**Vascular disorders of the limbs : described for practitioners and students / by Sir Thomas Lewis.**


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# VASCULAR DISORDERS OF THE LIMBS

THOMAS LEWIS


THOMAS LEWIS.

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# VASCULAR DISORDERS OF THE LIMBS

DESCRIBED FOR
PRACTITIONERS AND STUDENTS

*BY THE SAME AUTHOR*

CLINICAL DISORDERS OF THE HEART BEAT

CLINICAL ELECTROCARDIOGRAPHY

THE MECHANISM AND GRAPHIC REGISTRATION OF THE HEART BEAT

LECTURES ON THE HEART

THE SOLDIER'S HEART AND THE EFFORT SYNDROME

BLOOD VESSELS OF THE HUMAN SKIN AND THEIR RESPONSES

DISEASES OF THE HEART, DESCRIBED FOR PRACTITIONERS AND STUDENTS

CLINICAL SCIENCE, ILLUSTRATED BY PERSONAL EXPERIENCES

DEPARTMENT OF CLINICAL RESEARCH
UNIVERSITY COLLEGE HOSPITAL, LONDON

# VASCULAR DISORDERS OF THE LIMBS

DESCRIBED FOR
PRACTITIONERS AND STUDENTS

By SIR THOMAS LEWIS
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# PREFACE

THE great advance in certain branches of medical practice during the present century has depended chiefly, not upon dramatic discoveries, but upon a phenomenal growth of accurate observations of fact, and the gradual welding of these together by new conceptions to form simpler and truer systems of thought. In disorders of the circulation in the limbs, progress has been along these fundamental lines. During recent years, very many new discoveries have been made; though most of these have been insufficiently obtrusive to attract attention from any but those closely studying cognate problems, in reality each has formed a solid addition. Together they comprise a very impressive contribution to our knowledge of the way in which vascular disorders of the limbs originate, and of the manner in which such disturbances of circulation produce their symptoms.

These discoveries have led to close revision and to important changes of nomenclature; in particular our attitude towards states formerly regarded as disturbances of the vasomotor system has been altered. They have stimulated much work in which new and efficient tests of vascular function and capacity in man have been devised, thus aiding accurate recognition of pathological states, and the assessment of their seriousness, during life. They have added greatly to our comprehension of the way in which the diseases concerned manifest themselves. Alongside these changes, new methods and improvements of old methods of treatment have inevitably developed.

Much of this advance has happened in the past eight or nine years, a period during which in our department of clinical research my colleagues and I, by concentrating upon these problems, have had exceptional opportunities of observing progress. At the beginning of this period I published my book *The Blood Vessels of the Human Skin and their Responses*, recounting a long series of studies of the peripheral circulation. It was clear at the time that these studies had been brought to a point where similar methods might with advantage be applied to many disorders of the circulation in the limbs. A beginning was shortly made in the case of "Raynaud's Disease"

and these and related researches have continued until to-day. Simultaneously interest awakened in many other institutions in different lands and progress has been unusually rapid.

On reviewing this work as a whole, I judge that the time is opportune to attempt to outline conceptions of certain peripheral disorders of the circulation in a way that may prove useful to those engaged, not in research, but in practice. This book is not a comprehensive account of vascular disorders of the limbs. I have hesitated little in omitting from consideration even important disorders such as phlebitis, varicose veins, and aneurysm, because their inclusion, though warranted to satisfy comprehensiveness, would overweight the book and interrupt the general trend of argument; I have hesitated the less because I have nothing of practical importance to add to the excellent accounts of such maladies that are already available.

Because I am writing to students and practitioners, description is almost confined to methods that they can use themselves; intricate tests and tests requiring special training are omitted or dealt with briefly. At the same time I have tried to incorporate enough to ensure recognition and adequate management of the different conditions. By this simplification it is hoped to place the essential among new methods and ideas at the service of a greater number of sick people.

I have included no references to original papers; those who desire to become more intimately acquainted with the new observations upon which the chapters are based, will find most of these recorded or referred to in the later volumes of the journal *Heart* and the first two volumes of the journal *Clinical Science*.

In conclusion it is my pleasant duty to record my thanks to Dr. G. W. Pickering, in collaboration with whom so much of the work upon which this book is based has been done, and to Dr. H. P. Himsworth, for the care with which they have perused and criticised my manuscript and proofs. I also desire here to express my cordial appreciation of help received from the many of my medical and surgical colleagues on the staff of University College Hospital, who have so kindly and so often referred to me cases of vascular disorders of the limbs that they have felt would interest me. Without this generous co-operation, which I have gratefully enjoyed for many years, much of the work of my research department relating to the questions discussed could not have been done.

THOMAS LEWIS

*January* 1936

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# CHAPTER I

## THE CIRCULATION IN THE LIMB AND ITS TESTING

### Functions and Control of the Circulation

To recognise and manage various disorders of the circulation of blood to the limbs, familiarity with the arrangement of the chief arteries and veins of the limbs and with their anatomical relations to other important structures is required. It is also necessary broadly to understand the functions of the circulation to the limb, and the manner in which it is controlled.

#### FUNCTIONS

The circulation to the limbs often participates, actively or passively, in responses of the whole peripheral circulation; the mechanism and purpose of such general reactions only concern us incidentally. The circulation to the limbs will be regarded mainly from the standpoint of its local functions.

Of these functions the first to be named is that of transporting to or from the extremities the substances needed for or arising out of metabolism, and thus to ensure the proper nutrition of the tissues. This function is common to the circulation as a whole. The metabolic needs of most of the tissues of the limb, however, are not great; the blood-flow required to nourish skin, subcutaneous tissues, bone and resting muscle, is small; only the muscle in action makes large demands.

The second function is to be found in the mobilisation of the forces of inflammation in defence against various forms of injury experienced; this also is common to the circulation as a whole, though the limbs are particularly prone to damage, owing to the vigour and frequency of their movements.

A third function is the maintenance of temperature within safe bounds, so that the tissues, and especially the skin, may be guarded against damage from continued over-cooling or over-heating.

A fourth function, and the last that will be named, concerns the part played by the limbs in controlling body temperature. The surface of the limbs actually forms about two-thirds of that of the whole body and, under conditions of civilised life, the limbs are amongst the most exposed parts. Thus the circulation to the limbs plays a dominant rôle in regulating heat loss.

## CONTROL

*General Reaction and Nervous Control.*—Though the capacity of vessels of all orders, large and small arteries, arterioles, capillaries, minute venules, small and large veins, may affect it, general rate of blood-flow is chiefly regulated by the state of the small arteries and of the arterioles. This is not only true of the limb as a whole but of the body generally. In the case of the limb, however, there is, in addition, a mechanism almost peculiar to it. This consists of direct connections between arterioles and venules through special sluice gates called arteriolo-venous anastomoses. These anastomoses, just visible to the naked eye when dissected, are present in great numbers in all the digits and in the palm of the hand and sole of the foot. Their walls are very muscular and are richly innervated. The anastomoses when open allow a veritable flood to pass from arterioles to venules, and provide an exceedingly effective mechanism for rapidly warming up the limbs from their very tips.

When the vessels of the limbs participate in general reactions of the peripheral circulation to calls regulating blood pressure, the small arteries, arterioles, and arteriolo-venous anastomoses of the skin and directly subjacent tissues are chiefly brought into or out of action. Precisely the same cutaneous vessels participate in the regulation of body temperature, in which the limbs play so large a part.

These general vascular reactions affect all the limbs, and all parts of them, together; and they are controlled by the central nervous system through the vasomotor nerves, distributed in the limbs by way of their mixed nerves. The sympathetic nervous action is reinforced by the simultaneous release of hormones, such as adrenaline, into the blood stream.

*Local Reaction and Metabolite Control.*—The nutritional requirements of the tissues fluctuate greatly from moment to moment and from place to place, and these are met by exchange through the walls of the minute blood vessels. This exchange calls for fine discrimina-

tion in the distribution of blood through these vessels, if the supply is to be economic. An economic system of distribution exists, and consists of fluctuations in the size of the appropriate vessels of the smallest orders. These fluctuations are mainly controlled automatically by metabolism itself, the products of local metabolism acting directly upon the requisite vessels and keeping them in a state suited to the needs of the tissue at a given moment and given place. The circulation to muscles is chiefly regulated in this way, the supply varying with activity. A blood-flow debt to a given territory, accumulated during temporary and local arrest of circulation, is satisfied by an increased blood-flow after the circulation is released, this compensation depending upon a similar mechanism. Fine adjustments of the circulation to suit local needs are made by similar responses of the smallest vessels in instances of injury, the substances being released by the actual injury and giving rise at once to vascular responses in the corresponding territory.

*Local Control by Temperature.*—In addition to the two chief controls described there is a third, which assumes unusual importance in the case of limb vessels. Arteries, arterioles, capillaries, and veins all react directly to temperature; they shrink when cooled. The metabolic need of tissues falls as their temperature falls, and the reaction of the vessels to cold is consequently appropriate from this standpoint. Vessels of the body that lie deeply, or that lie superficially in the trunk, are protected against large changes of temperature; but the limbs, and especially the ends of the limbs, vary greatly in temperature in circumstances of everyday life and their vessels are therefore unusually affected. The direct and considerable influence of temperature on the exposed blood vessels of the limbs contributes to the maintenance of body temperature; the closure of vessels in a cold limb limits the amount of cooled blood returning to the body.

This direct influence of temperature is also important from another standpoint, namely, in testing the vessels of the limbs. In drawing conclusions from the size of a vessel or from the extent of its pulsation, the temperature of the limb must always be taken into account or must be controlled.

The statements of this brief introduction will be supplemented as occasion arises. We proceed to describe methods of examining the vessels of a limb. It is convenient to place these tests in the forefront of the book, because in subsequently describing various con-

ditions, such an arrangement avoids needless repetition. Tests are of three kinds according to whether they explore rate of blood-flow at the moment, the state of various vessels and especially their capacity to dilate, and the intactness or otherwise of the vasoconstrictor nerves.

## Tests of Blood-Flow Rate

The absolute amount of blood flowing to a limb can be measured by enclosing the limb in a plethysmograph, and ascertaining the rate at which the volume of the limb increases during the first few seconds after the venous return is stopped by the pneumatic pressure of a cuff encircling the base of the limb. Another method is to measure the rate at which heat is eliminated from a limb lying in a calorimeter containing water below blood temperature. The latter method often provides a valuable gauge, but does not give an absolute measure of flow in cubic centimetres per minute. It accurately detects change of flow from moment to moment in the superficial vessels, but relatively neglects that in deeper lying tissues. Both methods are too cumbersome to employ in the routine work of the clinic, but both have been used extensively to determine the values of simpler tests, which may be described in more detail.

### SKIN TEMPERATURE

Perhaps the most reliable method of gauging the state of blood-flow to a resting limb clinically is to estimate its surface temperature. The skin temperature depends upon the amount of heat brought to it by the blood stream, and upon the amount of heat lost from the surface. It is determined chiefly by the rate of blood-flow through the skin, and by the temperature, humidity, and movement of surrounding air. We try to judge as to how far, if at all, skin temperature departs from what is usual in the circumstances. The temperature of the skin can be measured accurately by means of suitable mercurial thermometers or thermoelectric junctions; but the value of actual readings is limited owing to the range of temperatures displayed by different regions and in different normal circumstances; as will be made clear, the use of apparatus is not often necessary. Under ordinary resting conditions, indoors, the trunk and head and neck present the highest surface temperatures; the temperatures of the limbs are lower, and those of the leg and foot are lower than those of the arm and hand. These differences are present and conspicuous only at low room temperatures. Owing to

the arteriolo-venous anastomoses, already described, when blood-flow increases to a limb the fingers warm more quickly than the hand. Thus it is not unusual for the temperature of the fingers to be higher than that of hand or even of forearm when the limb is warm (Fig. 1). But when the limb is cool or cold it is the rule for its temperature to decrease steadily as it is traced to the extremity. For the reasons indicated, temperature variations are greatest in the most distal parts of the limbs, and are less prominent as the limb is traced to its base (Fig. 1). Thus the digits provide the most sensitive temperature gauge of circulation to the skin of the limb as a whole.

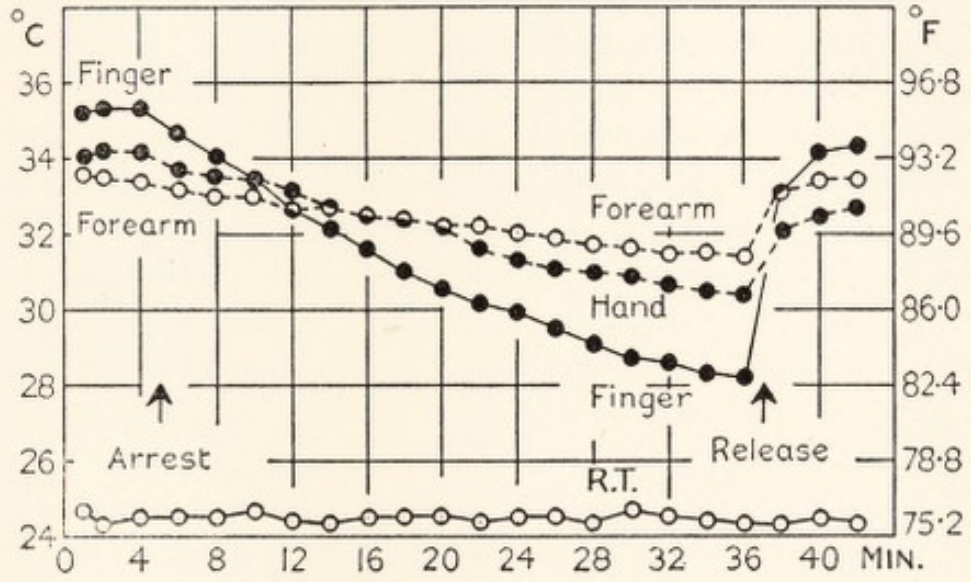


FIG. 1.—Continuous records of skin temperature from the index finger above the nail, the back of the hand, and the forearm, of a normal subject. The finger was at first the warmest of the three. At the first arrow the blood-flow to the arm was arrested by the pressure of a pneumatic cuff. The temperature of the whole arm fell steadily towards that of the room (R.T. = room temperature); that of the finger fell fastest, its surface being greatest relative to its bulk. At the second arrow the blood-flow was released. The finger warmed up most quickly.

Skin temperature cannot fall more than a degree or two below that of the surrounding air and then only if the circulation is arrested and evaporation is occurring. Actually, in cool rooms (16° to 18° C., or 60·8° to 64·4° F.), the temperature of the tips of the fingers rarely falls below 20° C. (68° F.); in warm rooms fingers of resting normal subjects rarely exceed 34° C. (93·2° F.) in temperature. Thus there is a range of about 14° C. (or about 25° F.). The skin of the forearms and upper arms varies much less than this in similar circumstances.

Much more important, however, than the precise temperature is a comparison of relative temperatures of the skin of one person and another, or of one limb and the symmetrical limb. Speaking broadly, normal subjects ordinarily clad and resting in rooms at temperatures of 20° C. (68° F.) and over display warm fingers (around 32° C. or

90° F.), while in rooms occupied at temperatures of 16° C. (60·8° F.) they display cold fingers (around 22° C. or 72° F.). The fingers and toes tend to maintain temperatures not far below blood heat, or not far above the temperature of the surrounding air, rather than intermediate temperatures. It would be unusual (abnormal) for the fingers to be maintained at 32° C. in the cool room or at 25° C. in the warm room. Daily experience is sufficient to teach an observant person to detect any gross departure from normal temperatures usual to the circumstances, and since individual variation is considerable among healthy people, it is only the gross departures that can be regarded as useful practically. The most important of all indications of vascular disorders or disturbances to be derived from temperature observations, is the finding of different temperatures in symmetrical areas of skin of the two limbs of a given individual. Patients will usually give useful information of inequalities of temperature in hands or feet, though they are apt in their replies to confuse a subjective sense of coldness with actual coldness. In comparing the limbs objectively it is necessary to be sure that they are examined under similar conditions in respect of coverings, air currents, etc., and to ascertain that they have been equally treated for a period of many minutes before the observation is made. To compare the two limbs, one of which has recently been in use, or has been bandaged or otherwise covered, would obviously introduce fallacy. When the circulation to one leg is deficient and this is suspected, but a simple comparison of temperature is unconvincing, one of several procedures may be serviceable. If in a cool bedroom the patient's two limbs seem equally warm when first uncovered by bedclothes, simple symmetrical exposure to the air will usually induce a clear difference between the temperature of the feet within ten minutes. In other instances both limbs may be immersed together in water, water at room temperature in warm rooms, or water at blood heat in cold rooms, and the limbs thoroughly dried, symmetrically disposed, and allowed to rest for further observation. The limb in which the circulation is the more active will remain warm longer on cooling or will become warm quicker on warming.

It is erroneous to believe that delicate instruments are necessary to detect differences of skin temperature. The practised hand when warm will detect small differences of temperature between various areas of skin with certainty, it will compare the temperatures prevailing over large surfaces of two arms or two legs more speedily than will any instrument; and speed is very important when, as is

frequent, the temperature of exposed limbs is changing. There is no better method of quickly ascertaining that one limb is cooler than its fellow in its length, or of ascertaining the extent of an area of relative coolness or warmness, than by feeling the relevant skin areas. The observer should test his own hand by placing it against his neck to ascertain that it is warm; and he should use its dorsal rather than its palmar surface in testing. Perhaps the best testing surface for delicate observations is the dorsal surface of the middle phalanx of the flexed finger. Instruments are occasionally needed to decide actual levels of temperature when this is important; they are of particular value in comparing the temperatures of a given area from moment to moment when temperatures are to be charted as in Fig. 1.

When symmetrical limbs have been examined under similar conditions and the one found to be persistently cooler than the other, it becomes obvious that the cooler limb has the smaller cutaneous blood supply; and generally speaking this is due to a vascular defect in the arteries of the cooler limb. These vessels may be diseased, or their tone may be increased; differentiation between the two is dealt with later. Occasionally the warmer limb is the affected one, as when it has lost its sympathetic nerve supply.

While an examination of skin temperature is the most reliable clinical method of detecting decreased or increased blood-flow and in deciding the territory involved, aid is obtained by considering the colour of the skin.

### SKIN COLOUR

Strictly speaking, conclusions drawn from the vascular coloration of the skin are relevant only to the state of the circulation in the skin itself, though frequently directing attention to the state of flow in the limb as a whole. It is to be remembered that this coloration is chiefly derived from blood in the minute vessels of the skin and especially from the plexuses of minute venules in the sub-papillary region. It is important to distinguish between depth of colour and tint.

*Depth of Colour.*—Apart from differences in the state of the blood, depth of colour is governed by the amount of blood contained within the minute vessels of the skin at the moment. These vessels differ in size and in number in different parts; in some parts, like the face and hands, they are frequently permanently dilated. The size of these minute vessels has little effect upon, and is certainly

no safe index of, the amount of blood flowing through them; it is the size of the small artery and arteriole which determines rate of blood-flow.

The minute vessels may become engorged by passive filling; thus it is usual for the depth of colour in a limb to be increased when it is lowered and decreased when it is raised. Obstruction of outflow likewise congests the skin, although actually decreasing blood-flow through it. It is true that engorgement of the minute vessels and increased depth of colour may at times be associated with increased flow, for example, immediately after general vasodilatation; but this association is less frequent than with decreased flow. This is so because the tone of the minute vessels increases in physiological response to increased flow, and decreases in response to decreased flow, through them.

*Tint.*—It is the tint of the skin which has value in indicating rate of blood-flow through it. Blood that comes to the skin in an arterial state gives up oxygen and darkens in tint during its passage through the minute vessels. The slower it flows the more oxygen it gives up. Unless the blood is abnormal in colour before it arrives in the skin, a cyanotic tint of the skin always indicates a slow blood-flow; a violaceous tint is a sign of complete or almost complete arrest of flow. It is sometimes of practical importance to recognise, however, that the rate of flow is not the only factor concerned in the production of cyanosis; temperature of the skin plays its part. If the blood-flow to a limb is arrested, cyanosis develops much more rapidly if the limb is warm than if it is cold; and if the blood-flow to skin that is cold is small and incapable of increase, warming that skin by applying heat externally will increase the grade of cyanosis, sometimes very dramatically. These things are so because warmth has a conspicuous effect in increasing the rate at which oxygen is dissociated from haemoglobin and is used up by the tissues. It is also important to realise that the less emphatic of the cyanotic tints are normal to skin at certain temperatures; the hand of the normal resting subject becomes slightly but distinctly cyanotic after it is immersed in water at 20° C. (68° F.); speaking broadly, we may say that it is natural for skin to present some cyanosis over a range from about 15° to 25° C. (59° to 77° F.) and that such skin temperatures frequently result from external cooling. Moderate cold contracts the arterioles of the skin and slows blood-flow; thus a cyanosed cold skin may have physiological and not pathological significance. For this and other reasons it is

necessary to take temperature into account in using skin colour to judge the state of the vessels. It is also necessary to recognise different grades of cyanosis and to make this recognition as far as possible independent of the depth of vascular skin colour; a pale violaceous skin indicates as slow a blood-flow as does a dark plum-coloured skin. The tendency is to over-rate the degree of oxygen deficiency when the skin is turgid with blood, or when haemoglobin is in excess, and to under-rate it in the contrary circumstances. It may assist if the chief varieties of skin, regarded from standpoint of temperature and colour, are named and their significance briefly described.

### TEMPERATURE AND TINT

*Warm Pale Skin.*—This is a skin through which blood flows rapidly for many minutes. It is warm because flow is fast, pink because of the abundant supply of fully oxygenated blood, and pale because the skin is well nourished and minute vessel tone is therefore high.

*Warm Deeply Coloured Red Skin.*—Such skin has been irritated, by heat or otherwise, it is in a state of inflammation, or it is skin in which arterial vasodilatation has recently been brought about through nervous channels or by means of drugs such as amyl nitrite.

*Warm Deeply Coloured Cyanosed Skin.*—Unless the blood pigments are abnormal, this is skin to which the supply of blood is imperfect and which has been made warm by external heating.

*Cold Pale Cyanosed Skin.*—This is skin to which the blood-flow is very slow or absent. If the tint of the cold skin is violaceous or if the skin is blanched, the circulation to it is absent and has been arrested in it for many minutes. Minor grades of cyanosis are, as previously stated, of much less significance.

*Cold Deeply Coloured Cyanosed Skin.*—This is skin in which the circulation is very slow, and in which blood-flow has been failing for a long time or in which there is a process of low grade inflammation.

*Cold Deeply Coloured Red Skin.*—If the skin is sufficiently cold, 10° C. (50° F.) or less, the blood will not part with its oxygen, but the minute vessels are damaged and dilate, and thus the skin becomes bright red in colour although the blood-flow through it may be small.

### PRESSURE AND RETURN OF COLOUR

If an area of highly coloured skin is pressed upon and the pressure released, the blood flows back again at varying rates in different

subjects. In judging the circulation to the tested skin, this test has limited value. The blood returns slowly to the blanched area only when the skin is cold, coldness which is in itself sufficient to indicate slowness of local circulation. But a rapid obliteration of the area of blanching has no value, for this will occur in congested skin after the circulation to the limb has been arrested. In all instances the rate of return depends chiefly upon the pressure at which blood is standing in venules adjacent to and communicating with those that have been emptied.

