriptions which follow concern only them. Much of what is said, under the heading of Arteriosclerotic Arterial Deficiency, of the nature of a defective arterial circulation, applies to Buerger's Disease as well: for example, the tests for arterial deficiency and the

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† Clinical Professor of Surgery, Emeritus, Harvard Medical School.

reaction to injury. To avoid repetition, a reasonably complete account of these subjects is given under Arteriosclerotic Arterial Deficiency, and allusion is made to this in the description of Thrombo-angiitis Obliterans. Thus the account of the two diseases should be read together.

Pathology.—Both large and medium sized arteries are hardened by arteriosclerosis. Calcium salts are deposited in their walls, making them still more inelastic and causing them to cast a shadow in the X-ray plate. They are narrowed in a patchy manner by the formation of mural thrombi within them. This erratic narrowing, by restricting the arterial supply of blood, leads to ill-nourishment of the tissue which they serve, affecting especially the toes. The ill-nourishment is seldom equally distributed, as between two limbs and even as between toes, muscles and skin. It causes shrinkage of the tissues, that is, the skin, fat, and muscles, tending to make them inelastic and even fibrous. They cease to react in a normal way to injury and infection. But they need exhibit no obvious change in appearance. Such changes as are present are usually irreversible, though their further development is often checked by the establishment of alternate routes for the blood stream, the opening of a "collateral" circulation on a large or small scale.

When a more complete arterial deficiency is added by a thrombosis^{1*} which actually obstructs one or several vessels, then, more or less rapidly, death of some terminal part of a limb occurs—toes, foot, even lower leg—a change known as *necrosis* or *gangrene*. Necrotic parts are especially likely to be the scene of infection which often spreads to nearby tissues. But under favorable circumstances the gangrenous part is gradually isolated; a line of *demarcation* separates it from the adjacent living tissue and, after weeks or even months, it is finally cast off. Then healing may spontaneously occur.

Symptoms.—Premonitory symptoms, if any, are coldness and pallor

^{1*} The term, *thrombosis*, applies to a biological process which may occur within both arteries and veins. Certain elements (platelets) of the blood, becoming adherent to the wall of the vessel, are agglutinated into a framework in which the red and white cells and newly formed fibrin become entangled. The resulting firm mass grows by a process of secondary coagulation and, if it plugs the vessel, may spread along it for a considerable distance, becoming softer and more clot-like as it grows. Scientifically speaking, thrombosis thus differs from *coagulation* or *clotting*, which only occurs within blood vessels after death, or outside the body, or as a process altogether secondary to thrombosis (as just explained). To laymen, and indeed to many medical men, thrombosis and clotting mean the same thing.

A thrombus may be "mural," merely narrowing without obstructing, a blood vessel, or it may obstruct it for a considerable distance, growing on, as a rule, until it meets the vigorous current of an entering branch, when it is likely to become organized (invaded by fibrous tissue).

The cause of thrombosis is presumably a combination of circumstances among which injury, disease of the vessel wall and slowing of the blood stream are especially important. So far as the arteries are concerned, arteriosclerosis decidedly exposes them to thrombosis (and, as will presently be seen, thrombo-angiitis obliterans does also). Hence the familiar narrowing by mural thrombi and the occasional plugging of arteriosclerotic vessels.

of the feet and lower legs, moderate atrophy (shrinkage) of the soft parts and a peculiar lameness on walking. Known as "intermittent claudication" or "intermittent limp," this lameness, in the form of a cramp-like pain, sets in regularly when the individual has covered a certain distance at a certain pace. It is relieved in a few minutes by rest but returns upon the same amount of exercise. Evidently it is related to a deficient blood supply of the muscles, perhaps to an unnatural accumulation of waste products.

A more advanced stage is marked by a change toward a deep red or blueish-red color of the toes on dependency, by spontaneous pain in the discolored parts, especially on elevation to the horizontal or higher, and by the appearance of sores or actually gangrenous (necrotic) areas beside the toenails, on the tips of the toes or over a bony prominence such as the great toe-joint. This advance in the disease is usually, though not invariably, irreversible. Unless favorably influenced by treatment, it is the precursor of a serious gangrene.

In diabetics, the same events occur, but at a slightly earlier age, (since diabetes brings on arteriosclerosis) and, in addition, small joints often become infected and small bones necrotic to a degree that the deficiency in the circulation does not seem to warrant. These infected or necrotic bones or joints, without superficial gangrene, are usually painless. Resistance to infection or injury is even feebler than in uncomplicated arteriosclerosis.

A very advanced stage is marked by gangrene of toes and feet, by infection spreading into joints and upward by way of the lymphatics^{2*} (lymphangitis), by way of the subcutaneous tissues (cellulitis) and along the tendons and muscles. Such states, unless checked by amputation, are often fatal. A very sudden arterial thrombosis, closing a large vessel, may cause a rapid, extensive gangrene, with prostration and even death.