## Tests of Vessels

*Main Arteries.*—Presence of pulsation in a given artery is insufficient evidence that blood is being supplied to the corresponding territory, for the flow may be obstructed in smaller arteries (as in Raynaud's phenomenon) or in the arterioles (as in acrocyanosis). Conversely, absence of pulsation in an artery does not signify that there is no flow in it; the flow may even be rapid, as in the pulseless arteries of the legs in coarctation of the aorta. Thus pulsation is an imperfect guide to flow. The significance of pulselessness is that it is the chief sign of a proximal obstruction in the artery displaying it; an increasing proximal obstruction abolishes the pulse before it stops flow, and a complete obstruction does not prevent a return of flow in the distal part of an artery supplied by collateral channels. A diseased artery often presents smaller pulsation than does the normal one, but in strictly comparing symmetrical pulsations the two limbs should first be rendered equal in temperature; even in normal subjects the pulse in a cold limb is smaller than in a warm one.

If pulsation is not detected in a normal situation, as for example in the radial artery at the wrist, the usual line of the artery should be followed as closely as possible up the limb to determine where pulsation can first be felt. Thus absence of pulsation at the wrist leads not infrequently to the discovery that the radial artery turns to the dorsal side of the bone higher than usual in the arm; or it may lead to the observation that pulsation ceases abruptly at some level in brachial or axillary artery. Abrupt termination of pulsation in the line of an artery is obviously a most important sign when it can be obtained, and the site of change may be marked by palpable clot or other specific indication of the nature of the disease. The subclavian, axillary, and brachial arteries can usually be felt almost throughout their courses, so that the level of trouble can be fixed in

them; but from the point at which the brachial vessel divides over the tendon of the biceps to the appearance of its radial and ulnar branches at the wrist, arterial pulsation in the forearm is not usually distinct. The ulnar pulse can usually be detected; the radial pulse can usually be traced in the back of the hand till it passes between the 1st and 2nd metacarpal bones; the pulsation of the palmar vessels and of the digital vessels at the bases of the fingers are often palpable. All these pulsations are more prominent when the hand is warm. A pulse should not be regarded as absent until examination has failed to detect it in a thoroughly warm limb.

The femoral pulse is easily felt at Poupart's ligament and can be traced usually a distance into Scarpa's triangle; it can rarely be felt through the course of Hunter's canal. The popliteal pulse requires close attention; it is felt most easily with the subject prone and the knee flexed at a right angle. The next points of pulsation to be felt in the leg are usually those of the posterior tibial, well behind the internal malleolus, and of the dorsalis pedis in a line passing between the bases of the 1st and 2nd metacarpal bones. The pulse may be perceptible in the sole of the foot. The precaution of examining the limb in a warm state applies equally to leg and arm.

To supplement what can be detected by palpation in the main arteries an oscillometer such as that of Pachon may be used, the cuff being placed at given levels upon the two limbs alternately, and the extent of the oscillations of the anaeroid manometer compared at suitable cuff pressures on the two sides. By this means the level at which pulsation ends or abruptly diminishes can sometimes be fixed more accurately than by palpation, especially when the change occurs in a part of the leg in which pulsation is ordinarily impalpable. Instrumental examination may be employed on the fingers by enclosing them in small plethysmographs and optically recording the pulsations which they yield. Such instrumental helps are valuable from time to time in testing the patency of vessels, but are unessential in routine work.

The freedom of the lumen of two main arterial branches, the radial and ulnar in the hand, the posterior tibial and dorsalis pedis in the foot, can be tested by a simple procedure. Thus one of two main vessels, say the radial, is obliterated while the hand is held aloft and is pale; the hand is then lowered to see if it fills quickly with blood through the ulnar vessel; if it does not do so it is clear that the radial constitutes the almost exclusive source of supply, a fact which will be confirmed if on release of the radial artery the

hand at once flushes an unusually bright red. The test is most reliable when it shows a clear difference between the two hands.

When the flow of blood to a foot or hand is very imperfect, the extremity becomes unusually pale when lifted above the level of the body; it does so because this elevation empties the vascular bed through the veins, and blood enters the minute vessels from the arterial side too slowly to keep them ordinarily distended. If elevation of the foot (or hand) is to be used as a test of blood-flow to that limb, it should be used when the member is warm.

Disease of the wall of an artery can be detected often by feeling its walls and by examining the artery in its length for tortuosity; calcareous deposits are frequently detected by X-ray photography. But the size of the lumen cannot be observed by these means, and this is all important where the flow is concerned. The size of the lumen and points of obstruction can be displayed by injecting a substance opaque to X-rays into the living artery, radiographing the injected vessel at a suitable instant. Strontium, iodine, and thorium compounds have all been used successfully for this purpose; the latter, however, is radioactive. The method produces beautifully clear pictures in skilful hands, but is one that is unlikely to come into general use.

*Small Arteries.*—When the vessels of the normal hand are well dilated throughout, the skin of the hand always displays capillary pulsation, which is seen especially well in the palmar surface of hand and fingers. Immersion of the hand in water at 45° C. (113° F.) for 5 or 10 minutes produces adequate vasodilatation. Capillary pulsation fails to develop in the pads of the fingers in this test if there is material obliteration by disease of the lumen of any artery through which blood normally runs to the skin under observation. If, by heating, capillary pulsation can be induced in the palm of the hand but not in the finger pads, there is obstructive disease in the digital arteries. Similar tests may be applied in the case of the small vessels of the foot.

*Veins.*—The veins collapse when there is no pressure within them and cold often contracts them until they are no longer visible through the skin. In examining the veins of hand or foot to ascertain if these are normally patent, the member should be thoroughly warmed by immersing it in hot water; the foot should be examined in the dependent position and the hand after obstructing the venous return.

### TESTS OF CAPACITY TO DILATE

The flow of blood to a limb or part of a limb may be obstructed because there is structural disease of its arteries, or because vessels supplying it are in a state of high tone or spasm. The former is the cause of permanent obstruction; spasm of arteries is usually transient. Consequently the distinction between a spasmodic affection and structural obliteration rarely presents any difficulty; it may usually be effected by listening to the account of symptoms; it can be effected by immersing the limb in hot water, for heat relaxes arterial spasm. When fingers or toes become bright red in colour on heating, the skin presenting vivid capillary pulsation, it is known that the arteries supplying them can open widely; but simple reddening on heating is consistent with much permanent diminution of arterial channels. Information as to the degree of arterial disease and its extent can be obtained by certain special tests.

*Spinal or Local Anaesthesia.*—As is well known, spinal anaesthesia blocks the spinal nerves and produces sensory and motor paralysis of the lower limbs; it also blocks the vasoconstrictor nerves to these limbs, which leave the cord by the anterior roots, and it thus induces full vasodilatation in the legs. Local anaesthetisation of the nerves of the limb, ulnar, median, popliteal, or posterior tibial, blocks the corresponding nerves; motor, sensory, and sympathetic fibres suffer, and loss of function of the last produces full vasodilatation in the corresponding territories. Thus spinal anaesthesia may be used to test the legs, and local anaesthesia may be used to test hand or foot, to ascertain if the vessels are capable of dilating. It is to be stated that while blocking the vasoconstrictor nerves causes flushing and rise of temperature in the corresponding territory in cases of spasmodic obstruction of arteries, it may also cause it where the obstruction is structural; it is the manner and degree of flushing that distinguishes the type of disease in this test. When the obstruction is spasmodic it is released abruptly and completely and the rise of temperature, once it starts, occurs rapidly and reaches the normal value of about 32° C. (89·6° F.) when the limb is exposed in a room at 20° C. (68° F.). When the obstruction is due to structural disease, blocking the nerves releases normal vasoconstrictor tone, but owing to the obstruction which still remains, the temperature rises slowly and not so high as normally.

*Heating the Body.*—Vasomotor tone may be abolished from the

skin by heating the body. The capacity of the vessels of the limb to dilate may be tested by enclosing the trunk in a chamber, which is heated rapidly by lamps to about 60° C. (140° F.), the limb to be observed being exposed to the air of the room and its reaction observed for a period usually of a half-hour or more. A similar and even more reliable method of producing general vasodilatation is to soak one arm or both feet in stirred water at about 44° C. (111° F.). Both tests act in the same way, namely, by warming the blood that returns to the heart and proceeds thence to the heat-regulating centres. These are extremely sensitive to a rise in their

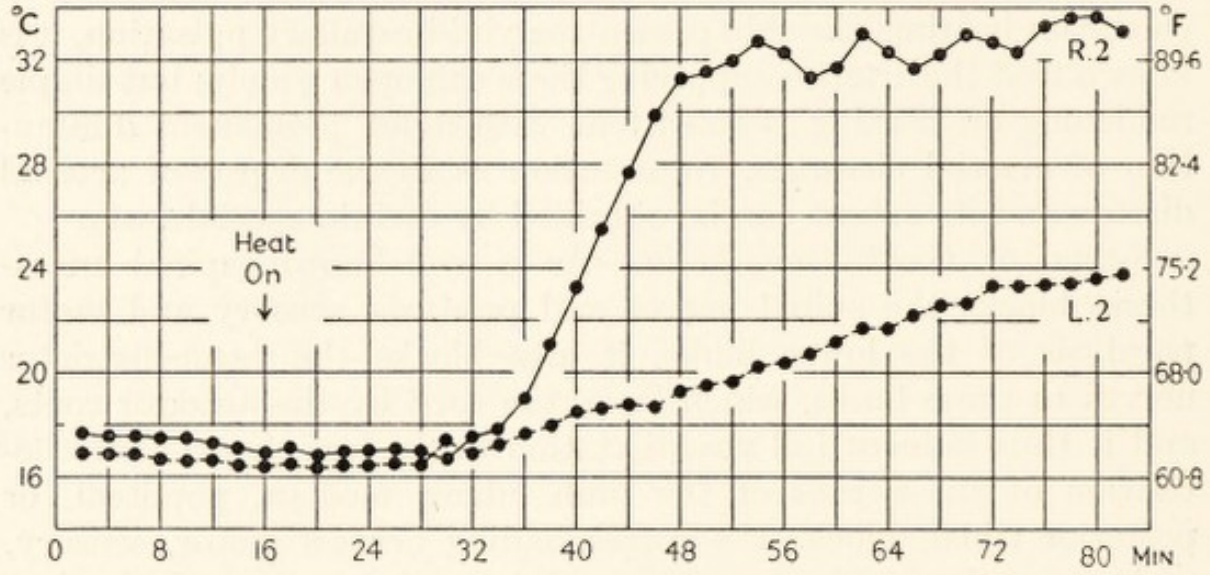


FIG. 2.—Continuous records of skin temperature from the right (R2) and left (L2) index fingers of a patient having obliterating disease confined to the arteries of the left arm. The room temperature lay between 16° and 18° C. throughout. The patient's trunk and legs were enclosed in a chamber and the latter warmed rapidly to 60° C.. As a consequence vasodilatation set in, the temperature of the right finger rising rapidly to reach 34° C., while that of the left finger rose slowly and reached 24° C..

temperature, their response being expressed in a lowering of vasomotor tone with consequent flushing and warming of the skin. In the tests attention is again paid to the height to which the temperature of the observed limb rises and to the rate at which the rise occurs (Fig. 2). This method of eliminating vasoconstrictor tone by raising body temperature cannot always be relied upon, however, in testing the legs, in which vasomotor tone may be very high and difficult to release.

*Reactive Hyperaemia.*—The tests just described, in which anaesthetisation or body heating is used to throw the vasoconstrictor nerves out of action, all require accurate measurement of skin temperature from moment to moment. The following method of testing is probably just as efficacious in estimating the degree of vascular

change; and it has the advantages of being simple in application and also of indicating the extent of arterial disease. It is necessary that this test should be applied with careful attention to detail. The limb to be tested must be thoroughly warm in its length, and for this purpose it is immersed for 10 minutes in water, which is maintained at 35° C. (95° F.); the limb is now raised above the level of the body, and if necessary massaged, until the skin is pale. The object of these preliminary measures is to ensure that vessels in spasm are relaxed and to empty the minute vessels of blood. The circulation to the limb is now arrested by throwing a pressure, exceeding the systolic pressure, abruptly into a pneumatic cuff encircling the limb. The circulation is kept arrested for 5 (or better 10) minutes, while the limb remains in the warm bath. The limb is lifted from the bath, dried, and its circulation released. The blood in returning to the limb pours into the vessels, which are now well dilated as a consequence of nutritional deprivation of the tissues. When the arteries are normal the skin becomes brightly coloured to the ends of the digits within 2 to 5 seconds of the release. In disease of the vessels, flushing of the digits may be delayed for periods varying up to as much as a minute or even longer, according to the degree of structural change. The territory affected by arterial disease is mapped out by noting the parts of the limb unflushed at the end of 5 seconds. In this test it will be noticed that in parts of a limb in which it appears rapidly, the flush lasts at the most for a few minutes, whilst, where there is disease and it is delayed in its onset, the flush lasts for much longer periods of time. This test is based upon a reaction explained on page 21.

*Arteries to Muscles.*—Normal reactions in the tests previously described depend upon the structural normality of the arteries supplying the skin of the limb. But since these main arteries of a limb supply both skin and deep structures, evidence of a defect extending high up the limb may be taken as one involving all structures. When it is considered desirable, the arterial supply of the muscles can be separately examined by ascertaining the work these muscles will do painlessly. The tests can be described more conveniently at a later stage (see page 40).

## Tests of Vasomotor Nerves

There are occasions on which it is desirable to test the integrity of the vasoconstrictor sympathetic nerves to the vessels of a limb.

This is easily accomplished by warming the trunk, or by heating a limb other than the one to be tested. General vasomotor tone is lost when blood temperature is raised, which can be done in either of the ways just mentioned. Thus, if the state of the vasomotor nerves of an arm is to be tested, the two arms should be exposed in a cool room while temperature readings are taken from right and left fingers; the trunk is now warmed, or the feet are immersed in hot water. If the vasomotor nerves of the arms are intact, the

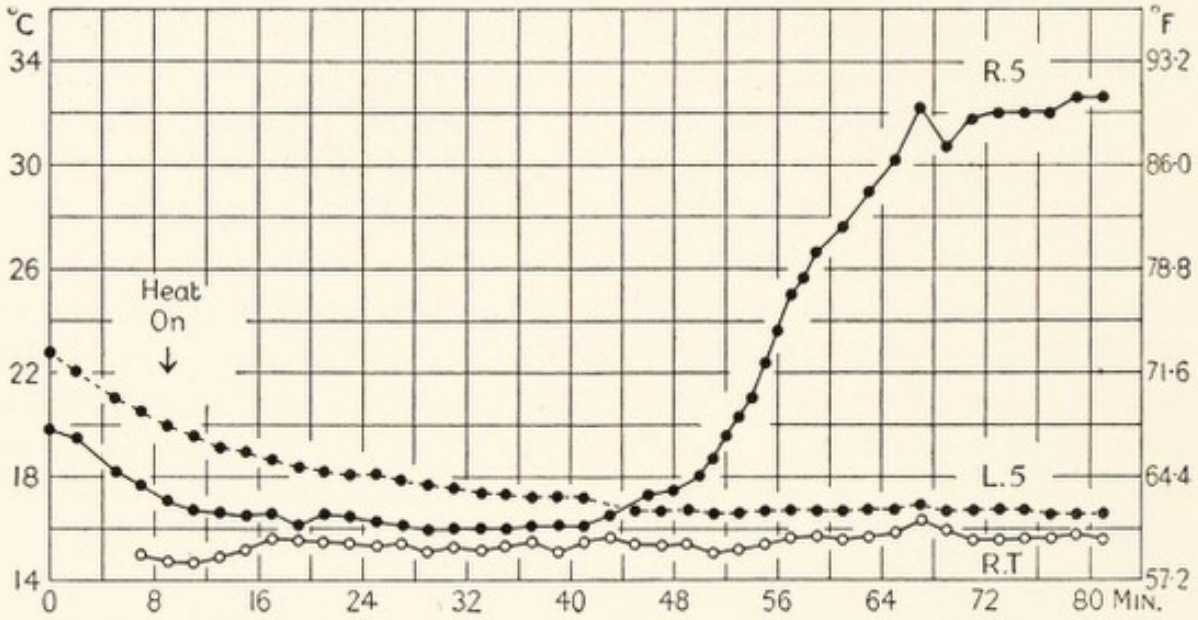


FIG. 3.—Continuous records of skin temperature from the right (R5) and left (L5) little fingers of a subject from whom the left inferior cervical sympathetic ganglia had been removed a year before. On being exposed in a cool room both fingers cooled down, the denervated finger (L5) being 2·5° C. hotter than the unaffected one. The patient's trunk and legs were enclosed in a chamber and the latter warmed rapidly to 60° C.. When vasodilatation set in, the temperature of the unaffected finger rose rapidly to 32° C., while that of the denervated finger continued to fall towards room temperature (R.T.).

temperature curves of the right and left fingers will rise similarly and together. If the vasomotor nerves to one arm have been divided or paralysed, the fingers on that side in a cool room will originally be the warmer; but, as vasodilatation occurs, it will involve the normal limb only, and the fingers on that side will become the hotter (Fig. 3). Defects of vasomotor nerve supply confined to the appropriate fingers are present and can be displayed in lesions of median or ulnar nerves. Cortical and capsular lesions such as induce hemiplegia do not effect a reaction in the limbs; spinal cord lesions prevent the normal reaction of the feet in response to heating of the arm, but not of the arm in response to heating of the feet.

# CHAPTER II

## EFFECTS OF CIRCULATORY ARREST

### Preliminary Effects

The symptoms of circulatory disorders of the limbs arise chiefly as effects of obstructed blood-flow. The effects of complete arrest of blood-flow have been observed closely in normal subjects, in whom the circulation to a limb has been stopped by inflating a cuff encircling its base to a point exceeding systolic blood pressure. These studies greatly facilitate a proper understanding of relevant pathological conditions.

#### ON TEMPERATURE

The instant the blood-flow ceases the surface temperature of the limb begins to fall (Fig. 1, page 5). The rate of fall depends on the difference between the temperature of the skin and the surrounding air, upon movement of the air, humidity, and other factors. In ordinary circumstances the fall is not rapid; thus more than a half-hour will elapse before fingers cool down from a state of ordinary warmth to room temperature. The temperature sometimes falls a degree or two below room temperature owing to evaporation. The rest of the hand cools far less quickly because its surface is smaller relative to its bulk. The cooling of the arm is for similar reasons slower still; hours elapse before its surface temperature reaches that of the surrounding air of a still room. A limb does not become " icy " cold, neither can it become cold "suddenly" through simple arrest of its circulation; such terms are frequently used in current descriptions, but they are inaccurate and often seriously misleading.

#### ON COLOUR

Within a few seconds of circulatory arrest it is usual to detect a slight but distinct paling of the skin, appearing gradually as blood drains out of minute vessels into the veins. As may be observed microscopically in capillaries of the finger-nail bed, this onward

flow lasts from ½ to 1 minute after the flow of blood into the arm has been arrested completely. The change in colour is greatest in a limb that is raised and least in one that is dependent. During the next few minutes the tint of the skin is noticed to become bluer. The change is more noticeable in parts of the skin that are originally high coloured, in the hand rather than in the arm; but the change is in fact universal. A fully cyanotic tint will develop within 2 or 3 minutes if the arm is very warm; it will develop much more slowly if the arm is cold. At the end of 3 or 5 minutes when cyanosis is usually nearly full, the depth of the vascular colouration is found to be increasing a little, for the minute vessels are now beginning to dilate in response to local accumulation of vasodilator metabolites (see page 21). Simultaneously another and conflicting process begins, leading to the appearances of small sharply defined areas of blanching of the skin. These white spots, first described by Bier, are due to minute vessel contraction of unknown origin, and they grow in size as time passes. The white spots with intervening areas of cyanotic skin form a mottling of the skin, which is characteristic of arrested circulation of long duration. In circumstances of arrested circulation with freedom of venous outlet, the white spots coalesce and the skin ultimately becomes uniformly blanched. The pallor of the skin which is usual in bodies several hours after death is a striking illustration of this effect of long lasting circulatory arrest.

## Nutritional Effects

### on nerves

Shortly after the circulation to a limb has been arrested its nerves being ischaemic gradually lose their function, resulting in sensory and motor paralysis. Different forms of sensation are lost at different rates, and the paralysis displays a characteristic march from the tip to the base of the limb. Paralysis of the fingers or toes occurs the more quickly, the warmer the limb, and the nearer to the trunk the circulation is arrested.

*Sensory Nerves.*—In the case of the arm, the first change noticed is the appearance of a little numbness at the very tips of the fingers; in a warm limb, with its circulation arrested above the elbow, this happens in 13 to 15 minutes. By the 16th or 17th minute numbness has spread to reach the bases of the fingers. At about this time or a little later sense of touch, deliberately tested, is found to be lost at the finger-tips. The numbness and the anaesthesia, which follow,

continue to spread centripetally in the arm at the rate of about 3 or 4 cm. per minute, until the whole of the ischaemic skin becomes anaesthetic within about a half-hour.

Pain sense is dulled and delayed a few minutes after numbness appears, and this change likewise spreads up the limb; actual analgesia follows much later. Sense of cold is lost about the same time or a little earlier than is that of pain, and follows the same course up the limb. Sense of warmth is among the last to disappear.

*Motor Nerves.*—Conspicuous weakness is found in the muscles of the thenar eminence by the 20th minute; these muscles are paralysed at about the 25th minute, by which time it is usual for the whole hand to have become anaesthetic; the interossei and extensors of the thumb quickly follow suit. The extensors of wrist and fingers are paralysed at about the 30th minute, producing wrist drop; the flexors of the wrist and fingers, and lastly the muscles moving the elbow joint, are paralysed in turn. Thus, the order of muscular paralysis is in general centripetal like that of sensation. Long after voluntary movement has become paralysed the muscles will react directly to electrical stimulation, faradic and galvanic.

It is particularly to be stressed that paralysis of a limb arising out of ischaemia does not happen abruptly, but is a very gradual development. Complete paralysis, extending far up the limb, does not occur until a half-hour at least has elapsed from the circulatory arrest.

*Recovery of Nerves.*—Release of the obstructed circulation is followed by very rapid recovery of all the nerve functions provided that circulatory arrest has not been prolonged unduly. Thus it occurs within $\frac{1}{2}$ to 1 minute, when arrest has lasted 25 to 35 minutes. Circulatory arrest lasting for hours permanently endangers the functions of the limbs, as will be described later.

*In the Leg.*—The events are comparable, the various forms of sensation are lost in the same order as in the case of the arm and in corresponding times. Similarly the motor loss begins in the toes and ankle and mounts to the knee. The paralysis spreads centripetally.

Experiment on animals and occasional cases of thrombosis and embolism in man have shown that if the abdominal aorta is occluded, then the paralysis may take the form of a paraplegia that comes on abruptly within a minute or two of the arrest and includes the sphincters. The rapidity of the event is here due to involvement

of the spinal cord in the ischaemia; this occurs in instances where the occlusion of the aorta is as high as the renal arteries; the cord is far more sensitive to ischaemia than are the peripheral nerves.

### MUSCLE PAIN

The nerves to the muscles are paralysed between 15 and 30 minutes from the onset of ischaemia. During the earlier period strong voluntary movements of the limbs are possible. But if a muscle continues to execute these contractions, pain is soon felt. For example, this begins in the case of the arm after about 30 or 40 strong grasping movements of the hand and increases with additional contractions. It is a diffuse, continuous, aching pain, felt over the ventral surface of the forearm and in the hand, between as well as during the contractions; it becomes exceedingly unpleasant after 60 to 90 contractions. The pain resembles that derived from muscles thrown into sustained contraction, as when a strenuous effort is maintained unduly, or when a muscle enters cramp; but under ischaemic conditions it is more severe. It continues unabated if muscular work ceases and ischaemia is maintained; the muscles are then flaccid. Thus it is not due to tonic contractions of the muscle; it is the result of the accumulation of stable products of muscular metabolism playing upon sensory nerve endings in the muscle mass. It is to be emphasised that the pain does not arise as a result of arrested circulation, but only if the muscles have done work under ischaemic conditions. The severity of the pain is governed by the strength of the contractions as well as by their number. With the pain tenderness develops in the muscle or group of muscles involved. Thus pain and tenderness are induced in the calf muscles by extending the ankle repeatedly, in the anterior crural muscles by dorsiflexing the ankle repeatedly, under ischaemic conditions. The pain remains until, with the continuation of ischaemia, the function of pain nerves is abolished or until the circulation is freed. If the circulation is allowed to return fully to the limb, the pain always disappears within a few seconds, the pain producing metabolites, which have accumulated and have been held in place, being rapidly dispersed when a supply of oxygenated blood to the muscles is restored. The rate at which the pain disappears depends, however, upon the amount of blood-flow restored to the limb.

The relation of these facts to intermittent claudication will be dealt with later (see page 40).

## After-Effects and Permanent Effects

When the period of ischaemia is short, ½ to 1 hour, recovery of sensory and motor functions is rapid and is complete. The chief after-effects of these short periods of circulatory arrest are reactive hyperaemia and disagreeable tingling.

### REACTIVE HYPERAEMIA

During arrest of blood-flow to the limbs, tissue metabolites having a vasodilator action accumulate in the tissues and produce an increasing relaxation of the vessels, small vessels of all orders being affected in this way; when the circulation is released again this vasodilatation allows a greatly increased flow of blood to the ischaemic area; the accumulated blood-flow debt is discharged, while the vasodilator metabolites bringing about this compensatory flood are removed and the flood subsides. The debt is compensated appropriately, for the intensity and duration of the hyperaemia are related to the duration of the preceding period of ischaemia. Circulatory arrest of a few seconds is sufficient to produce a distinct increase of flow subsequently. Long arrest produces an intense engorgement and reddening of the limb, which subsides in a time period equal to from ½ to ⅔ the duration of the ischaemic period. This important mechanism is one that controls peripheral blood-flow in its detail, regulating the flow to meet the needs of this area or of that. It relieves from the threat of necrosis tissues that have been rendered ischaemic and plays an important part in the establishment of collateral circulations (see page 23).

*Tingling.*—When the circulation to the whole arm has been arrested for 10 minutes and is released, tingling is almost always felt as a disagreeable symptom. It is felt especially in the tips of the fingers; it is felt over a greater area, is more intense and lasts longer, the longer the period of ischaemia. It is not due to a return of blood to the fingers in which it is felt, for it occurs if the blood-flow is released to the upper arm only. It is due to some condition developed in the main nerves of the upper arm during their recovery from ischaemia. Nevertheless it is intensified by stroking or tapping the fingers in which it is felt.

### PERMANENT DAMAGE

When the period of ischaemia is prolonged to periods of hours the nutrition of the tissues may be affected permanently. The several

tissues concerned are unequally susceptible to prolonged loss of blood supply; the relative susceptibilities of nerve and muscle are of particular interest. The nerves of the limbs lose their function under ischaemic conditions long before muscle becomes inexcitable; but long-continued ischaemia kills muscle more readily than it kills nerve. The times are not precisely known, but the death of muscle fibres is probably assured when they are deprived of blood for 6 to 8 hours, and their death results in a replacement fibrosis considered more fully under the term Volkmann's contracture (see page 35). Nerves recover after being ischaemic for 12 or even 20 hours, but longer periods lead to degeneration of their fibres and recovery is then a slow process and dependent upon regeneration. In skin, the first and very early evidence of damage may be said to be found in reactive hyperaemia. Ischaemia of the skin for periods of about 6 or 12 hours causes subsequent whealing; these or rather longer periods result in blistering; while loss of blood supply for 24 to 48 hours continuously, or for frequent shorter periods, results in necrosis of the skin.

## Occlusion of Main Arteries

Occlusion of the principal artery of a healthy human limb does not arrest the circulation to the limb and so the effects that follow, though of the same kind, are less pronounced and are different in other respects. Simple occlusion of the femoral artery at Poupart's ligament, of the brachial at the lower border of the axilla, or of the brachial at the elbow, can all be effected easily, and the occlusion maintained for a half-hour. In each case the pulse below is lost, and in none does it return within this time. The temperature of the limb falls, but the rate of fall is not half as fast as when the circulation to the limb is completely stopped by pneumatic pressure (Fig. 4). In the instance of compression of each of the arteries named, a slight grade of cyanosis develops and is maintained in the limb; in the case of femoral occlusion this cyanosis may be more conspicuous, but the discoloration never approaches the full discoloration of arrested blood-flow, and Bier's spots do not develop. Loss of sensation does not happen unless inadvertently an accompanying nerve is pressed upon. At the release reactive hyperaemia may be just perceptible for a few minutes; it has neither the brightness nor the duration of that following arrest of the circulation to the limb. These comparisons will suffice to illustrate the relatively incon-

spicuous consequences of local obstruction of a main vessel and the efficiency of ordinary anastomotic channels.

## COLLATERAL CIRCULATION

It has long been known to surgeons and to experimentalists that the principal artery of a limb can be ligated with impunity, and that the limb is almost always saved from death by the blood passing through collateral anastomotic channels. These channels, already capable of carrying enough blood to preserve the resting

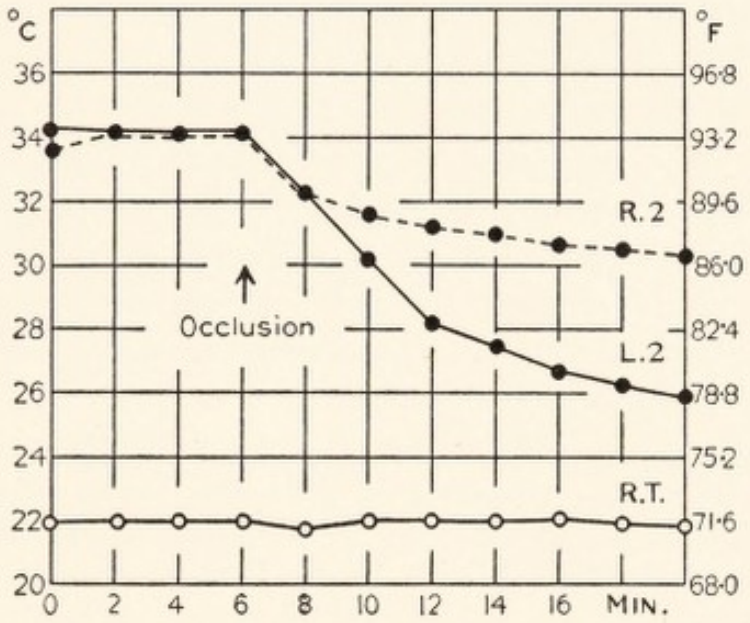


FIG. 4.—Continuous records of skin temperature from the right (R2) and left (L2) index fingers. At the instant marked by the arrow, the circulation to the left arm was arrested by means of a pneumatic pressure cuff, and the right brachial artery was occluded in the lower third of its course by digital compression. Both radial pulses were lost for the rest of the observation. The temperature fell much farther on the left side, on which the circulation was stopped completely.

limb, quickly dilate. Experiment has shown that much of the stream is restored within 3 to 10 minutes of tying the main vessel, and in carnivora the distal pulse usually returns within 24 hours, and sometimes much earlier. Records of precise observations upon man are few; one of the clearest statements is by Makins. After an unusually rich experience in tying wounded arteries during the war, he stated that after ligating the brachial artery 3 or 4 days suffice for the return of a weak radial pulse, and that after ligating the femoral artery the posterior tibial pulse remains impalpable for weeks. The formation of full anastomotic channels takes a long time; it entails growth in the walls of the arteries. The anastomotic artery, while preserving walls of appropriate thickness, becomes increased in diameter; it also becomes longer, and therefore sinuous.

Actual growth has been detected as early as the 6th day; it may be conspicuous in 6 or 8 weeks (Nothnagel). Thus, there are two processes: a dilatation of anastomotic channels, a process beginning within a few minutes; and a growth of vessels, a slow process occupying weeks, months, and sometimes years.

An early and too simple view of the development of anastomotic channels was that the rise of pressure behind an obstruction in an artery distends the vessel and its proximal branches. It was contradicted by the observation that the part of the artery between a ligature and the next proximal outgoing branch becomes constricted and ultimately obliterated. It was inadequate to explain the fact that dilatation occurs particularly or exclusively in those arteries, dilatation of which is appropriate to re-establish proper flow where it is needed. Only branches carrying blood to the anaemic area dilate; branches that may lie nearer to the tie remain undilated if they run to tissue the blood supply of which has been undisturbed. Actually the rise of pressure behind a ligature is insignificant and could not be held to account for either the immediate or delayed phenomena of collateral circulation.

It is the rule for the branches of an artery to link with each other to form a more or less well-defined network. When the main artery is tied, an earlier outgoing branch now pours its blood into its original territory and also into the territory of more distal branches, territory in which, owing to the tie, pressure has fallen. Thus the resistance to flow through the branch proximal to the ligature is substantially reduced from the instant of ligation, and flow through it consequently becomes increased. This early increase of flow through collateral channels is also encouraged by changes in the tissues of the affected territory. As has been described, when the circulation to a limb is stopped, the vessels of the affected tissues dilate; this dilatation is independent of both central and local nervous influences, it depends on the vasodilator action of metabolites accumulating in tissue deprived of blood. Owing to this mechanism any territory through which blood-flow is imperfect shortly becomes a region of vasodilatation and of lowered resistance to blood-flow.

The later process, entailing growth of vessel, should be regarded separately; the growth of arteries is associated with increased blood-flow through them. Long-continued and varied studies of Thoma convinced him that an artery varies in size in response to the amount of blood carried through it. An artery through which blood-

flow is decreased tends to close and its walls to atrophy, as illustrated by the great diminution ultimately occurring in the size of the main artery in the stump of a limb after amputation, and in the stretch of artery between a ligature and the next proximal outgoing branch. On the other hand, an artery through which blood-flow is increased grows in width and length, best illustrated perhaps by the great expansion that occurs in an artery on the proximal side of an arteriovenous fistula. It is established that there is a relation between growth and rate of flow. The changes, atrophic or hypertrophic as the case may be, have been regarded as resulting from changes in the artery's nutrition; this explanation, however, is probably inadequate. It is known that collateral anastomoses form whether the nerves to the limb are intact or not.

## CHAPTER III

### EMBOLISM AND THROMBOSIS OF MAIN ARTERIES

In embolism of the systemic arteries the clots come usually from the left auricular appendix or from the wall of the left ventricle. The most common heart conditions associated with embolism are mitral stenosis, auricular fibrillation, and failure with congestion, alone or in combination. In bacterial endocarditis the aortic and mitral valves are the main sources of detached thrombi. In coronary infarction emboli are derived from thrombi formed on the infarcted and necrosed wall of the left ventricle. Another source of embolic clots is the sac of an aortic aneurysm.

Emboli vary much in size and upon this depends the size of artery in which they become impacted. They are generally arrested where the blood passes abruptly from an artery having a greater to one having a distinctly smaller lumen, namely, where an artery divides into two branches, or where a main artery sends off a large branch. The bifurcations of the brachial, of the common femoral, and of the popliteal artery are common sites. Usually the clot will block both branches of such arteries completely; but it may lodge in and block the mouth of one completely, while protruding into the other channel and partially obstructing it.

A clot that has been brought to rest may move subsequently—it tends to be driven on and to become more tightly impacted; this movement may carry the embolus a little away from an arterial branching point and thus free one branch; it may be driven right on to the next dividing point. An embolus which rides across the saddle of an arterial subdivision may break into two parts, which pass on into the branches.

An embolus when arrested, temporarily or permanently, gradually gathers more clot, and comes to occupy more of the artery and its branches in their length. It is usual for clots to be found occupying many inches of the lumen of the main artery of a limb, and occasion-

ally the clot may extend through at least half the limb; probably the length of the clot is the reason why recanalisation with reopening of the vessel is rare. Clot riding over a saddle may extend far down a vessel without blocking it; it may trail free, it may become attached throughout its length to one wall only, or it may become attached in such a way as to be channelled.

Because thrombus forms upon an embolus, it is often exceedingly difficult to distinguish between a primary embolus or thrombus from the appearance of the lesion unless this is seen very early. Serial sections of the clot may reveal what is interpreted to be an embolus in its centre; a clot that is smooth and everywhere unattached to the vessel wall is embolic, but attachment and lack of smoothness are by no means decisive evidence of primary thrombosis. Judgement as to whether the clot has formed in the artery or been brought to it often depends on the nature of the malady with which it is associated, thrombi being particularly frequent in severe infectious disease such as typhoid, pneumonia, cholera, malaria, in marasmic states, and after severe operations; while emboli, as stated previously, are derived mainly from the heart, which on examination may provide decisive evidence by exhibiting the broken surface of a thrombus, which still remains attached to the wall.

## Symptoms

So far as is known there is no difference between the symptomatology of embolic and of thrombotic obstruction of an artery. With very few exceptions the symptoms and objective manifestations of both forms of obstruction are the direct or the indirect results of ischaemia. The effects of arresting the circulation to a limb and of closing a main artery have been described already, but neither of these descriptions is fully adequate. An intravascular clot rarely stops the circulation to a limb completely, for collateral channels are able to open up; and the effects are more profound than those of simple ligation for two reasons, namely, the vessels that would form the collateral channels are often diseased, and clotting in an artery is often so extensive that the mouths of important collaterals are closed.

Other things being equal, the position of the obstruction governs the severity of the manifestations, the nearer to the aorta the main artery is blocked the more profound is the disturbance. Thus symptoms are usually definite or severe when clots close any part of the main artery of the arm down to the brachial bifurcation, or of

the main artery of the leg down to the end of the popliteal artery. But the effects are very variable, even when these main arteries are the ones to be affected; there may be no symptoms and the obstruction may remain unsuspected until the distal pulse is missed; on the contrary, massive gangrene may occur.

*Local Signs.*—At the seat of obstruction the artery is dilated and contains a mass of firm clot; this may form a palpable cylindrical pulseless tumour, more or less sharply defined. Pressure over such a clot may or may not elicit tenderness; tenderness tends to develop on the days after the clot has formed. The tenderness is the result of inflammation of the adventitia and periarterial tissues, and it occurs most frequently and is most prominent in instances of the infected emboli of bacterial endocarditis.

When the main artery of the limb is completely plugged, certain changes occur distally. They have not been studied so carefully as have the changes following experimental arrest of circulation to the limb, though it is clear that the changes in the two instances are similar. The following statements can be made from actual case records of thrombosis and embolism.

*Pulse.*—The distal pulse is lost, and reappears only on rare occasions.

*Colour.*—Distal parts of the limb are at first pale; shortly they become cyanosed, then cyanosed and mottled, and later they develop in severe cases a more uniform cyanosis with congestion, before finally displaying the browner or blacker tints which mark actual necrosis. The changes of colour do not occur up to the level of the obstruction but begin about 6 to 12 inches short of this; they increase in degree as they are traced downwards, the most severe changes being confined to the most distal parts of the limb.

*Temperature.*—The temperature of the limb falls gradually if it is exposed to cold surrounding air; it falls fastest in the digits and here comes to approach the temperature of the surrounding air most closely. The patient often complains of a sense of coldness in the limb or of coldness of its contact with the normal limb.

*Motor Disturbances.*—Ischaemia of motor fibres in the peripheral nerves leads to weakness of the corresponding muscles, movements at the several joints are affected in a centripetal order. Muscular weakness, when present, is observed by patients who are using the limbs at the time. The muscles when examined are flaccid, though occasionally there is a complaint of cramp.

*Sensory Disturbances.*—The sensory disturbances are several. The

commonest is numbness. In the legs this symptom is rarely recognised by the patient until the diminished sense of touch is extensive; that is so because moving contacts are required to elicit the symptom, and because the patient's attention is often concentrated upon pain. It develops slowly, starting at the ends of the digits and spreading up the limb (see page 18). Sense of touch is lost earlier than sense of pain; analgesia like anaesthesia spreads gradually up the limb. A limb presenting extensive loss of sensation is one to which the circulation has been lost for very many minutes.

Coarse tingling described as "pins and needles" is not infrequent. It does not occur during the gradual failure of sensation, which follows abrupt and complete cessation of circulation in a limb (see page 21); experimentally, it is only known to be felt in response to an inflow of blood to a limb that has been ischaemic for many minutes; a slight temporary inflow suffices.

*Pain.*—This is the commonest initial complaint of the patient. When present it is almost always located at a point distal to the vascular clot; though it may be level with it, as when an embolus lodges in the profunda femoris. There may be no pain even in cases of clots obstructing the main arteries high in the limb. Once it comes, the pain grows rapidly in intensity, is described as continuous, diffuse, and aching or cramp-like in character; it is distressing owing to its severity or to its long duration. It may last for hours or even days.

*Cause of Pain.*—The view often expressed that a clot, carried onwards until it jams, causes pain by impact is unacceptable. Clot is soft; it has a specific gravity equal to that of blood, and the momentum of a blood-clot is no greater than that of a similar volume of blood. The idea was due to the frequent statement by patients that the pain comes suddenly, thus giving rise to the assumption that pain follows embolism as it follows a blow, on the instant; but no case is known in which the pulse has been observed to vanish at the instant the patient cries out in pain. Actually the instant of embolism of a limb is almost always unknown; it is timed only by symptoms. It is clear from observations on arrest of the circulation to a limb that both coldness and numbness of a limb are symptoms that come after considerable delay; neither of these symptoms can arise suddenly after embolism. Frequently a sense of coldness or extensive numbness is noted as early as the onset of pain, and sometimes one or both precede pain. From such observa-

tions it is manifest that pain may be a delayed symptom, even when it comes suddenly. Probably it is usually or always delayed. It is certain that the initial severe pain of embolism, which is being discussed, does not arise at the site of the clot; it is not felt there but nearly always much lower in the limb; moreover, if the artery is thoroughly infiltrated with novocaine the pain remains unabated.

It is highly significant that pain occurs only in cases in which large vessels are blocked and in which evidences of widespread ischaemia appear; it is to ischaemia and to no local event that the pain discussed is due. Embolisms of the smaller arteries of the limb occur painlessly, because they are not followed by such ischaemia. The pain in thrombosis and in embolism are indistinguishable; it is the obstruction that matters and not the manner of it. As has been related, the pain of muscular ischaemia, deliberately induced, is maintained until the circulation is released; the pain then vanishes within a few seconds. The pain of arterial embolism is similar in character; likewise it is long drawn; but if the clot shifts and frees the channel the pain disappears promptly. The removal of clot does not relieve; it is the return of blood-flow. The clot can be removed surgically between clamps, and either clamp can be undone, without changing the pain; but when both clamps are taken away the pain vanishes as soon as the stream of blood to the limb becomes restored. Another fact supporting the ischaemic source of the pain, and muscle as its point of origin, is the tenderness that is often elicited by pressure over the muscles involved. This tenderness must be distinguished from that which sometimes develops one or two days after embolism out of periarterial inflammation at the seat of the clot.

Recognising that the initial pain of embolism is ischaemic and frequently, if not always, muscular in origin, we are able to understand how in one case numbness, and in another pain, appears first; a limb that remains quite still after losing its circulation becomes completely numb before pain arises; a limb that is working at the time of arterial obstruction develops severe pain within a few minutes.

Pain as a severe initial symptom of embolism is the rule when the artery supplies a limb, the heart, or the bowel; these organs all contain a large proportion of muscle. Embolism of non-muscular organs like brain and spleen is painless.

## Embolism or Thrombosis of Given Arteries

It is difficult to describe the symptoms and signs of occlusion of this or that artery accurately, because the clot extends in them and in their branches for variable distances, and branches that should serve as collateral channels often become blocked simultaneously. A brief description in the case of chosen arteries will serve to indicate the usual extent and severity of the symptoms and signs, and to form a guide in the diagnosis of the level of obstruction.

*Aortic Bifurcation.*—At the bifurcation of the abdominal aorta the clot frequently extends down into the iliac and even into the femoral artery. Pain is usually severe and felt diffusely in the lower limbs; it may be confined to the foot or to the thigh. Sometimes there are preliminary pains in the loin, associated with haematuria, or colic in the lower abdomen. The limbs become flaccid and cold, and movement is lost from the knee joint or even from the hip joint downwards. Discoloration and sensory loss extend up to the middle of the thigh or even to the groin. These manifestations usually proceed to gangrene of the toes, of the foot, or even of the whole leg. The symptoms and signs are usually symmetrical, with loss of all pulsation in the limbs; but they may begin unilaterally and may remain more pronounced on one side than the other.

*Common Femoral.*—The pain may be confined to the foot, to the lower leg, or to the region of the knee; it may be felt on rare occasions as high as the thigh. Movements of the toes and usually of the foot are lost; the foot becomes anaesthetic; numbness, coldness, and discoloration usually extend to the region of the knee. Gangrene of toes, of the foot, or of the leg may follow.

*Popliteal.*—There may be no symptoms. Pain is generally confined to the foot but may occur in the lower part of the leg. Movements of the toes are usually lost and those at the ankle may be weakened. The foot shows discoloration, coldness, and numbness. Gangrene of a toe occurs occasionally.

*Axillary.*—Pain may occur in any part of the limb from shoulder to hand, movement of the fingers and at the wrist is lost, and at the elbow it is often weakened. Discoloration, coldness, and numbness usually extend up to the middle of the forearm. Gangrene of fingers and occasionally of larger portions of the limb may occur.

*Brachial Bifurcation.*—There may be no symptoms. Pain occurs in the hand and forearm. Movements of the fingers and even at the wrist may be weakened. The fingers often become discoloured and

numb, and these changes may extend up to the middle of the forearm or even higher. Gangrene of the terminal parts of fingers may occur.

*Smaller Arteries.*—Occlusion of the radial or ulnar or of the anterior or posterior tibial arteries usually gives rise neither to symptoms nor to signs other than loss of the corresponding pulse.

## Course

The course of the malady in cases of non-infective embolic closure of main vessels is very variable. It varies with the artery closed, and depends largely upon whether gangrene supervenes or not. When the clot obstructs the bifurcation of the aorta, massive gangrene and death is usual. Occasional cases have recovered movement and sensation, with or without subsequent intermittent claudication. Recovery is more likely after surgical interference than without it. In obstruction of the common femoral artery gangrene is frequent and, when it occurs, death is not infrequent. In simple popliteal and brachial obstruction gangrene is less frequent, and complete recovery of function is the rule. When arteries of a smaller order become blocked, recovery of all functions is the rule.