THE COURSE OF ARTERIOSCLEROTIC ARTERIAL DEFICIENCY is usually slow. In the absence of injury, of infection and of serious exposure to cold, a mild arterial deficiency is compatible with restricted or even

^{2*} The lymphatics are very fine, delicate vessels which collect the tissue fluid, all over the body, and empty them into the great vein (vena cava) just before it enters the heart. The lymphatics of each limb pass through a group of lymph-nodes, or glands, which act as filters and, by the activity of their abundant cells, free the lymph from bacteria or other noxious material carried in it. Thus the lymphatics have an important defensive function in freeing the tissues of organisms hostile to the body. But in carrying out the vital function, the lymphatics themselves often become inflamed, a condition betrayed by red streaks visible in the skin and leading from the infected focus in the toes or foot toward the root of the limb. Such inflammation is called *lymphangitis* and the secondary inflammation of the lymph-nodes is called *lymphadenitis*. Lymphangitis is always a danger signal, indicating that infection is out of local control in the region where it has started. And if the lymph-nodes fail to put up a successful defense, bacteria will enter the general circulation.

relatively normal activity for many years. An individual may be unaware of any disorder, though subject to the bad effects of any of these accidents. He or she would be most apt to notice cold feet, lessened vigor of locomotion (or even intermittent claudication) and pallor with reduced size of the legs.

DIAGNOSIS.—When invoked as a cause of a circulatory disorder, a diagnosis of arteriosclerosis should be looked upon with doubt if made upon individuals under sixty years of age, though undoubtedly the disease occasionally occurs at an earlier age, and especially, as already explained, in diabetics. But there is no definite clinical picture of arteriosclerosis per se. The hardening and narrowing of the arteries certainly restricts the blood supply of the limbs, which require at moments of active exercise a greatly increased supply of all the materials carried to them in the blood. But even these vessels are usually able to care for the needs of parts not in very active use. Thus, until the disease is serious enough to interfere with the nutrition of *resting limbs*, it need cause no signs. In other words, the disorder caused by arteriosclerosis is at first functional and only in its more advanced stages destructive. The blood stream, restricted in certain areas, is often supplemented, even fully restored, through small vessels opened up under the “stimulus of necessity,” a “collateral” circulation.

The tests detailed below are especially helpful in determining the state of the circulation in the lower limbs. By their aid and by the light of the symptoms and physical signs, an authoritative diagnosis should be made.

TESTS FOR ARTERIAL DEFICIENCY.—To prove that an arterial deficiency exists, it is not enough that the individual should be aged, or even that his palpable arteries, such as those of his wrists and ankles, should feel hard, that is, arteriosclerotic, to the touch.

The X-ray.—If a deficiency is known to exist, the presence of calcification (bony change) in the arteries as shown upon an X-ray plate, is evidence of arteriosclerosis as opposed to any other sort of arterial disease. Calcification on the X-ray plate, however, in the absence of the signs of early or advanced arteriosclerotic disease already described, is not proof of clinical arterial deficiency, though it suggests that such may be present, or may readily occur. It suggests, in fact, a latent, not active disease. Moreover, calcification in the vessels of one part is no proof that a similar state will be found in another, though it is of course suggestive. For that reason, courts should require, and Counsel seek, X-ray plates of the region of injury involved in the parts under litigation.

Color Changes.—The normal color of the fingers and toes is some shade of pink, varying from a very pale tint almost to red. The shade is influenced by the state of the surrounding temperature, for the finer

vessels of the feet and hands respond to cooling by constriction (causing pallor and conserving heat) and to warming by dilatation (reddening the skin and dissipating heat).

At the rather neutral, standard temperature of 70°-75°F., the extremities should preserve at least a faint pinkness, though the temperature of the skin will take on, not that of the body (98.6°F.) but nearly that of the air. And in these surroundings it will feel, objectively, to the examiner's touch, neither hot nor cold. As against this background, the following color changes give sufficient evidence as to the state of the circulation.

1. A dead-white, cold extremity is one in which the arterial circulation has, at least for the moment, become so restricted that it may be said to have ceased.
2. A blue, cold extremity is one in which the circulation is greatly restricted and probably slowed.
3. A red, cold extremity is one, like the preceding, in which the circulation is greatly restricted and slowed, and only differs from the blue, cold extremity in that the blood has not given up its oxygen (thereby failing to make the usual change from arterial to venous blood).
4. A red, hot extremity is the seat of inflammation. Even a deficient circulation permits *some* warming from this cause.

Simple Clinical Tests: Elevation and Depression.—The color of the feet at room temperature being known, the feet are elevated by the examiner to an angle of about 30° with the horizon for a period of two minutes. If the toes retain any pinkness, the arterial circulation is at least fair. Retention of most of the normal pinkness means a normal circulation. The state of the two feet may be compared.^{3*} While the feet are elevated, the shade of pinkness may be studied by compression of the tip of a great toe. The white compression-spot makes a contrast with the surrounding skin and should normally regain its color in several seconds.