The course also varies owing to events at the site of the original obstruction. Sometimes a clot moves on from a bifurcation at which it blocks both branches to a position in which it blocks one only; in such instances rapid recovery from all or from most symptoms may follow. In other instances the evidences of ischaemia rapidly increase, indicating spreading thrombosis or new embolism. In cases where gangrene does not supervene rapidly, the usual course in obstruction of the main artery of leg or arm is one of steady improvement, with subsidence of pain and return of natural colour as collateral channels open. Thus it is not uncommon for signs of ischaemia to decline rapidly within the first few days after the accident, even when the common femoral artery has been obstructed. But the pulse in such cases does not often return, and the last stages of functional recovery are often very slow and incomplete. Weakness of movement and dulling of sensation may last for weeks, or may remain so long that ultimate recovery depends on nerve regeneration. The vascular supply of the muscles may be reduced permanently and the patient eventually exhibit the phenomenon known as intermittent claudication (see page 38). In rare cases the acute failure of its blood supply is followed by death of muscle and contracture (see page 35).

When emboli are infected the course is modified by local changes. According to the activity of the inflammatory process, infected clots produce tenderness, tenderness with swelling and other signs of inflammation such as local reddening of the skin, local aneurysm, and local abscess.

### Treatment

Treatment consists of measures designed to release the obstruction in the artery, and of after-care.

When the chief artery of a limb is blocked by clot and the clot is allowed to remain in place, the block in that artery may be regarded as in general permanent. It is the permanence of the obstruction that justifies surgical intervention, for even if this fails, the state of the artery as a conduit is no worse than before. It is to be remembered, however, that numbers of these cases recover without interference, owing to the opening of collateral channels, and operation is to be recommended only in those cases in which pain is severe and prolonged, or the signs of motor and sensory loss are extensive, profound, and stable. A few hours may be allowed to elapse from the onset of symptoms, but if removal of the clot is to be attempted it should not be long delayed ; delay means extension of the intravascular clot, with adherence of the clot to the vessel wall; and adherence to the wall, while hampering clean removal, encourages thrombosis to recur. Thus, if operation is deemed advisable, the sooner it is done the better; the chances of success rapidly decrease after 12 hours, and are almost gone after 48 hours have elapsed.

The operation of embolectomy is usually undertaken under local anaesthesia, and entails exposure of the artery and removal of the clot through an incision in the wall of the artery between clamps, each clamp being then loosened separately to ascertain that the vessel is clear by observing a flow of blood. Portions of clot lying proximally are sometimes washed out by the blood current aided by massage, or by instrument introduced into the vessel; clot may be drawn or sucked up out of the distal segment of the vessel. The artery is sutured and the clamps finally removed. If the obstruction has been removed, pain is relieved, and the limb becomes bright-red in colour (reactive hyperaemia); the distal pulses reappear. In the hands of skilful surgeons this operation has proved conspicuously successful on many occasions, leading to complete and permanent restoration of blood-flow through the main artery of the limb. In a number of cases clot has been removed successfully even from the

bifurcation of the abdominal aorta and from its immediate branches. Instances are on record in which dangerous emboli have been removed successfully from two limbs of the same patient.

Many of the patients are cardiac cases and surgical interference may on that ground occasionally be deemed inadvisable. Sometimes it has been found possible to massage the clot onwards in the artery with conspicuous relief of symptoms.

When an embolus or thrombus has blocked an artery and the time for surgical interference has passed, a return of circulation through collaterals should be encouraged by keeping the body of the patient warm rather than by heating the limb (see page 49). Muscular pain if present will pass away; movement will increase it; so while pain persists the limb should be kept at rest. Otherwise passive movement and massage will encourage recovery. Massage may be used, and continued in cases in which sensory or motor loss has become stable.

The appearance of gangrene will call for special treatment; this is outlined in Chapter V.

# CHAPTER IV

## POST-ISCHAEMIC CONTRACTURE; INTERMITTENT CLAUDICATION

Two states arising in limbs to which the circulation has been arrested or reduced need separate consideration; they are Volkmann's post-ischaemic contracture and intermittent claudication.

### Post-Ischaemic Contracture (Volkmann's Contracture)

Contracture of this kind has been brought about most usually in cases of fracture or dislocation in the region of the elbow in young subjects, and has been the result of the overtight splinting of the forearm. It can also happen in those cases where the elbow is fixed in an overflexed position and local swelling occurs. Similar contracture may arise occasionally in other ways to be mentioned presently. It may be produced in the lower instead of in the upper limb; but the forearm has been the affected part in the great majority of cases and serves best as present illustration.

The story is nearly always of considerable pain, following in the first few hours after the bandages are applied and frequently accompanied by swelling of the hand; the paralysis of the forearm muscles, with rigidity when passive movement is attempted, is discovered when the wrappings are removed next day, by which time the mischief is done. Contracture gradually appears during the following weeks, the wrist becoming flexed, metacarpo-phalangeal joints extended, and the last joints of the fingers flexed, to constitute a characteristic and disabling deformity of "main-en-griffe" type within the short space of a few months.

It was Volkmann who in first describing this malady recognised that the muscles of a limb are brought to a massive state of ruin by being deprived of their blood supply. He concluded that muscles so treated enter a state of post-mortem rigidity, which from its

onset prevents passive movement and precludes recovery of the affected fibres. Volkmann's conclusion has been substantiated fully by subsequent observation and experiment, though the nature of the malady has from time to time been a focus of controversy, largely owing to the fact that nerves and other structures in the limb have often been injured in the original accident or have also been exposed subsequently to pressure.

To understand and to recognise this form of paralysis it is particularly necessary to separate it from forms arising out of injury to peripheral nerves. That the muscles are, and the nerves are not, the seat of damage is proved, shortly after the bandages are removed, by the absence of sensory defects in the skin in uncomplicated cases, and by failure or relative failure of the muscles to respond to all electrical currents. The changes in the muscle fibres, their early rigidity and opacity, their rapid degeneration with loss of nuclei, the reactionary and long-lasting local inflammation, which follows and leads to replacement fibrosis, may also be cited; these things are known to occur and also characterise ischaemic necrosis of muscle induced experimentally; the picture is distinct from the flaccidity and secondary atrophy in muscles following nerve degeneration. The pathology has been confused by the simultaneous occurrence of nerve injuries in some patients, distinguishable usually because they present from the start the well-defined features of lesions of the peripheral nerves of the forearm. Characteristic contracture is said to have followed the use of tight rubber bandage or strap; there are more records of nerve than of muscle injury following such bandaging;[1] records of true ischaemic contracture following this procedure are in fact rare. The reason is that the rubber tourniquet is not deliberately applied for a sufficiently long period of time; for when the period is adequate to produce necrosis of muscle, it is evidently long enough to bring risk of gangrene to the whole limb. Similarly, arterial thrombosis and embolism can rarely be expected to yield

[1] *Paralysis from Pressure.*—If a narrow band or cord is tied very tightly around a limb it may directly damage the main nerves. Paralyses of this origin have been described in men arrested and bound. Locally the nerves of limbs so treated may be narrowed until their ordinary microscopic structure becomes unrecognisable. The paralysis which follows is characteristic of local nerve injury, being both sensory and motor; but the muscles respond through their nerves to faradism applied directly below the region of damage. These cases differ curiously, however, from those of nerve transection in that the nerves do not usually degenerate; movements may begin to be restored in a few weeks and may be normal in a few months.

transient loss of blood-flow for just the adequate period to produce death of muscle only; there is either recovery by the opening of collateral channels, or all tissues of the limb succumb. But enough cases of one or other type have been recorded to show clearly that ischaemia by itself, in the absence of preliminary injury to the limb, or possibility of serious pressure on nerves, will determine contracture.

The time that the muscle of a limb must be deprived of its blood supply to ensure the subsequent occurrence of contracture is not accurately known from observation upon human beings; it is established that compression of the limb for 24 hours is enough, but there are indications from experiment upon animals that a period of 6 to 10 hours is adequate, and usually required, to produce long lasting damage to the muscles. Such times are similar to those known to elapse after death in ordinary circumstances before rigor mortis is at its height in man.

It is important from both the practical and theoretical standpoint to realise that it is unnecessary that the main artery of the limb should be compressed and occluded by splint pressure. Much lower pressure than that required to collapse the main artery will, when exerted directly upon the muscle itself, collapse its capillaries and completely arrest the blood-flow to it. A pressure of 50 or 60 mm. Hg upon skin is known to stop all blood-flow to it; and pressures of this order will have a similar influence upon the circulation to muscles. The point is very relevant to the mechanism of the disease discussed, and has not as yet received adequate attention. It is notable that necrosis of the skin and other superficial tissues is often described as developing over the front and upper part of the forearm in patients in whom splint pressure subsequently leads to contracture; evidently splint pressure has here produced anaemia and necrosis of the skin directly and not indirectly through arterial compression, and this consideration points to the muscular necrosis arising similarly. Two further facts strongly support this view, namely, that the pulse has been noted at the wrist before the bandages have been removed from those subsequently developing contracture, proving that the ischaemia has not been general to the limb. And cases are described in which destruction of muscle has been confined precisely to the region pressed upon by a tight elastic bandage, there being no affection of the muscle distal to the bandage; this delimitation is explained by the muscle being rendered ischaemic only where it has experienced direct pressure.

### MANAGEMENT

Ischaemic contracture has become a comparatively rare malady since the usual manner of its production has been recognised. Its avoidance depends upon the use of well-moulded splints, padded to give elastic and well-distributed pressure, and upon early re-examination of the limb with immediate redressing if pain supervenes. In considering measures intended to avoid ischaemic contracture, it is to be emphasised that the presence of the pulse at the wrist or of normal sensation in the hand is no gauge to safety; the pressure that renders the muscle ischaemic may be pressure exerted directly upon it rather than upon the brachial artery or its branches.

When muscle fibres have been rendered ischaemic sufficiently long to destroy them and the limb shows clear signs of paralysis, recovery depends upon the state of the remaining fibres and their ultimate hyperplasia. Massage, and passive and active movements, should be undertaken as soon as is possible, and as thoroughly as possible in the circumstances of the case. Apart from these measures the ultimate treatment is surgical, and in skilful hands much improvement is to be obtained by dividing tense bands of fibrous tissue, by tenotomies, followed by re-education. The earlier belief that crippling is permanent has given place to much greater optimism, as the management of the condition has improved and as instances of advanced or complete functional recovery have been observed increasingly.

## Intermittent Limping or Claudication

The term was first used by the veterinarian Bouley in 1831 to describe a condition of limping in the horse, developing after a short period of exercise and recovering rapidly with rest, but recurring when the exercise was repeated. In the horse the condition was found to be associated with obliterative disease of the main artery of the leg. A similar condition was soon described in human patients, and came to be recognised as a manifestation of gross disease of the arteries of the affected leg. When the syndrome was described in man it became known that limping was the result of pain, and pain was realised to be the chief symptom.

Intermittent pain in the leg, coming on rapidly on walking, occurs in a variety of maladies, the most frequent being the arterial disease

of the elderly or diabetic, and thromboangeitis, all conditions in which the arteries are gravely diseased and the flow to the limb severely reduced. It also occurs in cases of aneurysm of the main artery of the leg, arteriovenous fistula, old standing embolism or thrombosis of the main artery, and in occasional cases of coarctation of the aorta. In all these instances there is the same common factor, namely, an obstacle to the free passage of blood to the affected limb. Not infrequently, in cases where coronary arteries are also diseased, it is associated with angina of effort; the relation of pain to exercise presenting remarkable similarities in the two maladies.

The pain experienced by these patients is precisely that previously described as resulting when normal muscles are made to work with the circulation to the limb completely arrested. It is a diffusely felt, continuous pain, sometimes described as cramp-like because of its resemblance to the pain experienced in cramp. The early idea that the attack is one of actual cramp has had to be abandoned, for tonic spasm of the muscles supervenes very occasionally and is by no means essential or even usual when pain comes. The pain rises rapidly in intensity until it causes great discomfort or distress. Pain of similar character is often felt by healthy subjects over the front of the lower leg when skating in tightly laced boots, or in the calves when walking fast in tight puttees; the reason is in all instances to be found in relative ischaemia of a working muscle. The pain produced in a working limb to which the circulation is arrested is stable, continuing after muscular exercise ceases and so long as the blood-flow remains arrested. The distinction between this pain and that of intermittent claudication is that in the latter case the pain subsides in a short time if exercise ceases. It subsides because the blood-flow, though inadequate during actual exercise, is sufficient to restore the resting muscle to a more normal state and to keep it so as long as no further exercise is undertaken. A proper understanding of the mechanism permits a clear interpretation of symptoms and affords a gauge of the severity of the vascular lesion. The narrower the arteries the smaller will be the supply of blood, and the more closely will the behaviour of the pain approach to that experienced during arrest of the blood-flow. The smaller the supply of blood the earlier will the pain come during walking, and the more slowly will it recede when the subject rests. In the most severe cases the pain comes almost, if not quite, as quickly as it does if the circulation to the limb has been stopped, and it lasts after exercise has stopped for very many minutes. In the least severe cases the pain is felt only

after walking very fast and for a considerable distance, and it vanishes almost at once on halting.

Although any gauge of intermittent claudication is, strictly speaking, an index of the vascular supply of the muscles used in walking, in practice it may be used as an index of the vascular supply to the limb as a whole; for it is disease of main arteries, rather than of their small branches, which is usually responsible for the relative ischaemia of muscles with which we meet clinically.

Some patients provide sufficiently clear histories of their muscular pain to make its recognition easy; they tell of the conditions under which pain comes in a way that provides a useful gauge of the severity of the affection. It is interesting to learn from such people how constant may be the amount of physical exercise inducing pain, constant enough to bring them to a halt at the same place in the road day after day. But sometimes in dealing with such people, and more often in dealing with less observant or less intelligent subjects, specific questions must be asked or appropriate tests must be applied. In questioning patients with a view to determining the presence of a suspected muscular ischaemia, close attention should be paid to the continuous character of the pain, and to its location over the region of the muscles used. Answers will be more reliable if the questions are put while the patient is actually feeling pain, induced in the ordinary way. Where necessary, pain may be induced for comparison in the symmetrical limb, by exercising it with the circulation artificially arrested. In patients suffering from intermittent claudication it is generally possible to bring on pain and tenderness in the region of the calf by persuading the recumbent subject to press the balls of the toes firmly and rhythmically against a resistant surface by plantar flexion; and in the same patients it is usually possible to bring on pain and tenderness over the anterior surface of the leg by persuading them repeatedly and strongly to dorsiflex the foot against resistance. Such tests are not only valuable on occasion in deciding the characteristics of the pain, and thus aiding diagnosis, but they are of particular value in gauging the severity of the malady and its progress. Here the test, as far as circumstances allow, should be a standard one, the movements being at a known rate and of a force repeatable on different occasions, and the gauge will be the number of movements required to induce pain, checked by the time this pain takes to subside after movement is stopped. In gauging progress the patient's unaided experience may be misleading; some learn to escape pain in walking

by throwing less strain on the affected group of muscles, and here deliberate tests of these muscles may show that improvement is imaginary and not real. A test often has value when applied to the leg of which there is no complaint. If the two legs are both affected, but unequally, and this is rather the rule than the exception, the patient will almost always complain of pain in the worse limb only. That is so because pain in the worse limb restricts walking to a point that is less than adequate to invoke pain in the sounder limb; thus in everyday life the sounder limb really remains untested, but its defect can be displayed by exercising this limb separately. It is to be recognised, too, that these patients' tolerance of exercise may be masked by breathlessness or by anginal pain; this, by limiting the exercise taken will conceal a weakness in the legs, just as, reversely, a severe intermittent claudication may conceal angina of effort by prohibiting the amount of exercise necessary to induce the latter.

In patients tending to display intermittent claudication, the addition of a state of general anaemia aggravates the malady by reducing the oxygen supply to the muscle; the appropriate treatment of the blood state here leads to improvement of the local symptom.

Lastly, it is to be pointed out that pain, comparable with that resulting in intermittent limping, occurs in similar conditions of the vessels of the arm. In the arm such pain is less frequent because obliterative disease of arteries of this limb is far less common than it is in the leg. And, when it occurs, pain does not form so dominant a complaint, because movements of the arms are not often repeated with the automatic rhythmicity which is customary in the case of the lower limbs.

The treatment of intermittent claudication is inseparable from that of the care of the limb as a whole, which is considered in the next chapter.

# CHAPTER V

## ARTERIOSCLEROSIS; THROMBOANGEITIS OBLITERANS

### Arterial Disease of the Elderly and Diabetic

The term arteriosclerosis, while forming a convenient chapter heading, will be used no further, for it has come to include different lesions affecting one or more coats of the arteries. Any one of these lesions may lead to decreased elasticity, dilatation, or deformity of an artery, to narrowing of lumen, or to a combination of these changes; the lesions are of unknown origin and no attempt will be made to distinguish them. Here we are concerned only with changes in the vessel that interfere with its function as a conduit of blood. Such lesions are located in the intima; they may or may not be associated with changes in the media that are characterised by calcium deposited as plaques or rings. The intimal lesion may consist of elastic or connective tissue proliferation, with or without atheromatous change, or of similar processes combined with the formation and organisation of thrombi. The ways in which these changes arise and develop are still controversial questions. It must suffice to state that they lead to gradual narrowing of the vascular channels conveying blood to the extremities and often culminate in obliteration. The narrowing leads to a slowly developing and chronic state of relative ischaemia of the limb. It occurs most conspicuously in the lower limbs, though not confined to them, and especially in the popliteal artery and its branches.

Arterial disease, such as is responsible for serious limb symptoms in the elderly, is much more frequent in males than in females, and is often, though by no means always, associated with high blood pressure. Many of the patients suffer also from angina pectoris or from cerebral disturbances. The arterial disease of diabetes also occurs much more often in males than in females, and this despite

the greater prevalence of diabetes in elderly women; it is usually complicated by high blood pressure; it may be accompanied by any of the serious manifestations of diabetes.

### SYMPTOMS

Although the arterial disease is bilateral, its degree usually differs sufficiently on the two sides to make the symptoms unilateral for months, years, or indefinitely. Premonitory symptoms are absent or inconspicuous, consisting of a sense of coldness or feeling of heaviness in the limb, with perhaps weakness, and a little numbness or tingling. Often there is a complaint that the feet cannot be warmed in bed; cramp in the legs occurs more frequently than in the unaffected, but is not distinctive.

In most cases symptoms do not occur until the disease is advanced and signs of deficient blood-flow are obvious. A first complaint may be that a toe has become numb, that the leg is discoloured or that it has become swollen, but usually the first insistent complaint is of pain. This may be of several kinds.

Intermittent claudication, already fully described in the last chapter, is frequent, the pain being felt usually in the muscles of the calf, in the lower part of the front of the leg, and occasionally in the foot; it is recognised by its continuous aching quality, by its relief with rest and aggravation by movement, and by the possibility of provoking similar pain deliberately, changing its location, and that of the tenderness associated with it, by changing the group of muscles tested.

The foot or leg is often discoloured, the toes and dorsum of the foot being especially prone to display an increased depth of colour and a tint more than normally cyanotic. Such discoloration is associated with coldness of the skin in which it appears, and is attributable to faulty nutrition, or to actual damage by cold, inflammatory changes developing. Sometimes the increased depth of colour is so conspicuous that the skin seems finely telangiectatic. Skin so affected may be a little swollen, it is often very sensitive to friction. It is also sensitive to warmth, so that the patient keeps his feet outside the bed-clothes. Either friction or warmth starts burning pain. This pain may often be induced, too, by hanging the leg down; the skin is then seen to congest unusually, the increased tension being responsible for the pain (see erythralgia, page 93). The pain may be very intense.

Pain in the leg, however it arises, brings disuse, and this, with defective nutrition aiding, leads to wasting and to weakness of the limb. Examination of the limb at this stage discovers the signs of diminished circulation and of arterial disease; the limb is cooler than its fellow; usually the pulses of the foot and often of the lower leg are not to be found; capillary pulsation cannot be induced by heating the toes; natural vasodilatation cannot be induced in it, or can be induced only partially, by the methods described in Chapter I; X-ray examination often displays calcareous deposits, which outline the diseased vessels of foot and leg for a shorter or longer part of their course.

Ultimately necrotic changes supervene. The earliest part to be affected is usually the great toe, and the nail bed is the most frequent seat of trouble. An indolent form of ulceration occurs at its margin, and this continues and spreads until the nail is undermined and its bed rendered necrotic. Another frequent seat of early mischief is a corn on the toe or side of the foot, the corn ulcerating and the ulcer continuing and spreading. In both these instances the early lesion is often traumatic. It may arise from a neglected and ingrowing nail, or from careless cutting of or application of caustic remedy to a corn. In other cases necrosis begins in a blister or in a simple abrasion. In all, the simple initial lesion progresses because the natural healing power of the tissues is lost. Their vitality is reduced to a precarious point, and infection is ill-tolerated. An injury or the penetration of infection is liable at any time to result in a more massive necrosis, probably induced by thrombosis in the last remaining and imperfect arterial channels. A toe, already tender, deeply coloured and cyanotic, assumes a fuller cyanotic tint, and this rapidly darkens further and the colour changes from purple or deep violet to dark brown. That, with loss of sensation in the skin, is the first decisive evidence of necrosis. As the tissues blacken, they emit an odour of decomposition, and the necrotic area becomes sharply demarcated from the remainder of the toe. As a rule the necrotic tissue dies and shrinks, gradually shrivelling and hardening, while between it and the rest of the toe a thin line of granulations divide dead from living tissue. Separation of the necrotic mass with such bone as it encloses occurs after a period of months, and throughout this period pain is often severe and persistent. In other cases the original area of necrosis, starting under the nail or in a larger part of the toe, increases, or is from the beginning larger, swelling is more in evidence, a part of the foot supporting the toes and more toes are

involved, the tissues are moist and ooze, the necrotic and sounder tissues are less clearly to be separated. Infection plays a larger part, and in the most dangerous types sets up ascending lymphangeitis or cellulitis of the leg, which if unchecked ends through septicaemia in death. Putrefactive organisms invading the tissue liquefy it and create the characteristic foul odour. Wet and dry gangrene are discussed on page 85.

### Thromboangeitis Obliterans

Curiously, this disease has escaped being classed as arteriosclerosis, perhaps because it occurs in the young. It has its onset usually in the thirties, and shows a strong tendency to attack Russian and Polish Jews. It is confined almost, if not exclusively, to men. The lesions in the arteries are characteristic. The media is uncalcified, the intima shows no atheroma; but, within a relatively intact main elastic lamina, lie great masses of organised tissue, permeated by small vascular channels. This new tissue plugs the artery, or fills a large part of its lumen, so that what blood circulates passes through small crevices. The new tissue is sometimes quite clearly blood-clot in a state of fresh or more advanced organisation; at other times it may be interpreted as proliferated intimal tissue. In many arterial sections it is obvious that the new substance has been lain down in successive layers, at wide intervals of time. The walls of these arteries display evidences of chronic inflammation also in their outer coats, which are often bound firmly to surrounding veins, nerves, and other structures. The extent to which the lumen of the artery is invaded varies much at different levels. The affection of the arteries in advanced cases is remarkable for its extensiveness, involving the main artery and all its chief branches throughout the leg. It is the rule for the arteries of both legs to be involved, though to different extents; the arteries of the arms are frequently affected too, but generally in less degree; the visceral arteries participate rarely. There is no other disease which brings such universal ruin to the large and small arteries of a limb; and there is none in which the extent of vascular obliteration is so disproportionate to the symptoms, or in which obliteration of all the chief arteries of a leg may persist for many years without gangrene of toes threatening. This is due to the gradualness of the process and to the development of an extraordinary meshwork of impalpably small arterial anastomoses in which the blood now flows throughout the length of the limb.