On lowering the legs, the time required for pink flushing of the feet and toes is normally five to ten seconds. A flushing time of twenty to thirty seconds is very slow, indicating a much restricted and slowed arterial flow. If a slow flushing is followed by a deep red or bluish-red color in the toes, while they are allowed to remain dependent, this indicates again a very poor circulation.

^{3*} Such comparison should be required by the court when the litigant alleges that a single foot or leg has had its circulation impaired as a result of injury. If the uninjured member shows precisely the same circulatory impairment, this speaks for disease as the sole cause, and against traumatic causation of impaired circulation in the one leg.

The Pulses of the Feet.—Palpable pulsations in the arteries of the feet are evidence of a good arterial circulation. Various degrees of feeble pulsations in any one vessel are significant. There being two palpable pulsating arteries in each normal foot—the *dorsalis pedis* upon the instep; the posterior tibial below the inner ankle bone—absence or feebleness of one arterial pulse is important unless the pulse in the second vessel is exceptionally strong (anatomic anomaly of no significance). In general, the state of the pulses corresponds remarkably well with the other tests just described.

When color and temperature changes indicate a deficient arterial circulation, it is rare to find any but absent or feeble pulsations in the arteries of the foot in question.

On rare occasions, absence of pulsations will be noted in the presence of reasonably normal color tests. Such a finding indicates that a collateral circulation, by way of very fine vessels, has replaced the normal flow through the usual large pulsating arteries. This is far more often seen in thrombo-angiitis obliterans (see below) than in arteriosclerosis.

THE RELATION BETWEEN ARTERIOSCLEROTIC ARTERIAL DEFICIENCY AND EXTERNAL VIOLENCE

Since arteriosclerosis is a process of slow development associated with advanced age, it is certainly not caused by injury, but rather by the natural wear and tear of life. The effect of violence, therefore, is to aggravate the disease, and the background for this aggravation is the narrowing of the large and small arteries (arterioles) and the slowing of the circulation. An injury works harm in several ways. Generally speaking, a bruise or cut, by killing living cells, calls forth in the tissue the reaction of repair.^{4*} This reaction is marked by an outpouring of blood and by the collection, in the bruise, cut or lacerated part, of what is known as an exudate (serum, blood cells, fibrin). In the exudate, the process of disposal of dead and useless material goes on, in preparation for the laying down of the framework which is to replace and unite separated parts. Uncomplicated repair of tissues other than bone, normally requires eight to twelve days. Its extent is of course related to the severity of injury and its success depends upon an active circulation and freedom from infection. Blood must be carried in and worn-out material carried off by capillary^{5*} blood vessels hurriedly formed in great numbers to meet the emergency. By contrast with the normal, the arteriosclerotic part has narrow, inelastic blood vessels carrying a re-

^{4*} The cause of this reaction is not settled. Possibly a specific chemical substance is released by the injured cells.

^{5*} These are tiny vessels having walls composed of a single layer of flattened cells. The exchange of chemical materials between the blood within the capillaries and the tissue fluids outside them is very active.

stricted volume of blood at a slow pace. Injury, then, is followed by failure of repair, or, if not by complete failure, then by a process so slow as to be nearly useless. In fact, the feeble circulation, barely adequate to meet the requirements of everyday life, is called upon for excessive activity to which it cannot possibly respond, and injured tissue suffers and even dies.

Another adverse effect of injury is related to the choking of the local blood vessels by such exudate as is present, an accumulation of this material causing the familiar swelling of any bruised part. Pressure being made within the injured tissues against all the blood vessels—both the arteries carrying blood to the region of injury and the veins carrying it away—acts most effectively upon the easily compressed veins. As a result, blood can still enter the part but finds difficulty in leaving it, and so swelling increases, whereby a vicious circle of swelling and circulatory deficiency is set up, contributing materially to a total circulatory failure.

Yet another bad effect of injury is related to the unhealthy state of the arterial walls. Inelastic and poorly nourished, they are so easily injured that thrombosis^{6*} of blood occurs within them. Thus obstruction by a thrombus may cut down or altogether obstruct the blood supply of an injured part. Should this occur in a large artery, in the region of the knee, for example, an individual previously believed to be in normal health, might, for the first time, exhibit signs of a mild or serious arterial deficiency (page 267).

THE EVIDENCES OF INJURY.—The evidence that external violence has aggravated an arteriosclerotic arterial deficiency is chiefly the changed appearance of the injured part.

If a blow has been received, there will be some local swelling and an appearance of extravasated blood in the skin, that is, ecchymosis, but if the damage done by a blow is serious, the toe, toes or such part of the foot as is at and beyond the point of impact, will soon turn purple and finally black. The time required for this change varies. The deep color will perhaps come first at the most distant point, probably within twenty-four hours, after which the black color of gangrene will often require several days to a week or more to develop. Perhaps the part will hover, as it were, between life and death. Then, after many days of an appearance of purplish discoloration and swelling, some or all of it will take on the black color of gangrene. Only the tip of a toe may die, or the skin over a bony prominence. Ultimately, the gangrenous part, unless infections set in, will shrivel or, as it is said, mummify. Thus injury may cause the loss of a toe, several toes or even a part of the foot.

For discussion of the distinction between thrombosis and clotting see note 1 *supra*.

Pain, as a rule, is brought on or increased by injury in a part already the seat of arterial deficiency. The change of color in a toe or foot to deep red, reddish blue or deep purple, that is, the change showing aggravation of the deficiency, is usually accompanied by pain. Such pain is apt to be continuous, often preventing rest and sleep. But it tends to be increased by elevation of the limb and to be lessened or relieved by depression. And since elevation diminishes the arterial supply to the part, and depression, by gravity, increases it, one must believe that it is through a further cutting down of the circulation that the injury causes pain. An arteriosclerotic individual, whose foot is rather pale and cool, may have been quite comfortable until an injury causes some part of the extremity to become discolored, painful and threatened with gangrene.

All such terminal stages of the disease may of course occur in the absence of violence, though usually some minor accident brings them on, as for instance, too close cutting of nails, paring off a callus or corn, or the irritation of an ingrowing toenail. But if a bruise, abrasion, cut or laceration has occurred, even if of a minor sort, it may properly be blamed, provided it can directly be connected with the part in which the disease has advanced.

Evidence that the ulceration of gangrene alleged to have been caused by an injury is due to aggravation of arteriosclerosis, rests upon the advanced age of the individual, a history of early symptoms of arteriosclerotic arterial deficiency, the various tests already described, especially the appearance of calcified vessels in the X-ray plate and lack of the normal arterial pulsations in the injured (and perhaps opposite) foot. Evidence of arterial disease in the opposite leg supports the contention of a previously existing arteriosclerosis, but apparent normalcy of the opposite leg does not disprove the contention.

If the injury is alleged not to have been the cause of swelling, discoloration and gangrene, that is, signs of serious arteriosclerotic arterial deficiency, it would be necessary to show that the advanced state of disease actually antedated the injury. That noteworthy effects solely due to arterial deficiency should appear suddenly and spontaneously as a coincidence with the injury is beyond probability.

That arteriosclerosis did not enter into the state of disease precipitated by injury would be indicated by the individual's age (below fifty-five years, as a rule) and the absence of calcification in the vessels of the injured limb, as shown in the X-ray plate. But the fact that in an individual of an age consistent with arteriosclerosis, no previous symptoms of arteriosclerosis had existed, would not bar the diagnosis of arteriosclerotic arterial disease.

One foot seldom exhibits an arterial deficiency without some sign of

the disorder in the other, but differences between two extremities are very common, almost the rule. When an injury precipitates a serious situation in one foot, it is therefore not remarkable that the second foot should show only slight signs of a defective circulation. Such variations are related to the patchy, erratic quality of the arterial narrowing in arteriosclerosis and to the vagaries of thrombus formation in the large and small arteries.

Injury of a Large Artery.—It has already been indicated that thrombosis in a large artery may bring on suddenly the signs of an arteriosclerotic arterial deficiency. An injury is capable of precipitating such a situation, to which the arteriosclerotic vessel is in any case liable. A case in point would be an unusual, sudden strain or blow from a heavy object acting upon the back of the knee or some part of the calf. As a result, there might be no ecchymosis or swelling, yet the effect of the injury upon the rather stiff, inelastic artery would be to set up an occluding thrombosis within it. The individual might, immediately or in a day or two, experience intermittent claudication (limp) or the toes and foot might in addition show the purplish color and coldness of a greatly restricted arterial circulation. In either event, the pulse in the foot would be absent or greatly diminished in strength as compared to the opposite leg and the X-ray would almost certainly show some calcification in the vessels of the limb in question.

A Popliteal Aneurysm^{7*} (the main artery of the leg, in passing behind the knee, is called the popliteal), the most common non-syphilitic aneurysm, will produce a clinical picture almost identical with that of a thrombosis. While it cannot be said that injury usually causes such an aneurysm, which is really a partial rupture of the artery, it is probable that active bending of the arteriosclerotic vessel predisposes to it. Moreover, once the aneurysm is formed, heavy exercise or a blow may cause increased damage to the brittle wall of the aneurysmal sac and induce thrombosis within it. Though it is true that such may occur at any time, the relation of injury to a sudden increase in the size of the sac and to cutting off of the circulation from the leg beyond, might be convincing.

^{7*} An aneurysm is a local dilatation of a large artery. It is not caused by injury. Certain aneurysms are due to syphilitic disease which weakens the arterial wall, which is then dilated by pressure of the blood-stream. An injury or sudden strain may cause it to rupture, with a serious, even fatal outpouring of blood.

The popliteal variety is usually not syphilitic; nor is it due to injury. But since the artery is subject to a considerable amount of bending, it may, especially if arteriosclerotic, crack, stretch or tear. Actually, one side "blows out" perhaps, allowing blood to escape. As this occurs, the blood, which cannot escape to the surface, becomes clotted and soon a false sack is found about it, the central part being more or less liquid. Since the mass pulsates with each arterial beat, it is detectable back of the knee by the touch. The vessels, into which such an aneurysm leads, seldom receive a normal supply of blood, and thus a greater or less degree of arterial deficiency results.