Another feature of this disease is the frequence with which the veins of the legs show isolated or migrating thrombi, with subsequent obliteration of their channels.

The disease is generally believed to be one that primarily attacks and damages the vessel wall, leading through inflammation to secondary thrombosis; but it is uncertain whether this damage is the result of an infection or of a toxic agent. Nearly but not all the patients are heavy cigarette smokers.

### SYMPTOMS

The symptoms of onset and the individual manifestations and sequelae of the ischaemia are similar to those of senile or diabetic arterial disease. It would be superfluous to describe them in detail; to notice special features is enough. Occurring in young men the disease progresses slowly; and so the symptoms develop slowly. Intermittent claudication, which is particularly frequent, owing to the length of main artery involved, may be the only crippling manifestation and may remain unchanged over periods of many years; it may even become less crippling with time. A peculiar, though uncommon, feature is that, owing to the richness of superficial anastomoses, the limb in which the main arteries are the more extensively affected may actually become the warmer. Owing to the duration and to the extent of spread, it is not uncommon to find both popliteal or even femoral pulses lost in this disease, though the patient will usually complain of one leg only. It is also common to discover defects in the pulses of the arms when these are searched, and in the fingers Raynaud's phenomenon may occasionally occur because of disease of arteries in the hand. Owing to the long duration too, gangrene and amputation of one limb is followed at intervals of years by gangrene and amputation of others. Tragically, all four limbs are sometimes lost while the man's body remains sound. Death from gangrene is rare, early deaths are from intercurrent maladies or from accidents in visceral arteries, such as coronary or cerebral thrombosis.

In cases in which the veins provide symptoms, a history of painful red lumps in the legs will be obtained; the locally thrombosed veins will draw attention from time to time and the very slow extension of this clotting will be observed. In some cases, when the legs are warmed and hung down, it will be found that the natural veins on the dorsum of one or both feet have already been lost; often they are replaced by a meshwork of small cutaneous venules.

## Management

### General

Methods of treating the several forms of chronic obliterating disease of the arteries of the limbs have so much in common that they are best dealt with together. It is the foot that nearly always needs treatment, and if this is fully described it will form a sufficient guide to corresponding treatment of the hand. The management of high blood pressure and of cardiac defects in cases of arterial disease, the management of carbohydrate metabolism in cases of diabetes, demand appropriate measures which, however, are not within the scope of this book. So far as general treatment is concerned it will be enough to impress the necessity of simple living, and especially of light diet; this is called for because exercise in these cases is usually limited and the diet should be correspondingly low, and because immoderate feeding is still widely believed to predispose to arterial disease; the diet in diabetes obviously requires special control. It is sound to insist on moderation in the consumption of alcohol and sometimes, and especially in the young or in diabetics, to recommend complete abstention. The use of tobacco should be forbidden in all cases that have developed serious signs of chronic arterial disease.

Early recognition of the disease is very important to treatment; for by regulating the general habits and by nursing the limbs from the earliest stages of their affection onwards, much can be avoided that otherwise soon becomes untreatable. Early recognition comes in some cases by paying more than usual heed to complaints of the legs; it will come oftener by searching the pulses of the legs in the old, and always in elderly hypertensives and diabetics.

### Precautionary measures

When the circulation to a leg or foot is known to be defective, although the skin is still in good condition, treatment should begin. Cleanliness should be scrupulous, the feet being very carefully and thoroughly dried after washing; moistness of the feet is always to be avoided, socks being changed often if necessary and a simple powder used. The socks should be soft and thick and the shoes close but well-fitting. These precautions are intended to avoid any chance of the skin rubbing or blistering under friction. The nails should be cut straight, and the great toe-nails, if thick, should be

thinned down by filing their surfaces, to render them more supple, and thus to prevent them from exerting lateral pressure, which so often results in early ulceration. Nail cutting should be managed so carefully as to avoid any possibility of snicking or abrading the skin. Similarly, corns must be dealt with very circumspectly; surface paring after bathing is permissible, deeper cutting is unwise because it may wound. Substances intended to kill the surface layers should not be applied. The feet must be protected well from cold. The patient should cultivate the habit of treating his feet almost as though they were of porcelain, being on guard against stumbling or striking the toes upon hard and resistant objects, and alert to prevent others damaging the feet by treading upon them or otherwise. Shoes should be examined periodically to recognise the earliest signs of inwardly projecting nail. Rubber soles or rubber boots can often be worn with advantage. Regular but gentle forms of massage of feet and legs is beneficial.

### SYMPTOMATIC TREATMENT

Utmost care of the feet along the lines laid down becomes imperative if symptoms appear.

*Abrasions.*—Abrasions, however slight, should be dried up, and this desiccation is done most quickly by resting the raised feet under a filter of dry gauze in a current of warm air. The broken skin should not be placed in water nor fomented. The abrasion may be protected by a dry and sterile dressing, so arranged that it will not adhere to the injured skin.

*Discoloration or Limited Gangrene.*—If the skin develops any discoloration or tenderness, the foot should be kept at body level, at rest, and under close observation. Increased circulation to the foot should be encouraged by methods to be described, when there is the early threat, or when there is an actual breach of surface. When there is limited gangrene, treatment should be at first conservative; the toe should be desiccated and allowed to separate itself. It is a mistake to think that this wastes time and that there is always a quick recurrence of gangrene; the original necrosis is often determined quite locally by thrombosis. Radiant heat may be used to desiccate a necrosed toe, but it is less safe than a current of warm air driven by fan, and should not be used unless it is manifest that the supporting tissues are well supplied with blood.

*Infection* in its early stages, and of small surfaces, may be treated by applying non-irritant antiseptics, like Dakin's solution, and by

subsequent desiccation. Deep or spreading infection is a chief indication for amputation.

*Pain* of the erythralgic type is treated by resting the part, keeping it raised and cool. When this pain is persistently severe, as in early gangrene, morphia is indicated; when long maintained and severe it causes insomnia, and in this and other ways undermines general strength and the resistance of the central nervous system. It then demands amputation. The pain of muscular ischaemia, if severe, immediately brings its own remedy, rest. But patients should make no demand on their muscles that induces pain. Where it is a main complaint, massage of the involved muscles will benefit.

### VASODILATATION AS TREATMENT

The objects of the methods to be described are to increase blood-flow to the limb as a whole, in the hope of encouraging anastomoses, or locally to bring even a little more blood to an extremity threatened by incipient gangrene. Many methods have been used.

*Warming.*—It has become a regular practice to treat threatened feet by exposing them directly to warmth or actual heat. There are grave objections to this plan. Warming or heating a normal foot increases the circulation to it. When, however, the arteries to the foot are diseased little or no increase of flow may be brought about, but it is quite certain that the warming or heating will increase the metabolism of the tissues and thus increase the blood-flow requirement. Thus it is apparent, on theoretical grounds, that a local application of warmth may do harm and not good, and experience bears this out. Hot bottles applied to the foot imperil the tissues; a burn is dangerous, and these feet burn more easily than normal; but even the use of warm bottles is not often wise; the circulation to the feet should be increased by warming the subject's trunk and thighs; the warming of the feet should happen indirectly through increased blood-flow; at least enough direct heating will happen by the passage of warm air from body to feet beneath the bed-clothes. To apply moist heat is inadvisable, and radiant heat, as already stated, may be used only in special circumstances.

*Vasodilator Substances.*—A number of such substances have been used. The nitrites, owing to their fleeting action, are recognised to be useless. So is acetylcholine for the same reason, and because given intramuscularly it is broken down and loses all virtue before it enters the blood stream. Little or no benefit is known to follow the use of many other substances, such as various extracts of

muscle or of pancreas, parading under various trade names. There are no known substances that can be safely used to produce an adequate and sufficiently persistent dilatation. There is no known remedy of this kind so persistently potent as warming of the body, and this is simple, economical, and safe.

*Periarterial Stripping*, sometimes and wrongly called "sympathectomy", is an operation in which the periarterial tissue and adventitia are cleanly stripped for several inches off the main artery of the limb. After a widespread vogue it is gradually being discarded; the fact that it has been found in many cases to produce little, certainly no lasting, vasodilatation, has proved too strong even for theory. There are those, however, who are convinced that it sometimes conspicuously relieves pain.

*Suction.*—The limb is inserted into a suitable box, through a neck which is sealed about the limb by a rubber cuff. The pressure in the box is lowered by 50 to 100 mm. Hg, and blood is thus drawn into the limb. Simple suction of this kind is useless, for it also obstructs the venous return and blood-flow is only very temporarily increased. But if after a half-minute the suction is released or replaced by pressure, blood will leave the limb by the veins. An alternating action draws blood in and expels it, and has been proved to increase flow. The treatment is used for ½ to 1 hour a day, the grade of suction being settled by the patient's reaction to it; it should be painless. It requires special apparatus, is still on trial, but has given some promising results.

*Sympathectomy.*—Lumbar sympathectomy, for the details of which the writings of Gask and Ross may be consulted, consists in removal of the last 2 or 3 lumbar sympathetic ganglia (the 2nd to 4th ganglia where these can be identified) with the intervening stretches of sympathetic cord. The ganglia are removed to ensure that there is no regeneration, and that the result is permanent. The operation produces persistent vasodilatation of the corresponding foot and increased warmth of foot and leg. Sweating is lost over the foot. The operation may be performed on one or both sides; it is a safe operation in good hands, and is rarely responsible for adverse effects; it is said that the bilateral operation is occasionally followed in the male by sterility. Sympathectomy is the most certain means of producing long lasting vasodilatation that we possess. It has been used in both thromboangeitis and arterial disease of the elderly, but especially in the former. Improvement is the rule, and many striking successes have been observed. In cases in which inter-

mittent claudication predominates, the symptom may diminish but rarely disappears after operation. When pain at rest is prominent it is generally relieved or abolished. In cases showing early gangrene it is not unusual for this to cease extending, and for slow separation and healing to occur. But the results of operation are variable and in a number of the cases improvement is not seen; this is not surprising where disease is extensive and gross, for in these much vasodilatation is hardly to be anticipated. A preliminary test, that of spinal anaesthesia, is sometimes used, to ascertain how much vasodilatation may be expected. Cases that respond well are the most favourable for sympathectomy; but the result of operation is still unpredictable in many and especially in those in which the reaction to spinal anaesthesia is small. It is not yet known to what extent sympathectomy when undertaken early slows the progress of disease.

### AMPUTATION

Amputation is undertaken in cases of arterial disease when gangrene has appeared, and the chief indications of its desirability are several. It is necessary when the gangrene is extensive, usually when it involves several toes or when one or more toes and the adjacent parts of the foot are attacked; it is imperative when massive gangrene of the foot has appeared. Intolerable and intractable pain is another clear indication. When a toe has become gangrenous and, after seeming to settle down to a given line, spread is definitely ascertained, amputation is usually desirable. Quick extension often means infection, which, if it involves deep tissues causing swelling, or marks out lymphatics, requires immediate removal of the limb to avert danger to life.

The extent of gangrene is rarely a full guide to the proper level for amputation; in gangrene in the elderly or diabetic when conservative treatment has failed, it is sound to follow custom and remove the limb through the lower third of the thigh, for in these diseases it is expedient to amputate at a level where the main artery of the limb is patent and relatively sound, and to operate so high as to make it reasonably certain that the amputation will be final. But in thromboangeitis obliterans, the level to which the main vessel is open is not a good guide; excellent results are often obtained when the artery is obliterated at and well above amputation level. In this disease surgical treatment could be more conservative with advantage. The ideal test would be one showing the level at which the

blood supply of the tissues is reasonably normal, and the nearest approach to this is reactive hyperaemia (see page 14). It has been suggested as a basis that if a bright reaction appears in the warmed heel within 15 seconds of circulatory release, amputation may be just above the ankle, and that if it takes this time or but little longer to reach the bases of affected toes, these alone should be amputated.

# CHAPTER VI

## VASOCONSTRICTION. LOCAL ARTERIAL SPASM

### Vasomotor Nerves

We recognise that the vessels of the body generally are governed by vasomotor nerves. These nerves belong to the sympathetic system. They issue from the spinal cord and join the sympathetic chains; in the ganglia of the chains the postganglionic fibres have their cell stations, and the fibres themselves pass on through the grey rami to the somatic nerves. The vasoconstrictor nerves to the limbs pass either through the cervico-brachial or lumbo-sacral plexuses and are distributed to the limb through the mixed nerves which issue from the plexuses. For practical purposes the vasoconstrictor nerves may be regarded as distributed to the vessels exclusively in this way, and not, as has sometimes been believed, along the walls of arteries. To the vessels of the skin the distribution is such that a given nerve or nerve twig contains the sensory, sudorific, pilomotor, and vasoconstrictor nerves of a common territory. Thus, if the ulnar nerve is cut at the elbow, sensation is lost and sweating ceases over a well-recognised area on the inner aspect of the hand, and in this same area of skin the vessels become relaxed. Vasoconstrictor nerves exert a tonic action on the vessels, which may increase or decrease in various circumstances, and which is abolished by section of the nerves. They are known to act on main arteries and their branches, on arterioles, on minute vessels including capillaries, on veins, and on the recently investigated arteriolo-venous anastomoses, which have been referred to previously as playing so important a part in maintaining the temperature of the extremities.

In addition to vasoconstrictor nerves there are probably vasodilator nerves, also of sympathetic origin, though much less is known of these and they have not as yet acquired any practical importance. Very probably vasoconstrictor and vasodilator nerves

come in and out of action reciprocally; but it is simpler and adequate for most purposes to concentrate attention upon the more generally recognised vasoconstrictor nerves.

We are unable to say if any central control of circulation occurs by way of the posterior nerve roots. As is well known, posterior nerve-root axons divide and supply a branch both to the skin and to the cutaneous blood vessels; but it is still unknown if centrifugal impulses normally travel by this pathway.

## Vasoconstriction in Health

Vasoconstriction of central nervous origin is produced conspicuously by the emotions anger and fear; it is produced reflexly, but very temporarily, by painful or very cold stimuli applied to the surface of the body and indeed by any strong sensory stimulus. Vasoconstriction is also produced when the blood returning to the regulating centres is cooled. A slight change of temperature of a large surface of the body or a conspicuous change of temperature of a small surface is enough to call forth this direct nervous reaction after an adequate latent period. These vasoconstrictions, like those studied in animal physiology, affect the cutaneous vessels of the body generally, including those of the limb. Of nervous vasoconstriction limited to parts of the body such as a limb, or showing local discrimination in the degree of its effect, we have little knowledge.

Vasomotor nervous regulation is supplemented by the action of substances circulating in the blood, the most certain instance being that of internal secretion of the suprarenal gland, which is released in circumstances of general vasoconstriction. The nerves and the substance act together and exercise a general governance over vascular tone.

## Vasoconstriction in Disease

Many diseases have been thought to be characterised by changed, and some by increased, vasomotor nervous tone. High blood pressure in essential hypertension possibly results partly from increased vasoconstrictor nervous tone, though modern observations tend to indicate that the main increase of arterial resistance is not nervous in origin. Paroxysmal hypertension, as it is called, a state in which attacks of high pressure, universal blanching of the skin, and rapid pulse action are characteristic, is generally ascribed to an out-

pouring from the suprarenal gland, the symptoms of the attack being similar to those following the injection of adrenaline. The rare attacks of distress with blanching and hard pulse, described long ago by Nothnagel, were possibly though not certainly similar. The manner in which cutaneous coldness is brought about in the rigor of fever, and often in mild infections, is not fully known, though here again the vasomotor nerves are probably largely involved. Although we are aware that some subjects present almost habitually cold and others almost habitually warm hands, a subdivision of patients into categories of relatively high and relatively low vasomotor nervous tone is not yet justified. We do not know if in the cold-handed person the difference lies in nervous system or in the blood vessels; no test has yet been devised accurately to gauge vasomotor nervous tone. It is to be observed that all examples so far cited are general states and do not apply to a given limb or to a particular part of a limb. As in health, so in disease, increased vasomotor nervous tone confined to one limb or to a part of it is unknown, a fact which will become more apparent in proceeding.

Of substances foreign to the body and producing vasoconstriction when taken by the mouth, the most familiar and important is ergot.

### ERGOT POISONING

Our knowledge of the serious vascular manifestations of ergot poisoning in man is derived mainly from early records, in which the symptomatology is imperfectly described. The affected are usually young or middle-aged adults of either sex. In subjects in whom vascular, and not nervous, symptoms predominate, symptoms appear in the limbs after infected rye bread has been consumed for periods of many days or weeks. The first symptoms are described as consisting of tingling pain, or painful cramps. They are succeeded or accompanied by sensations of coldness, actual coldness, pallor, marbling of the skin, and numbness. Thus the symptoms are those of obstructive arterial disease in general. The pulse is small and hard; the actual heights of the raised blood pressure are unknown. The skin of the affected limb now begins to show more definite nutritional changes in the form of bruises or blisters, or infection beneath the nails. The darkening of the skin, which marks the onset of gangrene, may be delayed for weeks. The lower limbs are more often affected than the hands. Gangrene may be confined to fingers or toes, or may involve the whole foot

or the leg to the knee; it is usually symmetrical. The early stages of gangrene are associated with severe burning pain in the affected limbs, with desiccation of the tissues, which at first waste and later shrivel. The gangrene even when extensive is dry, separation is slow, the mummified limb may be worn by the patient for a long time and its detachment may be accidental, or may call simply for division of the bone.

Single experimental doses (0·2 to 0·5 mg. of ergotamine or of 0·2 mg. of ergotoxine) given to man intravenously cause giddiness, pressure in the head, and tiredness; the skin of the face, hands, and feet becomes cyanotic for as long as 48 hours, and systolic blood pressure is raised by 20 or 60 mm. Hg. Experiments upon fowls have shown it to be easy by feeding with infected grain or by daily intramuscular injections of ergotoxine (0·1 mg.) to produce and maintain almost complete stagnation in the comb, the result of continuous arterial spasm. It is improbable that this stagnation from spasm ever finally arrests the circulation or causes necrosis directly; it is more probable that the latter comes indirectly. The stagnation damages the vessels, and leads up to stasis—complete blocking of the vessels by impacted corpuscles—or to thrombosis in the vessels; the terminal parts of the comb, which are the first to lose circulation completely and permanently, become gangrenous in consequence. Presumably the sequence of events in man is similar.

Ergotism, owing to its present day rarity, has little practical importance. But its vascular manifestations are of profound interest in exemplifying spasmodic obstruction of vessels, which continues if poisoning continues and then leads, through local thrombosis, to necrosis in the distal tissues of the limb.

## Local Vasoconstriction

### Local arterial spasm associated with impact

It has been established, since Kroh described such cases, that arteries of the size of brachial or femoral can exhibit a state of sharply localised spasm following impact. The record has usually been of a wound by bullet, fragment of shell or of grenade, passing near to but not touching the artery affected, and giving rise to local bruising or extravasation of blood; the blow from the propeller blade of an aeroplane has sufficed. Surgeons, exploring the wounds, have repeatedly found the artery locally pulseless, or have found it actually contracted to the state of a thin cord. The transition from

normal to contracted artery may be abrupt; pulsations are found above but not below the affected part. Vessels in this state have been seen to resume pulsation while simply exposed, or after irrigation with hot saline; they have been opened and no clot found obstructing them. The degree of spasm is variable. It may close the artery completely, for in some instances the extremity of the limb may be reduced to a condition of coldness, lividity, insensibility and immobility, indicative of arrested blood-flow and threatening massive gangrene. But no instance of gangrene seems proved to have arisen in this way; the spasm relaxes, the full pulse returns distally and recovery seems to be invariable. Cases in which at the time the circulation to the limb is completely lost are comparatively rare; and in these it is not obvious how uncomplicated closure of the main artery can produce the result. Perhaps such cases are not uncomplicated, or perhaps they are exceptional in the paucity of anastomoses or are unusual in the relation of the stretch of vessel in spasm to the mouths of collaterals. More often the distal pulse is lost or greatly diminished, but the colour of the limb is little altered and the vitality of the limb unthreatened. The local spasm described may last surprisingly long, usually for several or many hours, not infrequently for a day. The mechanism of the spasm is not clear. It is usually ascribed to stimulation of the nerves of the periarterial tissues, but this is merely hypothesis, and difficult to accept in that large arteries cannot be brought to occlusion and maintained there by sympathetic stimulation. The correctness of the assumption is no more probable than that the spasm comes, as might be suggested, through the action of extravasated substances upon the affected artery; as previously mentioned, extravasation is usual in or around the wall of the vessel. A similar local spasm occurs and may last for hours when the adventitia of a main artery has been stripped off in the operation called periarterial stripping.

It is noteworthy that in the very many records of cases presenting arterial spasm from injury pain is scarcely ever mentioned.

### INCREASED ARTERIAL TONE ASSOCIATED LOCALLY WITH DISUSE

Disuse is a potent cause of local diminution of blood supply and of discoloured skin. Such changes will be described in Chapter X, where they can be correlated more conveniently with vascular changes occurring in cases of disease of the nervous system.

### INCREASED ARTERIAL TONE DUE TO LOCAL COLD

Exposure of a single limb to cold, as when it is immersed in cold water at 15° or 20° C. (59° or 68° F.) causes after a time a distinct decrease in the size of the main arterial pulses, abolishes the palpable pulses in arteries of the size of the digital vessels, and notably decreases the recorded volume of general pulsation in the limb that has been cooled. The skin may pale a little. The veins of the dependent limb are notably reduced in size and distinctness. These changes are due mainly to the direct action of cold upon the vessels concerned. But in subjects presenting warm hands in most ordinary circumstances, exposure to local cold never reduces the circulation to the fingers to the extent that blood-flow to them is arrested; neither does it do so if simultaneously vascular tone is generally raised by exposing the whole body to cold air, thus bringing more strongly into play the central vasoconstrictor nervous mechanism. In other words, the circulation cannot be brought to a state of arrest locally in normal people, though it is clear from the tints assumed by the skin that it can be brought close to arrest, by exposing the body and limb to cold. Actual closure in these circumstances is abnormal and will be commented upon at greater length in the next chapter.

## Differentiation of Spasmodic from Structural Disease

Obstruction due to spasm of an artery supplying an extremity is in the rule transient, obstruction due to structural change is in the rule permanent; but there are exceptions both ways. Thus spasm may be maintained for days under the influence of ergot, though it is probable that structural change in the form of thrombosis always replaces it if obstruction is maintained much longer. On the other hand an embolus, having at first totally occluded the main vessel, may be forced partly or wholly into a branch and thus free the main artery of the limb; or a short clot may at times be opened up by canalisation and by expansion of the newly formed channels. These exceptions, owing to their rarity, are without great practical importance. They have theoretical importance and serve to illustrate difficulties that will occasionally arise in attempts to differentiate, even though close search is made and full testing is carried out in the manner described earlier (see page 13). From this standpoint the particular example of ergotism may be emphasised, for experiment

teaches that spasm so brought about relaxes neither to centrally induced vasodilatation nor to local heating.