Cuts and Abrasions.—The role played by a cut or abrasion is similar to but not identical with that of a blow. Again an injury is made, calling for repair which is not forthcoming. A lifeless wound is left, which remains open and which almost necessarily becomes contaminated by bacteria. The infection may remain local, and indeed the neighboring tissues may establish an inflammatory zone about the injury, a feeble defense easily broken down. In some instances, appropriate treatment may cause the wound finally to heal, but the tendency is for the wound to become the source of a slowly spreading process marked by infection and death of the tissues. Neighboring toes, joints, indeed both bony and soft parts, may become gangrenous. Frequently, in spite of treatment, loss of a leg is threatened. And, because diabetes decidedly lowers the resistance of the tissues to infection, its presence should be suspected when a particularly feeble response to injury and infection is observed.

Thermal Injuries: Exposure to Heat.—The effect of any application of heat to a part is to increase the rate of its metabolism, that is, its local chemistry. To support this locally increased activity, blood must freely be supplied. Should the arterial circulation be deficient, the heated tissues, failing to receive fuel to support their activities, break down. This may properly be called a burn, which occurs at a temperature lower than that at which this destructive reaction normally takes place. Or, in other words, a degree of heat which vascular tissue would easily bear, harms or destroys avascular tissue.

Cold, as is now realized, is, up to a certain point, well borne by tissues having a deficient circulation. The effect of cold is to retard metabolism, to slow down chemical processes. A cooled part takes on a dead-white appearance and becomes subjectively numb. When already near to gangrene, and kept at a temperature barely above the freezing point, it suffers little harm from even prolonged exposure to such surroundings. When actually frozen, it dies, that is, sloughs, in medical parlance, but actually the conditions under which cold preserves a poorly nourished part or kills it, are not precisely known. Nor is it known exactly under what circumstances cold, short of freezing, excites such contractions of the blood vessels of a vascular part as to damage it seriously. Individuals with a *normal* circulation may suffer marked contractions of small arteries, as a response to cold, in a very chronic or obstinate form; and treatment, intended to relax such spasms before permanent damage can be done, is important. For the ill-advised application of heat to even normal tissues which have been exposed to the constrictive action of cold is nearly as harmful as such application would be to tissues having a deficient arteriosclerotic circulation.

ECONOMIC ASPECTS OF ARTERIOSCLEROTIC ARTERIAL DEFICIENCY.—
For both men and women, the early stage offers no economic hardship.

The individual may not be able to walk fast or far and may require heavy foot coverings in cold weather, but long hours of standing cause no hardship.

As soon as discoloration and spontaneous pain set in—a frequent, prompt effect of injury—the individual is handicapped for any occupation requiring long hours of standing. If spontaneous pain is present, even a sedentary occupation requiring close attention may be impossible.

Ulceration and gangrene, with which pain is almost invariably associated, are totally disabling. It is difficult to conceive of any occupation, however trifling, at which the individual can work or to which he can give attention. Moreover, hospitalization is often, though not invariably, required, not necessarily for a long period, but to put the patient in the way of such treatment as will relieve pain, favor healing and perhaps permit ultimate local amputation, as of a toe. But relief such that restoration to an active life and an occupation requiring normal vigor is secured cannot be considered probable. Such an event would be rare.

Amputation of a limb is often required. In that case, all of the leg below the knee will invariably be removed. Hospitalization for many weeks will be inevitable. And a surgical fee must be added to the hospital expense. A determined person, otherwise well, will afterwards be able to secure and use an artificial leg, but, again, will be unable to resume an active, vigorous life; and the average individual will be unable even to use an artificial leg.

It will be seen that serious aggravation of the arteriosclerotic state by injury is likely to disable the individual, doing away with wage-earning at his or her original trade or occupation. In addition, hospitalization will usually be required, often for a considerable period. Amputation of a limb will call for prolonged hospitalization, a surgical fee and, in some cases, the expense of an artificial leg. Much of the disability is inherent in the age and lack of vigor of the injured person. That is, a disorder which would not disable a young or middle-aged individual, usually disables the victim of arteriosclerotic disease.

THROMBO-ANGIITIS OBLITERANS: BUERGER'S DISEASE

Although the veins as well as the arteries are involved in this disease, arterial deficiency is again the essential feature. But the individuals attacked are far younger than is the case with arteriosclerosis, and the disorder is capable of taking a greater variety of courses. For the more elastic blood vessels of early life are able to establish new pathways, circumventing local arterial obstruction to a degree altogether beyond the capacity of the harder, older vessels of arteriosclerosis.

Thrombo-angiitis obliterans is almost exclusively a disease of males,

so much so that a diagnosis, in any illness of a similar nature in a female, is only accepted after the most rigid scrutiny.

The disease is most apt to show itself in early adult life, that is, in the thirties and forties. However, it is not at all rare in the twenties, and has even been recognized in boys of fifteen to twenty. When the disease occurs in the sixth decade, a distinction from arteriosclerosis is difficult and of no great importance.

No cause is known. Some adverse influence of an inflammatory sort affects the blood vessels of the limbs, the legs primarily and almost exclusively. Seldom, and rarely in a serious form, it may involve the hands. No actual infection has ever been proved. Yet certain aspects of the disease suggest that something irritative and even destructive gains access to the blood vessels from the toes (and fingers), and there is no question that tobacco smoking has a decidedly bad influence upon the course of the disorder, which is not to say that it is necessarily a cause.

Pathology.—The basic lesion is an inflammation of the walls of both arteries and veins of the limbs. With this, as a natural consequence, thrombosis (see footnote 1* *supra*) is invariably associated. Vessels of medium size are the ones first involved. Buerger maintains that the most characteristic, acute, inflammatory changes, marked even by occasional abscess-like collections of pus cells in the thrombus, are seen in the superficial veins, that is, those outside the muscular mass of the leg. However, these superficial veins are involved in only about one third of the cases. It is probable that some good-sized artery (and vein) belonging to one of the three main, deep systems below the knee is first attacked. Progressing in relapsing fashion, with intervals of quietude, the thrombosis may extend in either direction, finally occupying the greater part of one or more of the three main trunks, and even, in the most serious form of the disease, penetrating into the smaller vessels of the feet and toes. In that case, gangrene, very much like that of arteriosclerosis, sets in. The thrombosing process need not necessarily obstruct completely the arteries and veins it involves. Narrow channels are often left as the thrombus is converted into scar—organized, in medical parlance—but no vessel which is attacked ever again carries more than a small fraction of the original blood-stream. The tendency, in the average case, is to diversion of the arterial blood from the large and medium to the finer vessels or arterioles, a process which requires years. Sometimes, the femoral artery—the main vessel of the leg—is obstructed, high up in the upper thigh, in which case the blood is often able to flow through newly opened channels about the blocked vessel and supply the peripheral parts through the relatively normal ones beyond. Actually, as already stated, it is when the small vessels of the feet and toes are

diseased that the condition is most serious, for then a collateral circulation cannot be established to supply these distant parts.

Symptoms.—The disease is able, occasionally, to establish itself without causing any symptoms. Pain is a late, never an early symptom, and even intermittent claudication on walking, the one characteristic early evidence of the disorder, need not be observed in persons pursuing a sedentary occupation. As a rule, the individual first notices on walking, just as already has been told of arteriosclerotic vascular disease, a cramp-like sensation in the calf of the leg. This appears constantly when he walks for a particular distance, at a particular pace; so that one is accustomed to say that the painful lameness occurs after walking one or two blocks, or perhaps a quarter of a mile. If the individual attempts to continue walking, the pain increases, finally involving the whole lower leg and radiating up the thigh. But if the victim sits down or even stands still, the pain, in a few minutes, disappears and he can walk on as before. Hence the term “intermittent claudication,” or “intermittent limp.”

At this early but characteristic stage, which may persist for months or even years, the appearance of the affected limb—the two are seldom involved to an equal degree—is usually quite normal. If any change is evident, the foot and toes may appear a little pale. In that case, the individual will be apt to notice some slight feeling of coldness or even a tendency to “numbness” in the foot. Atrophy of the muscles, so common in arteriosclerotic arterial deficiency, is absent.

A later stage is marked by some degree of redness or blueness of one or more of the toes, especially the great toe. A part of the forefoot may even show the change, with which some slight swelling is apt to be associated. The discoloration is increased upon dependency and disappears on elevation, but in that position is succeeded by an unhealthy whiteness. At this time, spontaneous pain usually sets in. It has a disagreeable, burning quality, is apt to be made worse by elevation and diminished by depression of the limb. Even now, no ulceration or gangrene need be present, though both are threatened. But the subject is crippled. The affected foot is often sensitive to handling. Even in the absence of injury, the disease tends to progress. For dependency leads to swelling (edema), which in turn interferes with the already deficient blood supply of the part, a vicious circle.

A late stage is marked by actual ulceration or gangrene. The tip of a toe, a spot beside a badly curved nail, or over any bony prominence often becomes deep purple and then black; or a small ulceration forms in one of these situations. The disorder is much like that of arteriosclerosis, though there is a rather better resistance to infection. Pain,

however, is decidedly more severe, persistent and disabling. If uncontrollable, pain rather than gangrene enforces amputation.

The Course of Thrombo-angiitis obliterans may be judged from the above account of the pathology and symptoms. It is slow and subject to progress and recession. Fulminating cases are rare. Very chronic ones, marked by an intermittent limp in one leg, which persists for years without change, are not very uncommon. Injury is very apt to lead to so rapid a development of the disease that it appears actually to have caused it. But ill-advised treatment of an "ingrowing" toenail or of a corn or callus is nearly as apt to be the exciting, aggravating factor.