The present also affords an opportunity of stating that, owing both to exceptional circumstances of the kind described, and to insufficient knowledge of differential tests in the past, instances of structural disease have been reported as spasmodic when there was incomplete justification for so doing. Critical research has in fact failed to reveal instances of vascular spasm or local contraction, other than those of the nature cited in the present and following chapters.

Much has been written of pathological states in which both structural disease and increased vasomotor nervous tone are considered each to contribute to the end result. I have been unable to find any satisfactory evidence that such states occur, though it will be evident that vasomotor tone of normal measure may coexist with structural disease such as would itself narrow the vascular lumen.

# CHAPTER VII

## SPASMODIC ARTERIAL OBSTRUCTION; RAYNAUD'S PHENOMENON

Since Raynaud wrote his papers, the term Raynaud's disease has been applied to a number of distinct conditions, which are included in this chapter and in that which follows it. Raynaud wrote of conditions of spasmodic discoloration of the digits and of symmetrical gangrene, believing these all to constitute one specific malady, and supposing the fault to exist in the vasomotor centres, overaction of which resulted in vasoconstriction. Investigations in my laboratory have proved this traditional view to be based on inadequate evidence and have focussed attention, not upon the nervous system, but upon the blood vessels themselves. We now know that transient discoloration of the fingers and symmetrical gangrene are manifestations occurring separately or together in a variety of maladies, which it is quite necessary to separate strictly from each other; it is no longer permissible to use the term Raynaud's disease in its original loose sense. But if for the moment we still consider these different maladies as a whole, it becomes increasingly clear that serious local faults are present, and less and less clear that there is any condition in which local discoloration or gangrene of digits is due purely to overaction of vasomotor centres. In some groups of cases disease of the digital arteries is being found, with such narrowing of vascular lumen that natural increases of tone result in closure. Undoubted and various cases of this type, including examples of thromboangeitis obliterans, are described on page 74; others, in which structural narrowing is strongly suspected to be the basis of vascular disturbance are described under diffuse scleroderma on page 72. These cases, almost by common consent, are finally lost to the old vasomotor category. The remainder, for reasons that will be discussed, seem rapidly to be following suit.

It will be convenient to apply the term "Raynaud's *phenomenon*"

to any state in which loss of circulation to digits occurs spasmodically, displaying itself in transient attacks of discoloration. But, if the term "Raynaud's *disease*" is still to be employed, the confusion of the past must be avoided and the term restricted to what may reasonably be regarded as one disease. Here it will be used to comprise cases of intermittent spasm of digital arteries, with or without local nutritional changes; these cases are grouped for description under the two headings that follow.

## 1. Raynaud's Disease or Intermittent Spasm of Digital Arteries

The simplest form of Raynaud's disease starts usually in childhood or in adolescence; occasionally it begins later. In its very mildest form it is a very common malady among young people, occurring in about 20 per cent of nurses and students and equally in the two sexes. This mildest form, in which single attacks occur at rare intervals, comes little under medical observation. The more pronounced forms are less frequent, but nevertheless common, and they affect women more than men. The malady has a very clear tendency to be familial, but definite relations to habit and to infectious disease are unknown. The subjects are usually spare or thin; it is usual for them to have suffered previously from cold extremities, and an early or contemporary history of chilblains is the rule rather than the exception. It has been said that the subjects are unusually nervous or excitable; such has not been my experience. The great majority present no demonstrable abnormality of the central nervous system. The affection displays itself in attacks indicating arrest of circulation to digits. These attacks affect the hands; the feet are often exempt from them, or they are affected to a less extent. At an early stage or in mild cases only one or two fingers may be involved, but it is the rule for the 2nd to 5th digits to become discoloured and for the affection to be symmetrical. The thumb may also be included, as may the whole hand with or without the lower part of the forearm. Similar phenomena are found in the feet. Very rarely the nose, ears, or cheeks may become discoloured.

*Provocation.*—The attacks are brought on by general exposure to cold, as by bathing in cold water, or by exposure out of doors; they are more apt to happen on cold than on warm days and so are either confined to winter months or are much more frequent during these. The precise conditions provoking attacks vary with the severity of

the malady and may be exemplified by the hand. In the mildest form the circulation is lost to a finger, or to the ends of a few fingers, after bathing long in cold water on cold days; in others even short immersion of the body in cold water will always induce attacks; in others attacks are frequently or regularly experienced when out of doors in cold weather; in the severest cases attacks occur both in winter and summer, and indoors as well as out of doors, and involve all the fingers to their roots and perhaps the hand as well. In all but the mildest cases it is usual for exposure of a kind that has once proved adequate always to provoke attacks, so that the number of attacks in the month, week, or day, depends upon the number of adequate exposures. Another frequent provocative cause is immersion of the hands in cold water; but water of optimal temperature for this purpose is about 15° C. (59° F.), and not water of icy coldness, which even in these cases tends to produce a red reaction (see page 102). When the hands are cold, excitement, fright, or anger, or the receipt of a painful stimulus, may determine an attack.

*Manifestations of the Attack.*—In a given finger the attack usually starts at its tip, the arteries closing there first, and spreads for a variable distance towards the base; the finger may become affected simultaneously in its length.

The manifestations in the finger develop much as they do in loss of circulation to the whole limb (see page 17). When the circulation is arrested the finger pales a little, and gradually becomes greyer as the blood in the minute vessels gives up its oxygen. This pale greyness of the fingers, which first signals the onset of the attack, usually escapes the patient's notice. In the severer cases, after the blood-flow has been arrested for many minutes, the skin pales conspicuously and often becomes completely blanched and waxy in appearance. An almost similar appearance may be brought about by massaging the blood from a finger early in the attack, but the spontaneous waxiness is due to active contraction of the minute blood vessels; this contraction is a secondary effect of arrested blood-flow, and is not peculiar or essential to the attacks described (see page 18). If the fingers are high coloured between the attacks then they are more obviously cyanotic in the early stages of the attack; the waxy finger will also give place to a conspicuously cyanosed finger if the minute vessels at any time relax, for blood then flows into and fills them from the veins.

Both the waxy fingers, and those of full cyanotic tint, may be taken to indicate actual arrest of flow, the former does not bleed

when cut, the latter will ooze but a little. When the circulation to a finger has been lost for a half-hour or more, the finger becomes numb, the numbness beginning at the tip and gradually spreading upwards. It is the numbness which usually first attracts the patient's attention. In long-drawn attacks out of doors the fingers cool to air temperature and on very cold days become painful; this pain is characteristically aching, and may be very disagreeable.

When recovery occurs from the attack, the finger begins to be invaded by redness from its base and, as it is invaded, the transition from redness to blueness is marked abruptly. The redness represents reactive hyperaemia (see page 21), the colour being bright red and its depth unusual, and is accompanied by tingling when the previous arrest of circulation has been of long duration. The finger rapidly becomes warm and recovery is soon complete. The volume of its pulsation is then normal and capillary pulsation is visible at the finger-tip.

*Course.*—The course of this malady is very variable. Subjects displaying it in its mildest form very frequently become free from it as they advance towards middle life; in other mild cases, and in most of those in which the attacks are more easily provoked, the condition remains unchanged. Some of the cases progress unfavourably; the change is usually very gradual and spread over years; it may happen more rapidly. Those progressing unfavourably pass into the categories now to be described.

## 2. Raynaud's Disease or Intermittent Spasm of Digital Arteries with Local Nutritional Changes

The original symptoms in these patients, and they are nearly always women, are precisely the same as those of the last group, namely, attacks with discoloration and numbness of the digits on exposure to cold with recovery on rewarming. The cases differ from those of the last group in that the condition is progressive or occurs in more serious form from the beginning. The attacks are very easily provoked, occurring in summer as well as winter; and it is not a matter of occasional and brief attacks, but of very frequent and prolonged ones. In much of the intervening periods the state of recovery may be only partial. The state of partial recovery is peculiar. The hands at the end of a long attack are fully cyanotic; this cyanotic skin is invaded by patches of bright redness, single fingers become red; but the temperature does not rise in these red

areas, which gradually turn blue again. The shift from blue to red and red to blue may be repeated many times and over long periods without real recovery. The changes are due to transient, slight, and local relaxation of the obstruction, so that a little arterial blood creeps in here and there, and then the spasm reasserts itself. This state is termed "intermittent leakage". In these cases hours may elapse each day during which the blood-flow to the fingers is absent or occurs just perceptibly and transiently at intervals. Sometimes the fingers present a little chronic swelling. Clear evidences of atrophy are common, the fingers being narrow, and tapering to their ends; the pulp of the fingers is reduced, so that it no longer projects normally beyond the distal attachment of the nail; the nail itself is often overcurved in its length and from side to side, grows slowly, and is ridged. The bones are rarefied. In such fingers it is frequent for the skin to be less than normally mobile, it is relatively unwrinkled and smooth, feels hard and cannot be pinched up with normal freedom. Whether swollen, or showing these signs of atrophy with sclerodactyly, flexion of the fingers is limited. In those cases presenting the more obvious changes it is the rule for areas of necrosis to appear from time to time at the tips of the fingers; these occur in the form of minute scales or plugs of dead skin, which separate very slowly and painfully, leaving tiny depressed scars; or little necrotic areas with or without previous blistering come at the borders of the nail and discharge a little serum or pus and heal slowly. Any injury of such a finger heals slowly; chilblain may appear but is disguised, being dusky in colour and without swelling and heat. Gangrene of a considerable portion of a finger is very rare, but occurs; loss of substance happens in the rule bit by bit and very gradually. The terminal phalanges may become shortened. The pain in fingers presenting small necrotic foci is often excruciating, and at its worst seems to be associated particularly with sudden warming up of the fingers.

Examination of such fingers in the warm state generally reveals their arteries to be incapable of complete expansion, as indicated by a reduction in the volume of their pulsation and by inability to elicit capillary pulsation.

It is to be understood that the picture here painted is of an advanced state; between this type and those previously described there is a perfect series of intermediate forms. It is known that cases of the milder type develop into cases of severe type, but this does not necessarily happen.

*Course.*—These cases are essentially chronic. The patients suffer much inconvenience from the clumsiness of their fingers, which on that account are more liable to damage. The little necrotic foci appear and slowly heal, others follow; scarring increases, but its progress is so gradual that many years pass before an appreciable amount of the finger tip is lost; much pain is suffered. Very occasionally a larger piece of finger may be lost by gangrene. A number of these patients, and especially those in whom the condition comes rapidly, develop diffuse scleroderma (see page 72).

### *MECHANISM IN THESE CASES OF RAYNAUD'S DISEASE*

Consideration will be confined chiefly to cases in which discoloration of fingers is easily provoked, for these only have been investigated adequately; what is said may be taken to apply to such cases whether they present obvious nutritional changes or not.

The central fact is transient loss of circulation to the digits occurring on exposure of the body to cold. I have shown that this spasmodic loss of circulation is due to closure of the digital arteries, and that, irrespective of its nature, the fault lies in these vessels. The closure does not involve arteries of much larger size, neither does it include small arterioles or veins. But since the attack is induced by exposure to cold, to which all vessels normally respond, a general reduction of their size happens; but in most of them the degree of closure can be regarded as no more than natural. In the small arteries only is the response to cold manifestly abnormal; these are in a state rendering them particularly liable to shut on direct exposure to cold. In sensitive cases, the blood-flow to a single finger can be arrested at will by cooling this finger alone, or even by cooling a short stretch of it; for the digital arteries possess this liability to closure in their whole length. The state of closure once established can be released by warming the hands; and this can also be effected in the arteries of separate fingers, or even in the arteries at the base of a finger, by warming the finger or its base separately.

It has been indicated already that, when a normal subject is exposed to cold, arteries like the digital narrow under two influences; they constrict as a direct reaction to cold, and because vasomotor nervous tone increases. These same two factors operate in the fingers of the cases we are discussing, under conditions of general cooling. But, because in these cases there is an abnormality, the vessels do what they will not do in normal

subjects, they close to obliteration. The evidence we possess goes to show that the abnormal element is local, and not, as formerly thought, in the response of the nervous system. Thus, if vasomotor tone is deliberately reduced by warming the subject's body, immersion of the hand of a susceptible subject in cold water will still induce the attack; but if the hand is kept warm, an increase of vasomotor nervous tone, induced by cooling the body, will not provoke the attack. Again, if the circulation to the fingers of such a patient has become arrested by general exposure to cold, local destruction of vasomotor tone by nerve anaesthetisation does not bring instant release of blood-flow, which would happen inevitably if vasomotor tone were alone responsible; it brings delayed release, or the release fails. Likewise, as experience has shown, destruction of the sympathetic nerve supply to the limb by surgical intervention does not cure the malady, for it frequently happens that patients so treated continue subsequently to display attacks on exposure to cold. The local abnormality is the reason for this, for it remains untouched. If Raynaud's disease were a vasomotor malady, we should not expect it to be most prominent in the hands, but in the feet, in which vasomotor tone is ordinarily greater. And we should expect the spasm to affect the arterioles and capillaries as well as the small arteries. Although careful search has been made, no evidence has been found, from blood-pressure readings or in other ways, of an abnormal vasoconstrictor response. It is unnecessary to suppose it; the malady is adequately explained as a local one.

But although these facts show that the fault is not in the nervous system, that is not to say that the nervous system plays no part in the attacks. If under the direct influence of cold the arterial channels of a hand become unusually narrowed, but not quite obliterated, then subsequent cooling of the trunk, or an emotional disturbance, or a painful stimulus, by normally increasing vasomotor tone will cause the vessels to close completely and thus determine an attack. It is this kind of event that has been misinterpreted in the past, and has given support to the wrong idea that the vasomotor nervous system is primarily at fault. Further it will be apparent that anything reducing or abolishing vasomotor tone will on occasion bring an attack to an end, and continuing as an influence will tend to prevent the recurrence of attacks. This is the basis upon which the modern treatment by sympathectomy rests; its successful results are due, not to interference with the passage of abnormal nervous impulses, but to the destruction of normal vasomotor tone.

### NATURE OF THE LOCAL FAULT

It has been ascertained in cases of Raynaud's disease presenting nutritional changes in the fingers, both by examining the fingers during life and by histological section after death, that the digital arteries are incapable of full expansion and are narrowed by thickening of the intimal coat.

In cases in which spasm is easily induced, but in which there are no nutritional changes, the vessels, though in general found during life to be capable of very full expansion, may also present obvious though lesser grades of narrowing from disease, when examined histologically. Thus, in these cases of easily induced spasm, it may reasonably be supposed that intimal thickening contributes to bring a contracting vessel to a state of closure. When there is advanced disease, a normal increase of tone may perhaps be all that is required. But where disease is less advanced it is probable, and where only slight disease is to be found it is certain, that abnormally high tone must develop in the artery to close it. To the extent that abnormally high tone develops, it is to be regarded as due to the direct stimulus of cold. The reasons for the last conclusion have been given previously, but in the present connection the reason to be emphasised is that local cooling will close the vessel robbed of its vasoconstrictor tone, while increased vasomotor tone fails to close the vessel if the latter is kept warm. These facts illustrate powerful response of the arterial wall to the direct stimulus. We do not know as yet the precise extent to which structural change contributes in individual cases; but if we believe that in one patient it plays an important part, and that in another it plays a small part while abnormally high tone contributes chiefly, in all cases the evidence favours the view that the abnormal factors are local.

This is the conclusion for cases in which attacks are easily induced. Because these cases develop out of milder types, it is right provisionally to regard milder and severer types as stages in the progress of one disease. In an occasional case of mild type, in which the arteries have been examined, there has been little histological difference from the arteries of warm-handed people of the same age; provisionally, an increased direct response to the stimulus cold is to be regarded as the primary fault in these cases also.

Possibly the intimal thickening, which may develop in cases that progress, is secondary to diminished blood-flow through the arteries in the early stages; but this is unproved. In the normal subject con-

striction of the vessels, during exposure of the subject to cold, approaches nearly to closure; in the affection considered closure actually happens, under various degrees of provocation and for varying periods of time. Seemingly there is a complete transition. The milder forms of the affection are harmless, the more severe forms are crippling. The passage from one to the other very possibly comes when closure happens sufficiently often and for sufficiently long periods to affect the nutrition of the vessel wall, bringing changes in the intima, which ultimately predispose as in the instance of ergotism to local thromboses. The development of necrosis in the finger-tips is probably to be regarded as arising directly out of the thrombotic plugging of damaged vessels.

## *TREATMENT*

### GENERAL AND SYMPTOMATIC

A first principle in treatment is to maintain the local circulation, and this by relaxing vasomotor tone as well as by treating the affected vessels. Patients who suffer from intermittent spasm of the digital arteries should be given such instructions as will help them to take all reasonable precautions against attacks.

Exposure out of doors in windy, wet, or cold weather should be avoided. Riding, boating, journeying far on foot or in cars, in any but warm weather, bring risks of long exposure to cold, against which there should be ample safeguard. Bathing in cold water is unsuitable in all weathers. The body should be warmly clad, even to the point of discomfort; for womenkind short skirts and thin stockings are unwise; arms should not be kept bare in or out of doors. The temperature of the occupied room should not be lower than 21° C. (about 70° F.); a temperature of 23° or 24° C. (about 73° or 75° F.) is better. Except in hot summer weather the hands, when affected, should be covered with thick woollen or fur gloves whenever the patient is out of doors. The hands should be under the bedclothes at night, and the latter should be sufficient to keep the body warm rather than cool. The hands should be kept so warm that they feel warm against the face. When the feet are affected, thick stockings or gaiters should always be worn out of doors; outdoor shoes should be thickly soled, preferably with rubber, and should be quite water-tight; well-fitting gum boots are very suitable.

When cold, neither hands nor feet must be warmed before a fire, by contact with hot objects, or by immersion in hot water; but only

by soaking them in warm water, by contact with the body, or by friction. In advanced cases the patient should develop the habit of most carefully protecting the extremities from abrasions, bruises, burns, and other injuries. In earlier cases the hands should be used for any forms of work that warm them naturally; in the more advanced cases regular and thorough massage may be substituted.

The diet should be liberal, to develop weight, so that the body is no longer spare but well covered. Those who can do so should winter, or reside permanently, in warm climates.

The general directions here given are for the severer forms of malady; but in the milder types the patient should be advised to take as many of the same precautions as can be regarded as reasonable and appropriate.

*Pain and Necrosis.*—Pain calling for treatment rarely occurs unless a finger is inflamed. It is due to the finger becoming very cold in prolonged attacks of arrested circulation; or, on the contrary, to quick warming in the stage of recovery. Thus pain can usually be avoided if attacks can be avoided. When the pain during recovery from attacks is severe, recovery should be induced by alternate immersion of the hand in tepid and colder water, the tepid water being used to open the vessels very gradually, and the colder water to subdue pain if this threatens.

When there are minute painful foci of necrosis, healing is intractable and pain continues unless the hands are constantly warm. The best means of accomplishing this, short of sympathectomy, is by treating patients in bed. The fingers affected by necrosis must be guarded against friction or other painful contacts.

### VASODILATOR REMEDIES

The use of warmth, general and local, has already been described sufficiently.

*Vasodilator Substances.*—Many different preparations have been advocated and employed in the treatment of the severer forms of the malady, with the idea of maintaining the circulation to the extremities. The chief of these substances have been mentioned on page 49. As in chronic arterial disease, so in the present malady, and for similar reasons, they have little or no value. I have known manifestations of the malady to disappear completely in a patient who developed Graves' disease. Thyroid gland has long been used as a remedy; neither this nor any other substance is known that can be

safely used to produce adequate and persistent dilatation, with the exception that thyroid offers a prospect of real benefit in patients with low basal metabolic rates.

*Periarterial Stripping.*—I have seen no improvement follow from this operation.

*Sympathectomy.*—It has been shown beyond any doubt that sympathectomy greatly improves the circulation to the limbs in these cases. From the hands of mild cases it abolishes the attacks altogether. In severer cases it diminishes the frequence and severity of attacks, but does not abolish them; it decreases the frequence with which necrotic foci develop, but does not prevent them appearing entirely. It is the rule for the attacks to be abolished by sympathectomy in the case of the feet. That is so, in the first place, because the affection is almost always milder in the feet than in the hands; in the feet gangrene is almost unknown in this malady. It is so, in the second place, because when limbs are cold normal vasomotor tone is much greater in feet than in hands.

Because the vasodilatation that follows sympathectomy is not fully maintained, the initial effects of sympathectomy in this malady likewise decline, but more than enough effect remains fully to justify the operation in selected cases; both temporary and permanent results are often dramatic. From the simple standpoint of the vascular result the operation has everything to recommend it. The loss of sympathetic supply to viscera is seemingly without any serious consequence. This is true of the heart in the cervical operation and of organs in the lower abdominal region in the lumbar operation. Loss of sweating in the affected limbs is advantageous, for it helps to keep them warm. But the operation is not without its disadvantages. Apart from operative risk, and this is now slight in skilled hands, there is disfigurement to consider. In the case of the cervical operation there are scars at the base of the neck; more serious, however, is the permanent contraction of the palpebral fissure and of the pupil, which in unilateral operations results in obvious asymmetry. Some young women naturally refuse the operation on this ground, preferring to suffer from painful as well as discoloured fingers in cold weather. Another and serious disadvantage is the occurrence of neuritis; this is a complication of the cervical operation and especially but not exclusively of that which employs a dorsal route, in which intercostal nerves are apt to become involved in scar tissue. This neuritis may clear away in a few weeks or months, but is sometimes intractable, continuing for years

and counter-balancing any beneficial effects of operation. The possibility of neuritis must be borne in mind in recommending operation.

In the case of the feet where operation is so uniformly successful and almost free of complication, it is not often required. It is unjustifiable to recommend it for mere discoloration of the feet; but if the feet are often painful a symmetrical lumbar operation should be done. The threat of nutritional changes in the toes would also justify sympathectomy, but as stated previously, such changes are very rare.

In the case of the hands, the operation should be confined to cases in which nutritional changes are occurring or in which attacks are very easily induced, appearing every day in cold weather. The operation is not justified in mild cases unless there is clear evidence of deterioration; for many of the cases react well to general management, remain stationary, or even cure spontaneously. Even in the severer cases the gain and loss by operation must be weighed. The patient should always be informed of the facial disfigurement which is certain to follow operation. Belief or disbelief in the theory that the malady is due to overaction of the sympathetic should not be allowed to weigh for or against operation. The justification of the treatment is to be founded upon experience and not upon theory.

The arrangement of the cervical sympathetic ganglia in man is variable. Usually the ganglia corresponding to the 7th and 8th cervical and the 1st dorsal segments are fused to form the inferior cervical ganglion, while the 5th and 6th cervical representatives form a separate or almost separate mass. Together these are equivalent to the "stellate" ganglion of the cat. The cell stations of the post-ganglionic neurones lie in these ganglia. Temporary denervation could be effected either by simple section of the sympathetic cord below the ganglia, or by section of outgoing branches. But it is desirable to prevent regeneration and to make denervation permanent; and so ganglia are removed.

It is enough to take away the inferior cervical ganglion; it is highly improbable that any sympathetic fibres from 2nd dorsal or from 5th and 6th cervical roots supply vessels much below the elbow; the object is to denervate the hand, and relieve the vessels of their normal vasomotor tone.

The lumbar operation has been referred to on page 50.