A very interesting matter is the possible relation of the fungi of the skin and nails to the disease, that is, the dermatophytoses and onychomycoses, for which the layman's name is "Athlete's Foot." Certainly, secondary infections with bacteria are introduced through the desquamations, cracks and serious deformities of the nails due to these diseases. Conceivably, the tissues may become oversensitive to the proteins of the fungus. In any case, treatment of serious infestation with fungi is valuable in a prophylactic way and influences favorably the course of the disease. Allusion has already been made to the relation of tobacco smoking to thrombo-angiitis obliterans. One often sees a superficial wandering phlebitis heal and fail to recur as a result of withdrawal of tobacco. Particularly does one see occasionally a dramatic relief of pain from this same cause.

Considerable space has been given to an account of the pathology, clinical symptoms and course of this rather common disease to bring out its capacity for latency, for periods of activity and quiescence and for responding, favorably or adversely, as the case may be, to a number of factors. These must be kept in mind in analyzing the effect of injury.

In respect to evidences of a defective arterial circulation, reference should be made to the various tests described under Arteriosclerotic Arterial Deficiency. In general, these tests are equally applicable in both diseases. The X-ray, of course, will not reveal the calcification of the arteries characteristic of arteriosclerosis. But the colors in the skin, the reaction to elevation and depression of the limb, and the presence or absence of the arterial pulsations mean just what they do in arteriosclerotic arterial deficiency. Naturally, the comparative youth of those suffering from Buerger's Disease makes the small vessels of their toes and feet more responsive to external temperatures; and thus the color of the toes often changes rapidly (and individually) as their surroundings vary between coolness and warmth. Such vasomotor effects are partly responsible for the "red, white and blue" toes of Buerger's Disease.

There is no visible sign, aside from the appearance of inflamed thrombosed superficial veins, in a small proportion of cases, of the in-

volvement of veins in the disease. The tests relate wholly to the state of the arteries.

THE RELATION BETWEEN THROMBO-ANGIITIS OBLITERANS AND EXTERNAL VIOLENCE

The effect of a blow, crush, sprain or wound upon a limb, already the seat of thrombo-angiitis obliterans, is to bring out rather dramatically, in some cases, evidences of disease and hasten its course. The failure of repair in response to injury when the arterial blood supply is deficient has already been explained. This occurs in Buerger's Disease just as in arteriosclerotic states; and injured tissues become discolored, or partly gangrenous, or wholly gangrenous, according to the degree of arterial deficiency and the nature of the injury. Wounds heal badly and, in consequence, infection is threatened.

Over and beyond such immediate effects as the above, the disease itself is decidedly aggravated. Let it be supposed that a man of thirty, supposedly well, is injured by the dropping of the edge of a heavy box upon the base of his left great toe. The skin is not broken. He tries to continue work but finds the toe painful and gives up after a few hours. Beyond some black-and-blueness, the region of injury is little changed for a day or two, but soon the great toe becomes reddish-blue and increasingly painful. There is swelling of the toe and adjacent foot. In the course of a couple of weeks, a sore appears beside the toenail or the tip of the toe becomes black. Then it is noticed that the other toes are a little red, shiny and swollen. The adjacent foot shows a touch of this change. The man objects to handling of the toes and finds pain increased by elevation of the foot. Now perhaps it is observed for the first time that the arterial pulses of the left foot are absent. To the touch, it is cool below the ankle. On elevation, the toes and foot appear a dead white. On depression, the toes, especially the great one, are purplish, the forefoot red, but shading upward into a normal color.

The state just pictured is a fairly advanced stage of Buerger's Disease, brought on within a few days and well developed within a few weeks by a moderate injury. Perhaps an ill-advised attempt is made to amputate the partly gangrenous great toe, as a result of which a lifeless open wound is left. But even if healing occurs, the foot is left in some degree painful, the individual is crippled and cannot work. Thrombosis has probably spread from the region of injury by way of connecting blood vessels. Had there not been some background of disease present, the bruise would have healed after a short period of soreness, as most bruises do.

But this may not represent the whole story. When the condition in the injured leg has been stabilized, a careful examination will be apt to

show that the pulses in the second foot are weak—one of them may well have disappeared. There is often a suggestion that with the acceleration of the disease in one leg have been associated the first signs of trouble in the other. Just why this should occur—it need not—is far from clear. But it is a rather familiar observation that while an individual is confined to bed by a process of thrombosis (“phlebitis”) in the veins of one leg, a similar process occasionally starts in the other. Something of the same sort may occur here.

Other injuries than a bruise are capable of producing similar changes. These include accidental wounds and abrasions as well as the cutting of corns or calluses and purposeful but ill-advised operative treatment of a deformed nail or toe. Such things should be inquired into in connection with the alleged incitement or aggravation of thrombo-angiitis obliterans by injury.

THE EVIDENCE OF INJURY.—In the absence of other causes and even in the absence of any previously recognized symptoms, the appearance of reddish-purple discoloration and swelling, following within one or two to ten days of an injury, indicates that a latent thrombo-angiitis obliterans has been activated. Often a careful interrogation will of course disclose a story of earlier lameness on walking. The age of the individual will be of no great importance, provided it is under fifty.