### 3. Intermittent Spasm of Digital Arteries with Diffuse Scleroderma

The patients are almost always women, and the malady is usually well developed by the twenties or thirties; it occurs rarely in young children. It is the rule for the cases to present the attacks of discoloured fingers as in the last group, though they may be less pronounced; the reactions of the vessels to tests are the same in the two groups. There is sclerodactyly, but the fingers are more often swollen than tapered; when necrosis occurs it may take the usual form or be ulcerative and extensive. Scleroderma is not confined to fingers but is more diffused. The skin of the hands is invaded, the natural creases of its skin disappear, extensibility and mobility are greatly reduced, and the soft tissues acquire conspicuous hardness. The stiffened fingers become semi-flexed; both flexion and extension are limited; the hands become more and more crippled. The forearms and even upper arms participate. The feet are relatively immune, but the toes are involved not infrequently and the feet and legs occasionally.

The disease often affects the face, tightening the skin so that natural folds and wrinkles are obliterated; the face becomes smooth and expressionless; its tissues are shrunken. The tightness of the skin and its immobility are particularly noticeable over the forehead and malar processes, and around the eyes and mouth. The mouth is reduced in size, and will not open as widely as formerly, owing to palpable loss of suppleness; the cheeks may feel hard from within and on occasion the tongue may suffer similarly. The forehead cannot be wrinkled, nor the eyes tightly shut, though the lids can be approximated. The eyelids may be so drawn that a little chink appears laterally between lid and eyeball. Blotchy telangiectases are frequent over malar processes and nose. The ears are often atrophic.

The front of the upper chest is often affected, less commonly the skin of trunk and thighs is involved.

Pigmentation of the affected areas is frequent. Arthritic changes are not uncommon; these may be atrophic, but also hypertrophic and proceeding to ankylosis. The arthritic changes combine with changes in more superficial tissues to immobilise the joints and render the patient helpless.

Sections through affected skin show the Malpighian layer to be atrophic, with loss of its deep processes. The deep layers of the skin

are fibrosed and this fibrosis extends into the subcutaneous tissues, and in the limbs into the muscles; in the fingers it binds skin to bone. Arteries like the digital vessels are surrounded by fibrous tissue and their channels greatly reduced by intimal thickening.

This grave malady may develop out of a condition at present indistinguishable from that last described, but it is unusual for this first stage to be long lasting. The changes in the texture of the skin and underlying tissues may appear simultaneously with the attacks of discoloured fingers. There are cases in which no history of digital asphyxia can be obtained and in which no such attacks are seen. Usually the malady becomes established rapidly and then becomes stationary or very slowly progressive.

Two views can be held. First, that intermittent spasm of arteries constitutes the original state and that this leads on to atrophy and fibrosis of the tissues in the affected vascular territories. The other is that diffuse scleroderma arises from a primary disturbance in the skin and subcutaneous tissues, an infection or toxic process, leading to a state of subacute or chronic inflammation; and that fibrosis of the tissues with changes in the vessels are secondary to this inflammation. The second view is supported by the occasional febrile onset, and by the frequent occurrence of oedema of skin or subcutaneous tissue in the regions later becoming fibrosed. It is the more acceptable, but its adoption brings the Raynaud's phenomenon, displayed by these patients, to be regarded as purely the result of disease of the digital arteries, and of vessels of comparable size in the face and other parts presenting transient asphyxia from time to time.

The malady is here separately described, not only because of its pathological interest, but also because its recognition and differentiation is important to prognosis.

*Course.*—In its early stages the affection is often seen to progress rapidly; such progress may continue, but more often the advance is arrested or, having invaded hands, arms, and face, it moves more slowly. When progressive the limbs of the patient become more and more crippled, the advent of arthritic changes helps to render the subject bedridden. In a few patients the skin of the body generally becomes rigid and the subject so hide-bound as to be almost incapable of movement.

*Treatment* of the hands and feet follows the lines already laid down on page 68. Massage should occupy a prominent place in treatment, should be administered daily, and continued for very many months. In addition to increasing the warmth of the limb and

reducing attacks of arrested circulation, sympathectomy is reputed to make the skin more supple; when this happens it is probably due to a reduction of oedema in the tissues affected.

### 4. Raynaud's Phenomenon Due to Local Injury or Vibration

Rare cases have been described in which an injury to a finger, such as a blow followed by swelling, has left the finger, otherwise in perfect condition, susceptible to cold, so that it frequently becomes blue or waxy white in cold weather. The symptoms are confined to the injured finger. Whether there is structural change in the arteries or not is unknown; but the abnormal responsiveness may disappear spontaneously after a year or two.

Since the introduction of various tools and machines which transmit rapid vibrations to the hands using them, cases of Raynaud's phenomenon have occurred amongst the users. Thus, some of those who for years have held vibrating pneumatic chisels, or have moulded boots by means of "pounding machines", develop this phenomenon. It is the hand holding the vibrating end of the chisel, and the fingers nearest to the end, which suffer earliest and chiefly; and it seems established that vibration is the cause. The fingers do not become discoloured during work; they acquire as the result of this particular work a permanent susceptibility to cold. The fingers become discoloured and numb in cold weather or after washing in cold water. The tested reactions of the vessels are similar to those described for previous groups. The affection may progress beyond mere discoloration to necrosis of the finger-tip.

*Treatment* consists first of all in changing the patient's work; subsequently it follows the lines laid down on page 68.

### 5. Raynaud's Phenomenon with Primary Disease of Artery

When the arteries of a limb are diseased, especially when the arteries of the palm or fingers are seriously involved, the corresponding territory presents the signs of relative ischaemia; the skin of the affected fingers is colder than that of symmetrical or neighbouring fingers. In such cases it is customary for the fingers to become more than usually cyanosed when the subject goes out on cold days or cools his hands in water. In some of these attacks of

discoloration the Raynaud phenomenon is manifested completely, that is to say the cyanosis becomes full, or the finger assumes waxy whiteness, and it becomes numb, subsequently showing reactive hyperaemia on its recovery. The cases here referred to are cases in which disease of the artery is primary; for example, instances in which thromboangeitis affects the arteries of the hand, or the same vessels have become obstructed by acute thrombosis (see pages 77-8) or consecutively to cervical rib (see page 80). Such cases are important because they must be differentiated for purposes of treatment; this is done largely by methods described in Chapter I. The cases are of interest, too, because they show conclusively that when an artery is greatly narrowed by intimal disease it may be brought to a state of occlusion by an increase of its tone, which presumably is natural in its degree.

## CHAPTER VIII

### GANGRENE (BILATERAL FORMS; CERVICAL RIB; GENERAL)

Cases of gangrene that are most clearly understood are those arising from obstruction of the main arteries of the limb, such as have been described under embolism and thrombosis, or in gross obliterative disease of these vessels. Such gangrene is almost always unilateral. Sometimes it follows in one limb months or years after it has come in the other. Exceptionally the gangrene may be bilateral from the start, as when emboli simultaneously enter the two limbs, or when the abdominal aorta becomes blocked by clot.

In this chapter certain forms of gangrene of more obscure origin will be described. We shall not consider instances of necrosis arising out of obvious physical or chemical injury, such as frostbite or burns by mineral acid, but shall confine ourselves to those in which the blood vessels of the limbs may be regarded as primarily involved. These will comprise certain forms of gangrene affecting the digits bilaterally, or quite symmetrically; and instances of gangrene of fingers following vascular disturbances in cases of cervical rib. These descriptions, with those that have gone before, will complete the clinical pictures and will permit a brief general description of the manner in which gangrene, consecutive to vascular trouble, originates.

Now symmetrical, or bilateral, gangrene has excited much interest since the time of Raynaud. This has happened because in so many of the cases no obstruction could be found in the main arteries; there was this difficulty in understanding it, and there was the unexplained symmetry. Speculation took command and plausibly blamed the nervous system, as in the case of the commonest form of symmetrical gangrene, namely, small necroses of the finger-tips discussed in the last chapter (see page 64). And the same assumption has crept in when a number of rarer and obscure forms have been described with-

out distinction from the latter, though it is most desirable to keep them separate.

### Bilateral Gangrene of Digits in the Young

This curious and fortunately rare malady attacks children or young adults; the sexes are equally affected. In many cases its origin is quite obscure, occurring as it does in apparently healthy young people in whom no pre-existing disease of vessels can be suspected. Very rarely there is a history of previous attacks of discoloured fingers or of chilblains. Usually malnutrition or other form of ill-health is apparent at the time. The disease has been recorded during the course of acute infections such as typhoid and typhus fever, during convalescence from pneumonia, and in other less clearly defined infections. The illness starts without warning, several fingers or toes becoming discoloured within a period of a few days; often the end of the nose or the ears are involved. The part is not attacked transiently or intermittently; it remains discoloured, recovering normal colour after weeks or months, usually with loss of tissue at its tip by a process of dry gangrene. It consists essentially of a single attack. The gangrene is considerable but usually dry, the end of the finger to the base of the nail, or to the base of the distal or middle phalanx, being lost. The necrotic tissue separates very slowly; healing may be prolonged for many months. The digits are almost always affected bilaterally, sometimes with strict symmetry, and parts of nose or ears may be lost. The area becoming necrosed is less than what is originally threatened, and deep discoloration may sometimes pass away from digit or nose without loss of substance.

Clinically these cases contrast sharply with those to which it is now considered necessary to restrict the term Raynaud's disease; the attack is single; the necrosis is massive; the ears and nose are frequently and seriously involved. The attack is not known to result from exposure to cold, though affecting parts that often become cold. The main arteries of the limb are unobstructed, the pulses are unchanged. There is no digital arterial spasm; in the prolonged attack the arteries of the fingers cannot be opened up by warming; pulsation is distinctly felt or recorded in them almost up to the discoloured tissue, but distally it is lost and cannot be recovered. An initial spasm of the vessels might conceivably start the process, but by itself could not result in permanent obstruction. It is now supposed, though it is as yet unproved, that the condition arises out of damage to the intimal coats of the digital arteries, which leads

up to their thrombotic occlusion. The idea gains support from the definite findings in the group next to be considered. It is of interest that to produce gangrene the process must occur in both arteries of each affected finger; though it need not be supposed to culminate in these quite simultaneously.

*Course and Treatment.*—The necrotic tissues separate slowly as previously stated, leaving shortened fingers. The ulcerated surfaces heal; the shortened fingers may exhibit quite normal vascular reactions, or there may be evidence of permanent damage to the arteries in their last segments. In such instances heating the hand will not bring capillary pulsation at the finger tips, and there may be complaint of coldness of the fingers and of attacks of blueness, pallor, and numbness on exposure to cold. The latter are not to be taken as evidence of a previous susceptibility to enter spasm on cooling, but are to be interpreted as a sequel to disease of the vessels imposed upon them at the time of the acute illness. It is the rule for the illness to end spontaneously with healing, though in very rare instances a second attack, in a slighter or equally severe form, may occur after an interval of months.

Treatment follows the lines laid down for gangrene of the toes in Chapter V. In the cases of the type here described, where pulsation can be felt in the vessels up to the region of gangrene, heat may be applied within the limits of comfort without fear of endangering the supporting tissue. Separation should be encouraged; amputation is not usually to be considered.

### Bilateral Gangrene of Digits in the Elderly

The subjects are elderly and of either sex, though males predominate. They may be in relatively good health at the time of onset, but have more often been suffering from wasting diseases, such as cancer, chronic tuberculosis, diabetes, or high blood pressure.

The illness has many points of similarity with that in the cases last described, with which perhaps it must ultimately come to be included. During the course of a few days, without exposure, and usually without warning, fingers of both hands become discoloured; the toes and the nose may also be involved. The circulatory disturbance progresses steadily to gangrene of the parts affected, much substance being destroyed. The circulation to the digits is normal to the border of the discoloured tissue; there palpable

pulsation in the arteries ends. The gangrene may be dry, or it may be moist and associated with fever and malaise. It is often accompanied by great pain.

The chief reason for separating these cases from those of the last group is that opportunities of examining the arteries have occurred. Now the digital arteries and those of the palm of the hand in elderly people nearly always show well-defined changes in the form of intimal thickening. The corresponding arteries in these cases show the same changes, though they are a little more conspicuous in degree; they are also the seat of recent and multiple thrombi, which plug the lumens of the vessels and evidently determine necrosis. The disease is found to obliterate both arteries of each affected finger.

Massive gangrene in elderly people, whether it involves a toe, a foot, or a foot and leg, is recognised usually to be associated with obstruction of a main artery. Here, however, the disease involves a number of small arteries; it is this fact which gives the cases special pathological interest, for it proves that gangrene of many digits, and of digits on two sides of the body, may result from multiple arterial obstruction. The reason why the final thrombi form simultaneously, or almost so, is undetermined; perhaps they are brought about by nutritional changes simultaneously affecting the endothelial lining of the vessels. It is quite usual to find small thrombi on the valves of the heart of patients dying of cachectic disease, and the origin may be similar in the two instances.

*Course and Treatment.*—Occurring in elderly subjects who are frail, and in whom the affected parts are often very painful, the malady often leads on to death from exhaustion or bronchopneumonia. In stronger patients the course and treatment are like those of the similar affection in younger people.

### Bilateral Gangrene with Haemoglobinuria from Cold

The patients recorded as subject to this rare malady have usually been males, frequently children, but sometimes adults. They are often affected congenitally or otherwise by syphilis. They suffer from attacks of haemoglobinuria brought on by exposure to cold, and experience one or more attacks of gangrene. The gangrene is usually bilateral, affects the tips of the ears particularly, but may also attack fingers, toes, or nose; in these it is usually sufficient to be disfiguring. It is generally clear that

the attack of gangrene has followed directly upon an exposure to cold. Gangrene may not occur, but attacks of cyanosis, or of whiteness of the fingers with swelling, which is evidently oedema, replace it, or gangrene and discoloration occur in different attacks in the same case. These cases are closely allied to, or pathogenetically identical with, cases of haemoglobinuria and urticaria developing in response to cold; this linkage is emphasised by the occasional occurrence of gangrene and urticaria in the same case. Haemoglobinuria from cold is due to the action of a circulating haemolysin upon the red blood-cells; the haemolysin unites with the blood-cells in the cold and they become haemolysed on warming up again. The fact can be demonstrated in a test-tube. In the patient the cooling happens as the blood flows through cold skin; haemoglobin is released when the blood returns to deeply lying parts and becomes warm again, and the liberated pigment is soon excreted by the kidneys. Urticaria from cold has been proved to be due to the action of a related circulating dermolysin; this can be transferred by injecting patient's blood serum into a normal skin, which then becomes sensitive to cold; the dermolysin unites with the skin-cells and these break down when warmed again. It is thought probable that urticaria in one case and necrosis in another are the result of different grades of injury to the affected cells of the skin; or, alternatively, that the chilled endothelium of the blood vessels in the skin is similarly attacked and damaged, leading to thrombosis, and thus to necrosis of the tissues supplied.

*Course and Treatment.*—The malady is a chronic one. When syphilis is associated energetic antisyphilitic remedies should be used, and offer the prospect of cure.

## Cervical Rib

Cervical ribs are usually bilateral though the degree of deformity is often asymmetrical and the symptoms consequently unilateral. The extra rib is attached behind to the 7th cervical vertebra and passes forward as bone, or oftener as a fibrous extension, to be attached to the true 1st rib behind the insertion of the scalenus anticus muscle. The brachial plexus, which in these cases tends to include less of the 1st dorsal nerve than usual, and the subclavian artery pass over the rib. Traced from behind forwards, the 7th cervical nerve usually lies on the rib, and the 8th, with or without a contribution from the 1st dorsal nerve, lies next in front. Then

comes the subclavian artery lying upon the end of the bony rib, upon its fibrous extension, or upon the true 1st rib.

The nerves, and the artery, which often arches higher than is customary in the neck, are subject to strain or compression, and consequently to damage.

*Symptoms.*—There are usually no symptoms. When these occur they come after adolescence is reached. They are of two kinds, namely, nervous and vascular.

The commonest nerve symptom is aching or burning pain in the hand, or in the arm, especially on the ulnar side; sometimes it is diffuse. Weakness and wasting of the small muscles of the hand occur; the abductor brevis and opponens pollicis are specially affected, sometimes the forearm flexors are involved. Sensory defects are frequent, consisting of diminished appreciation of touch and pain stimuli, and affecting the ulnar border of hand and forearm particularly but not exclusively. Such symptoms follow damage of the 8th cervical and 7th cervical nerves.

The commonest vascular symptoms are general coldness, blueness, and less often swelling, of the hand. These symptoms are usually brought by disuse (see page 99), itself the result of pain or weakness; they are then secondary to nerve damage.

The major vascular symptoms are rare; they occur alone, rather than in combination with the manifestations of nerve injury just described. They result from obliteration of the arterial channels of the limb by blood-clot. The onset may be sudden, with the characteristic picture of complete obstruction at the brachial bifurcation, in the radial artery, or in several branches. According to the position and extent of the clots the symptoms vary; there may be pain from ischaemic muscles; there may be simple discoloration and coldness of given fingers, coming spasmodically in response to cold, or showing more permanence; the discoloration may be associated with the numbness of ischaemia. Gangrene of finger-tips may develop. It has been noticed previously that certain of the common vascular signs arise out of nerve injury; here on the contrary, it is seen that pain and anaesthesia can follow vascular injury; in attempting to interpret the manifestations of cervical rib, these facts must be remembered.

The manner in which the arterial obliteration comes about has been disputed. It has been thought that it is due to pressure on sympathetic fibres travelling in the brachial nerve root, one view being that these sympathetic fibres are paralysed and that a conse-

quent trophic change happens in the main vessels, another view being that they are irritated, resulting in spasm of the vasa-vasorum; in either case nutrition of the wall of the main vessel is supposed to suffer and clotting to result. These views are highly speculative and cannot be regarded favourably. It is certain for some cases, and probable for all, that the obliteration actually arises out of direct damage to the main artery where it crosses the extra rib or where it becomes caught between rib and clavicle. It is known from clinical observation that the artery often becomes compressed in this region with loss of the distal pulse, when the arm is drawn downwards or the shoulder back, and sometimes when the arm is raised. Such compression, oft repeated, is calculated to damage the artery and to promote clotting within it; actually its walls have been found greatly thickened and adherent to surrounding structures, or the vessel dilated or aneurysmal and partly or completely filled with clot. All the vascular symptoms displayed could not arise, however, directly out of compression or out of permanent obstruction of the subclavian artery; but they could arise out of thrombosis with embolism. The thrombi form where the wall is damaged by compression; these, so it is supposed, become detached by repeated movement and from time to time, causing embolism of smaller or larger branches in the arterial tree. A large embolus plugging the bifurcation of the brachial artery would give rise to severe pain and quickly reduce the hand and forearm to an algid, cyanosed state, from which recovery would be slow and imperfect. Many small emboli lodging in digital and palmar arteries would gradually block these vessels and produce in the fingers the picture of Raynaud's phenomenon, or spasmodic closure, in response to such stimuli as cold. Emboli of brachial artery, or of its branches, followed by thrombosis, would bring actual gangrene to given fingers. Embolism would explain why the radial is affected more often than the ulnar side of the hand. Repeated embolism, or embolism followed by thrombosis, would explain why the blocking of a distal artery can be followed, as it is known to be followed, by a central extension of the obstruction. Thus thrombosis with embolism account for all the phenomena and for the interesting variations of symptomatology that are known to occur.

*Diagnosis.*—Recognition of cervical rib ultimately rests upon the characteristic X-ray picture. In cases in which vascular symptoms are merely the sequel to disuse, the diagnosis will be prompted by the symptoms and by the form of the sensory and motor paresis. In these, and especially in cases in which vascular symptoms arise from

obstruction of the arteries, diagnosis will often be reached or helped by noticing the pulsating swelling of the subclavian artery in the neck, the height to which it arches in the neck, the ease with which it may here be compressed against the rib causing loss of distal pulse, and sometimes by the readiness with which the pulse may be abolished by passive movement of the shoulder.

*Course and Treatment.*—The course of symptoms in these patients is progressive, and surgical intervention is strongly to be recommended. Removal of rib or fibrous extension, according to the structures pressed on or strained, is followed by relief or cure. In arterial cases improvement is ascribed to relief of arterial compression, and to the opening of collateral channels, which are able to persist if embolism ceases. Return of a radial pulse that has been lost is infrequent but may occur within days, weeks, or months of operation.

### CRUTCH PRESSURE AND GANGRENE

A state of affairs similar in almost every respect to what has been described for cervical rib may occur after the use for very many years of a crutch that presses upon and obliterates the axillary artery. In such a case the axillary artery may become thickened until it feels like a hard cord, while, presumably through embolism or thrombosis consequent upon malnutrition, the digital, radial, and ulnar arteries may become blocked successively and permanently, and gangrene of fingers supervenes.

## Origin of Gangrene

In this and previous chapters gangrene or necrosis of tissues of the limb has been noticed as a sequel to a number of distinct diseases of the vessels. The simplest way in which it comes is through closure of the lumen of the main artery by clot, this being embolic or thrombotic. The nearer the clot lies to the aorta, and the farther it extends in the artery, the more certainly does gangrene result. Obliteration of main arteries of the limbs may be a more gradual process, combining disease of vessel wall and mural thrombosis; gangrene is so determined in aneurysm, in the arterial disease of old men and diabetics, and in thromboangeitis obliterans. Here, however, it is to be emphasised again, that to produce gangrene a simple focal occlusion of the main artery rarely suffices; the clot must be so placed, or the vessels of the limb so extensively diseased, that the clot finally determining gangrene stops a channel to which

there is little or no alternative. A simple focal occlusion may at times induce an ugly discoloration of the leg; but the amount of blood-flow required to maintain life in the tissues of a limb that is progressively cooling is very small; and it is the rule for the amount of blood-flow to increase as time passes and collateral channels widen.

The manner in which gangrene is determined in these cases of structural disease is easy to understand, and these were the forms first attracting attention and receiving explanation. Later, new instances of gangrene were recorded in which the main arteries were noted to pulsate freely during life or were found patent after death. Arterial spasm was invoked as the determining factor, and this idea, once introduced, spread widely. It was assumed too readily that gangrene must be spasmodic in origin in otherwise unexplained cases. It is now recognised that certain of the cases previously believed to result from spasm are in fact due to the formation of multiple thrombi in small arteries; and it is probable that many other cases will ultimately come to be explained in the same way. Probably gangrene cannot be a direct and uncomplicated result of arterial spasm. When the circulation to skin is shut off, there comes into immediate action a mechanism producing vasodilatation and manifesting itself ultimately in "reactive hyperaemia". Constriction of the vessels, produced artificially by stimulating vasomotor nerves, is able to counteract this dilator mechanism in the early stages of its establishment; but as ischaemia is continued, the vasodilator factor increases in intensity and sooner or later the nerves will, as a result of ischaemia, themselves become functionless in the affected territory. Thus it would seem sure, even on *a priori* grounds, that a nervous vasoconstriction cannot be maintained long enough to kill the skin. Actually we have no knowledge of any clinical condition of arterial spasm of nervous origin continuing for days or even for hours. The idea is suppositious and based upon the difficulty hitherto experienced in understanding cases of symmetrical gangrene. That difficulty has largely disappeared since it has been discovered that symmetrical gangrene, even of fingers, may be a direct result of structural arterial disease.