In addition to the color changes and disappearance of the pulses at the ankle, a study of the arterial oscillations in both legs will often be important. If a blood pressure cuff connected to an anaeroid sphygmomanometer is firmly wrapped about the calf and inflated as if to record the blood pressure, the arterial beats will give a regular vibration to the indicator needle. The vigorous excursion of the needle in the normal person being known, a lessened excursion in a case under study suggests strongly that the large arteries in the leg are in some degree obstructed by the disease.

It goes without saying that, provided the individual has sufficiently recovered from the local disorder of the toes and can walk with reasonable vigor, the subsequent occurrence of the characteristic intermittent limp offers confirmatory evidence that Buerger's Disease is now established.

Evidence that the signs of disease, alleged to have been caused by an injury, is due to aggravation of thrombo-angiitis obliterans, rests upon the appearance of the toes and foot (by contrast with the previous state), the prompt appearance of the changes and the incapacity of the individual. The arteries will not appear calcified by X-ray. A normal state of the opposite leg does not deny the diagnosis of Buerger's Disease; but diminished strength or absence of one or both of the

arterial pulses in the opposite leg confirms the contention that the disease is present.

If the injury is alleged not to have been the cause of swelling, discoloration and other signs indicative of the disease, it would be necessary to show that these signs had been present before the injury and had persisted without significant change. As in arteriosclerosis, it is beyond probability that noteworthy effects of thrombo-angiitis obliterans should appear spontaneously as a coincidence with the injury.

That thrombo-angiitis obliterans did not enter into the disorder precipitated by the injury would only be indicated by the absence of characteristic signs of the disease in the injured leg, already described.

Thermal Injuries: Exposure to Heat and Cold.—To heat, the tissues in thrombo-angiitis obliterans react just as do arteriosclerotic tissues, that is, heat calls for a metabolic activity for which a blood supply is not forthcoming. Thus, the terminal parts of the foot are readily injured by heat, the skin destroyed and ulceration established.

Severe cold, that is, actual freezing, causes gangrene of the parts exposed. It must be supposed that, owing to the diminished arterial supply, frostbite, under these conditions, occurs more rapidly than is the case with normal tissues.

Exposure, short of freezing, is likely to increase the contraction of the smaller arteries of the toes and feet. There is present in these vessels an element of irritability, that is, their muscular walls are readily thrown into spasm by cooling. This is an exaggeration of a normal reaction to cold already described, and indeed an excessive vasomotor irritability in response to cold is not by any means peculiar to thrombo-angiitis obliterans. However, the effect of exposure to cold, if serious or repeated, is likely to aggravate the disease.

ECONOMIC ASPECTS OF THROMBO-ANGIITIS OBLITERANS.—The age of most individuals suffering from this disease holds out a promise of a considerable degree of recovery and some economic rehabilitation.

The mild form of the disease, of which the only evidence will be intermittent claudication and lessening of strength in, or actual disappearance of, the pulses in the feet, limits the individual's ability to work to the extent that it lowers his capacity for locomotion—and for running a machine requiring active use of his legs. Moreover, it exposes him to aggravation of the disease by injury. Only in a small proportion of all cases is treatment able to remove such incapacity as is present.

The serious form, marked by purplish discoloration, ulceration and even gangrene of toes, can frequently be checked and improved, usually after a considerable period of treatment, a matter of many weeks and sometimes months. Let this be called "relief," not "cure." The younger the individual, the better the chance of recovery. Even the loss of several

toes is consistent with a reasonably active life, though not with a strenuously athletic one. If relief for any reason is not obtained, the disease is totally disabling. For even if a limb is lost, the persistence of a wearing pain renders the individual unfit to give his attention to an occupation requiring intelligence and care. But relief seldom restores the individual to his previous full health. For one thing, he will seldom be able to walk fast or far, and he will never be able to run for any considerable distance. For another, he is liable to further accident and, consequently, to re-activation of his disease. He dare not engage in any hazardous occupation. On the other hand, he is free from pain; his general health is unimpaired, and he can give his full mind to his work, such as it is.

If relief of pain and ulceration is secured by amputation of a limb, the individual is fit only for a sedentary occupation. His amputation will leave him without a knee-joint in the amputated limb. Only an unusually determined person can lead an active life and never a strenuously athletic one. This is partly because the second leg is almost certain to be in some degree diseased. If both legs are seriously diseased, the individual must be held to be totally disabled, being fit only for such work as he can do in the house.

Relief of the disease is greatly at the mercy of the individual's eagerness for it and his determination to secure it. For example, if the painful period is prolonged, he may prove so lacking in resistance to pain as to have acquired the drug habit. Above all, if he will not abandon tobacco smoking, his disease will almost necessarily persist and indeed will probably be aggravated. Thus the victim of thrombo-angiitis obliterans whose disease is developed by injury may fail to seize an opportunity offered him for improvement and rehabilitation, a tendency which is emphasized by the demoralizing effect of the expectations of damages, insurance and so on. Such considerations as the above are significant even when the treatment secured is ideal, and not merely of average value.