Arterial spasm almost or quite to the point of occlusion and long maintained does occur clinically however. Examples of it have been described earlier (see page 56) as the outcome of concussion or bruising of arteries, or of periarterial stripping. But gangrene does not here result; the vessels relax and the circulation and the tissues

recover. The most notable remaining instances are the gangrene of ergotism and the small necroses of Raynaud's disease. Indubitably, increased tone may ultimately be responsible for death of the tissues in these; though in neither instance can it be said that it is the uncomplicated cause. In ergotism, where the arteries contract under the influence of poison, the final stage in the vessels is one of dilatation, the blood being in stasis or actually clotted. It is probable that the minute focal necroses of the finger-tips in Raynaud's disease are similarly brought about by stasis or by thrombus formation in the small arteries, arterioles, or smaller vessels.

*Dry and Wet Gangrene.*—Gangrene may be dry or it may be wet; which of these will occur depends on the amount of water in the tissue concerned at the moment blood-flow ceases, and on the rate at which the tissues subsequently lose moisture. Dry gangrene occurs in tissues deprived slowly of their circulation, and especially in parts that have exposed surfaces which are large relative to their volume; for both these factors favour loss of water from the tissues. Perhaps the most striking example of massive dry gangrene occurs in ergotism where, for days or weeks before actual necrosis happens, the blood-flow to the tissues is very greatly reduced and desiccation is occurring. Gangrene of the tips of ears or fingers is usually dry, because the surfaces which these structures present to the atmosphere are large. Wet gangrene is usual when the circulation to a big mass of tissue ceases abruptly; oedema of the tissues, whether this results from inflammation or otherwise, favours it. Massive gangrene of the leg is usually wet; so is gangrene supervening in inflamed tissue. It will be gathered that the type of gangrene, whether wet or dry, helps only to a limited extent in deciding its origin.

# CHAPTER IX

## VASODILATATION; FLUSHING

A BRIEF account of the vasomotor nervous apparatus has been given in the opening of Chapter VI, which subsequently deals with vasoconstriction. We may now take up the description of vasodilatation.

The word vasodilatation indicates in ordinary usage a dilatation of blood vessels such as will induce an unusual blood-flow to the affected territory. An increase in the size of the minute vessels has little or no effect on blood-flow; vasodilatation refers essentially to an increase in the size of small arteries and arterioles, in which control of flow is mainly exercised.

It is important clearly to distinguish between vasodilatation in the skin and flushing of the skin. Widening of the arteries and arterioles increases the pressure in and dilates the minute vessels; they dilate a lot if their tone is imperfect, as it often is in the skin of the face; they dilate only a little if their tone is good, as it is in the skin of the limbs. Thus a simple dilatation of arteries and arterioles immediately produces a bright flush in the face but only a slight flush in the skin of the arm. In both instances the increased irrigation and better nutrition of the skin results in a subsequent increase in the tone of the minute vessels; this is sufficient in the case of the arm and sometimes in the case of the face gradually to render the skin paler than usual, although, while the arteries and arterioles remain dilated, it continues to be hot.

Passive distension of the minute vessels, and consequent flushing, results both when the arterial inlet is increased and when the venous outlet is obstructed, for in both instances pressure in the minute vessels rises. A flush also results when, pressure remaining constant, the minute vessels lose tone; this they are very apt to do in all states of inflammation, acute or chronic, and in states of chronic malnutrition of the skin.

Briefly, vasodilatation may be present when the skin is unflushed; and flushing may come without vasodilatation; the two terms are by no means synonymous.

## General Vasodilatation

In health vasodilatation of central nervous origin is produced by embarrassment, and appears as the familiar blush. This though displayed vividly in the face, largely because the facial vessels dilate easily under internal pressure, is probably a general vasomotor effect; the reddening is sometimes noticeable in the hands, and I have seen deeply coloured cyanotic legs become bright red from knee to foot simultaneously with blushing of the face under emotion. Vasodilatation is also produced when the blood passing to the regulating nervous centres is warmed, as results after moving from a cold to a warm room, or after soaking a limb in hot water (see page 14); this is definitely a general vasodilatation.

Of substances producing general vasodilatation, two occur as natural tissue products, namely histamine and acetylcholine, in free or combined forms. The release of these substances into the blood stream in concentration sufficient to cause perceptible vasodilatation is, however, as yet unknown in physiological circumstances. Histamine when introduced intravenously dilates both arteries and minute vessels, and brings about vivid flushing with rise of temperature of the skin and a fall of blood pressure of many minutes' duration. Acetylcholine acts on the arteries but, owing to its rapid destruction in blood, gives only uncertain and fleeting effects when injected into a human vein. Of vasodilator substances foreign to the body, amyl nitrite and glyceryl nitrate (nitroglycerine) are the best known, the former for its profound but fleeting, and the latter for its more prolonged, effect.

In disease, the most striking instances of general vasodilatation occur in fever, where blood temperature directly influences the regulating centre, and in free aortic regurgitation and in Graves' disease, though the way in which it is brought about is not understood in either of these two diseases. Sudden flushing of areas of the face, neck, trunk and limbs, such as is seen often in nervous patients, and at the menopause, is to be distinguished from blushing, being vivid over a greater area and of much longer duration than the latter and unassociated with mental confusion.

## Local Vasodilatation of Nervous Origin

The clearest example of local vasodilatation of nervous origin is that which follows division of any part of the tract of sympathetic fibres from spinal cord to periphery; for normal vasoconstrictor tone is then lost. In the upper limb and face this happens when the corresponding inferior cervical ganglion is destroyed by disease or is deliberately excised; in the legs it happens in spinal anaesthesia, and in the foot when the lower lumbar ganglia of the corresponding sympathetic chain are excised. The vasodilatation produced in any of these ways displays itself by reddening and by increased warmth of the skin. The face becomes deep red, the colour of the hand deepens very distinctly, and that of the whole skin of the arm perceptibly. The degree to which the minute vessels become engorged depends upon their intrinsic tone in the region affected. The skin of all parts becomes hotter, but the rise is greater in the hand than in the face, for the initial temperature of the hand is lower. Greater warmth of the denervated area is maintained in most circumstances (see page 98); but increased depth of colour does not usually continue. It is usual for the skin to lose its increased colour, or actually to become paler than normal. This paling may begin within a few hours of the loss of sympathetic tone; it has always happened within a few days. The increased tone of the minute vessels is due to reduction in the concentration of natural vasodilator substances in the tissue spaces under conditions of increased blood-flow, and to an increased sensitivity, which the denervated vessels acquire to circulating vasoconstrictor substances. The point of practical importance is that flushing caused by loss of vasomotor tone is impermanent. More will be said about the behaviour of the denervated limb in the next chapter.

### Reflex local vasodilatation

There is some evidence derived from animals that a reflex vasodilatation in a limb may be started up by a sensory stimulus passing from the limb. There is no certain instance of such a reflex in man, but there are phenomena that are possibly to be explained upon the basis of such a spinal-cord reflex. Thrombosis of the common femoral vein in patients raises the temperature of the corresponding limb. Tying the femoral vein occasionally heals chronic ulcer of the leg in cases of gross arterial disease; a similarly produced vasodilatation is perhaps responsible. The rise of temperature in the

first example occurs early and continues for days or weeks, gradually subsiding. It is difficult to understand how this vasodilatation can be produced unless through reflex nervous channels. It is possible that the vasodilatation which has been reported to follow periarterial stripping (see page 50), and which lasts for a similar time, results similarly by reflex from injury of the vascular sheaths.

### Local Vasodilatation and Flushing of Metabolic Origin

Local vasodilatation, unlike local vasoconstriction, is extremely common and occurs in a variety of circumstances. It has been stated earlier that blood-flow is governed locally by the action of tissue metabolites; accumulation of vasodilator substances is responsible for most instances of local vasodilatation. In health, such metabolites largely regulate the blood-flow to muscle; during work the metabolites accumulate and cause an increased flow to muscles in use; arrest of blood-flow to a limb causes natural vasodilator substances to gather in it, especially in the skin, and these are responsible for reactive hyperaemia at the release (see page 21). In both these instances flow is adjusted to meet nutritional needs. Another and simple instance of appropriate physiological adjustment is the influence of warmth; a rise of temperature in the skin increases its nutritional need, and brings almost simultaneously an increase of metabolites and of blood-flow.

### INFLAMMATION

The most frequent way in which vasodilatation and flushing are produced locally in limbs is through the process termed inflammation. The vascular component of inflammation alone concerns us here; it is well seen during the development of the familiar lesions called urticaria (factitious or otherwise). These lesions itch and present three separate objective features. First, there is a quite local dilatation of minute vessels, producing a central red spot or line. Second, the central area wheals. Third, when the sensory nerves of the skin are intact and then only, a bright flare having an ill-defined and irregular margin appears in the surrounding skin. These separate phenomena together make up what is called the "triple response". All parts of this response are due to the same immediate stimulus, namely the release of a natural histamine-like substance from damaged cells into the tissue spaces. The triple response can be reproduced faithfully in all its parts, including itching, by directly introducing histamine into the skin. The natural substance, like

histamine, acts directly upon the minute vessels locally, relaxing them and increasing their permeability, whence the local red spot and the wheal. It acts also upon local sensory nerve-endings in the skin and, through an axon reflex, causes arterioles to dilate; by producing this flare it not only further increases blood-flow to the inflamed area, but also increases it to immediately surrounding parts. The release of substance and the vascular phenomena of acute inflammation as they have been described, can be brought about by any kind of injury, scratching, freezing, burning, or by introducing poisonous substances into the skin.

There are many reactions of a less acute kind, brought about more gradually and lasting longer, which almost certainly make use of the same mechanism, though in a modified way. The reaction of the skin to sunlight or ultra-violet light is a simple illustration. There is reddening, heat, and a little swelling; these last for days. Chilblain is another form of chronic inflammation, modified by occurring in skin that is almost habitually cold; there are others still more obscure. It is not always easy to recognise the ultimate cause of inflammation, or sharply to distinguish effects of inflammation and malnutrition; and thus when skin is reddened chronically and the cause is not fully apparent, the idea that the underlying process may be "vasomotor" in origin is apt to acquire undue credence. It is not long since this explanation was held for urticaria itself; it is still widely but wrongly held for the three states presenting flushed skin next to be considered.

### Acrocyanosis or Acroasphyxia

The condition occurs mainly in young women. The hands, usually to a little above the wrists, are persistently of higher colour than normal; they are unusually cyanosed when cold, they are bright red when very cold or when they are warm; often they present a mixture of the two colours red and purple. The hands are in the average much colder than normal and the palms are often sweaty. There may be a little numbness of the fingers to touch, heat, and cold, though these paraesthesiae are inconstant and are largely attributable to coldness of the hands. The hands warm up in a hot room and may then give rise to a sense of heat. The skin shows little change, remaining unthickened and supple; the nails are normal. Puffiness of the hands occurs in some cases, especially in winter time; occasionally the follicles of the skin are unusually prominent. Almost always the hands are equally affected, the feet are also affected or

escape discoloration, though they are cold. The fully developed state is infrequent; milder types are common, and in these high coloration is confined to the winter and chilblain is a frequent association. The discoloration may develop rapidly to become intense within a few weeks. There is little doubt that persistent coldness of the hands is largely concerned in producing the high colour of the skin; more of the arm was involved when short sleeves became fashionable. It is doubtful, however, if coldness affords a full explanation; for it provokes acrocyanosis more readily in one subject than in others. There is no constant clinical association; the malady is common in mental cases of lethargic types.

The minute vessels of the skin are very dilated, as is evident equally from macroscopic and microscopic examination. But the temperature of the hands and other tests show the blood-flow to the skin to be reduced greatly. The veins though contracted by cold are not occluded. The pulses in the main arteries are normal. The constriction is in the small arteries or arterioles of the skin. If the hand at the time is cyanosed and a small part of it is warmed, the latter soon becomes sharply defined as a bright red area; similarly if a little histamine (1 in 3000) is pricked into the skin, the skin reddens locally and its temperature rises. This is in contrast to the cyanosed skin in the attack of Raynaud's disease, where the obstruction lies in the main digital arteries, and in which reddening of the skin does not occur in similar tests, but only after these arteries open. In acrocyanosis all the arteries and arterioles are capable of opening widely; gross structural impediment is not present in any of their channels.

As with almost every known and unexplained vascular discoloration of the skin, functional derangement of the vasomotor nerves has been regarded, though without adequate reason, as the source of acrocyanosis. A vasomotor dilatation giving conspicuous widening of the minute vessels of the skin would always produce hot and not cold hands, since the arterioles would also be open. The arterioles are in fact not in a low but in an unusually high state of tone; and this increased tone is due to a fault in the vessels concerned, for it is not quickly relieved, as it would be were it vasomotor, by anaesthetising the nerves. The conclusion is that the vessels are unusually susceptible to cold, this susceptibility being in this instance in the arterioles of the skin.

### TREATMENT

The malady is not serious and treatment when called for con-

sists in avoidance of exposure and in maintaining the warmth of the hands in the manner described on page 68.

### Erythrocyanosis of the Leg

This very common state illustrates what may probably be regarded as one example of a common class of chilblain phenomena. It is described, though briefly, because occurring in the leg or elsewhere it is frequently but wrongly regarded as a "vasomotor" disturbance, and because it is relevant to the condition to be considered directly afterwards.

It is almost confined to young women; it is usually bilateral, though often more severe on one side than the other. It affects the lower part of the leg, the front and outer side above the malleolus being the common site. Here a deep vascular discoloration of the skin occurs, in which cyanotic tints of advanced grades prevail. A single large area is usually involved; it is often many inches long by a few inches broad; the outline is diffuse for the most part; there may be several smaller areas of discoloured skin around the ankle. Small or large patches may occur on the dorsum of the foot where the lower margin sometimes sharply marks out the upper margin of the shoe. The discoloration is disfiguring. The skin is often found tender; the tenderness has the peculiar qualities presently described under erythralgia, there is unusual sensitiveness to friction and to warmth. In the worst cases the skin is in an obvious state of chronic inflammation, being generally swollen, and resistant or nodular. The nodules sometimes break down and ulcerate. The remaining skin of the leg is often marked by prominent and reddened follicles. The affected skin, and indeed the skin of the leg as a whole, is cold to the touch.

As in acrocyanosis, while the minute vessels are dilated, the arterioles are unusually constricted. The inflammatory condition is proved by histological examination, which reveals lymphocytic infiltrations, largely perivascular, of the skin and subcutaneous tissue.

This malady is undoubtedly determined by prolonged cooling of the skin, though it requires also the more obscure background of susceptibility to explain it. It came in with short skirts and thin stockings and will go out with them. It comes or increases in the winter months, and improves or vanishes in the summer.

### TREATMENT

Those who for aesthetic reasons ask for help are loath to accept it when they find that advice conflicts with fashion and insists on warmer clothing. Those who have advanced to the stage of pain are more amenable. The burning pain may be severe and, as it is often provoked by dependency of the foot and by walking, may even be crippling.

The quickest and most sure relief in such cases is given by sending such patients to bed and by keeping body and feet thoroughly warm. If this warmth brings much pain, it must be applied moderately and increased as improvement occurs. When the skin is less sensitive, or after the skin has been brought to a healthier condition, massage of the legs is to be added and the patient should try to avoid recurrences by exposing the legs to cold and wind as little as possible. Sympathectomy may be done in very severe cases.

## Erythralgia

The term erythralgia is used to designate a redness of the skin accompanied by a peculiar form of tenderness. Pain is induced by stretching the skin between the fingers or by friction; this tenderness may be extreme, so that the gentlest manipulation elicits pain, and rougher contacts are hardly tolerated. Pain is also provoked by warming. To the normal skin, water at 41° to 42° C. (about 106° to 108° F.) conveys a pleasant sensation of warmth, and it is only when the temperature rises higher that pain begins; the erythralgic skin responds painfully to water at 40° C. (104° F.) or less. In certain circumstances, as when the skin is turgid with blood and in extreme cases without this extra tension, pain is induced by a rise of skin temperature to levels even below blood heat (about 30° to 31° C. or about 86° to 88° F.). In erythralgia, the skin may be said to be in a susceptible state, the pain nerves responding to stimuli of a strength quite inadequate in normal circumstances. It is a matter of indifference how the skin temperature becomes raised, whether from flushing of the part with warm blood or from an application of external heat, the result is the same. Cooling relieves or abolishes the pain unless a low temperature is reached; the application of ice gives pain indistinguishable from that induced by heat. The pain when provoked is of one kind, namely, a continuous pain of the kind produced by burns, a smarting, stinging, or burning pain as it is variously described.

A characteristic is the reaction to friction or stretching; this induces immediate pain, which quickly subsides, but shortly surges up again to continue for several or many minutes after the original stimulus has ended.

Erythralgia can appear in any part of the superficial coverings of the body, including the accessible mucous membranes and perhaps the subcutaneous tissues; it is not confined to leg and arm. It can be produced artificially, within a minute by burning the skin, after an interval of many minutes by scarifying the skin or by applying irritant substances, and after many hours by freezing the skin or by reddening it by means of ultraviolet light. The burning pain induced by the friction of clothing or by warming in skin recently reddened by sunlight is familiar; such skin characteristically displays the group of symptoms described as erythralgia. All the agencies mentioned produce in the skin a certain state of inflammation. Erythralgia is frequent in acute inflammations of the skin that are due to pyogenic organisms. It occurs also in many instances of more long-lasting inflammation, for example that caused by hard usage or by frost-bite. It is not infrequently found associated with a condition of general urticaria factitia, the wheals of which do not itch but give burning pain instead. It is frequent when the toes become inflamed and necrosis threatens in gross arterial disease of the leg (see page 43), in chronic chilblains of the foot, and in the condition erythrocyanosis just described; these last are conditions in the production of which diminished circulation plays an active part, reducing resistance to infection, exposing the tissues to the damaging action of prolonged and repeated over-cooling, and tending to render inflammation intractable when it comes.

In searching for a pathological factor common to all these states, we see inflammation as the clearest possibility. For this reason erythralgia has come to be regarded as linked with this process; whether or not it is exclusively a product of inflammation, or of a particular concomitant of inflammation, is still undetermined. Putting theory on one side, it is important for practical purposes to identify erythralgia when present, recognising that it usually consorts with manifest inflammation, but that it occurs also under conditions that are still obscure. It has nothing to do with abnormality of the vasomotor system; it always results from a local process. The mechanism of the pain has been shown, in nearly all the instances named, to be a release into the skin of an unknown substance, not histamine though presumably arising out of damage to the cells, which lowers

the threshold of the pain nerve endings to various forms of stimulation.

Erythralgia in a chronic and very painful form occurs most often in the foot; it may have one of the associations already named, or it may be obscure in origin. It may be unilateral or bilateral. In the foot the clinical picture, however it arises, is a striking one. The affected skin, usually cold, is red and very tender to the touch. When the foot is dependent its colour deepens conspicuously; this is purely a passive effect, being due to the hydrostatic increase of pressure in minute vessels, which lacking normal tone are unusually prone to dilate. Pain, of the characteristic persistent burning type is induced by heat and by the dependent posture. These patients are fearful of placing their feet in hot or even warm water; the warmth of a bed is often enough to induce severe pain, and they can sleep only with the feet projecting. Dependency of the foot brings pain by increasing tension in the skin; it is produced with equal ease by obstructing the venous return while the leg remains horizontal, or by any other act which stretches the skin. Walking provokes pain quickly, because it both warms the feet and rubs the skin. The pain is so easily induced in some subjects as to cripple them completely; they cannot set foot to ground without pain; and the pain when severe and intractable has driven some such patients to suicide.

*Erythromelalgia* (painful redness of the limbs) was a term introduced by Weir Mitchell to distinguish what he thought to be a specific disease. The conception as it has stood is of a disease in one or more limbs, in which attacks of burning pain are induced by warmth or dependency of limb and are associated with vasodilatation. It is clear that a number of distinct diseases, displaying the common syndrome here called erythralgia, have been included in this category; it is also clear that the view that there is a painful malady associated essentially with vasomotor storms is incorrect. The mistake arose partly out of the idea that reddening of the foot on dependency is significant of vasodilatation, whereas it is a passive affair. Secondly, the simple relation of pain to warmth, namely, that pain can be induced by applying warmth as well as by natural vasodilatation, was not realised. This new orientation of ideas makes it expedient that the term erythromelalgia, with its inevitable implication of vasomotor storms in the limbs, should be abandoned.

### TREATMENT OF ERYTHRALGIA

The treatment must be guided by the cause and has been dealt with already from place to place in this book.

In cases in which ultimate cause remains indeterminate, treatment must be upon general lines. Rest in bed for long periods is essential in severe cases. Opiates must be used very cautiously, to avoid risk of creating habit. When the limb is persistently cold, measures should be adopted to increase its circulation; but their application is limited owing to pain being aggravated both by friction and by warmth. Massage, diathermy, and other remedies used to improve the blood-flow in other states are not well tolerated; but they can be employed cautiously, and increased as tolerance to them is gained. The body should be warmly clad to keep vasomotor tone low, and the limb kept by this means, and with or without cover, as warm as it can be borne. The principle is to increase blood-flow and the nutrition of the part as far as possible without provoking excessive pain. These patients are often much better in summer than in winter. That is so because, although cold gives relief to pain, it aggravates the underlying condition, while warmth has the contrary effects. Treatment that is applied may follow the lines of this natural remedy. Periarterial stripping is not recommended. Lumbar sympathectomy deserves trial.

# CHAPTER X

## VASCULAR DISORDERS IN DISEASES OF THE NERVOUS SYSTEM

In this chapter, the manner in which vascular changes in the extremities are brought about in disease of the nervous system, and certain of their consequences, will be discussed. Consideration will be given mainly to what are commonly called "vasomotor disturbances" in the limbs. This term has come to be used almost as an equivalent of colour change in the skin. It should never be given so loose a meaning; strictly it can only be used to designate such changes in limb circulation as are known to result from disturbances of the vasomotor nervous system and in this sense its use is rarely called for. There is one aspect from which "vasomotor disturbance" will remain unconsidered in the present chapter, namely, local disorders of the circulation to the limb, such as have been believed to arise out of functional upsets of the central vasomotor apparatus. These beliefs have been discussed enough in previous chapters (Chapters VII and IX), which separately include a description of Raynaud's disease, formerly believed due to pathologically increased, and of erythromelalgia, formerly believed due to pathologically decreased, vasomotor tone.

A matter that will also engage attention is the part played by vascular changes in processes that are called "trophic". There are not to-day many phenomena of disease that can even plausibly be retained in the category of "trophic changes", if we mean by trophic change one that is due to the withdrawal of a direct nutritional influence of nerve cell upon affected cell. More and more of the individual changes regarded as trophic in the past have become recognised as resulting in ways various and quite distinct from this. It is within the scope of this book to discuss only those ways in which a circulatory factor plays an important part.

## Loss of Sympathetic Innervation; Vasomotor Palsy

It has been stated in the last chapter that vasodilatation follows division of any part of the tract of sympathetic fibres from spinal cord to periphery, and that this vasodilatation displays itself in temporary redness and in increased warmth of the corresponding skin. The vasomotor fibres never suffer alone in destructive lesions of the sympathetic nervous system, but sweating and pilomotor effects, in so far as they are initiated centrally, disappear simultaneously; in destructive lesions of peripheral mixed nerves, sensory

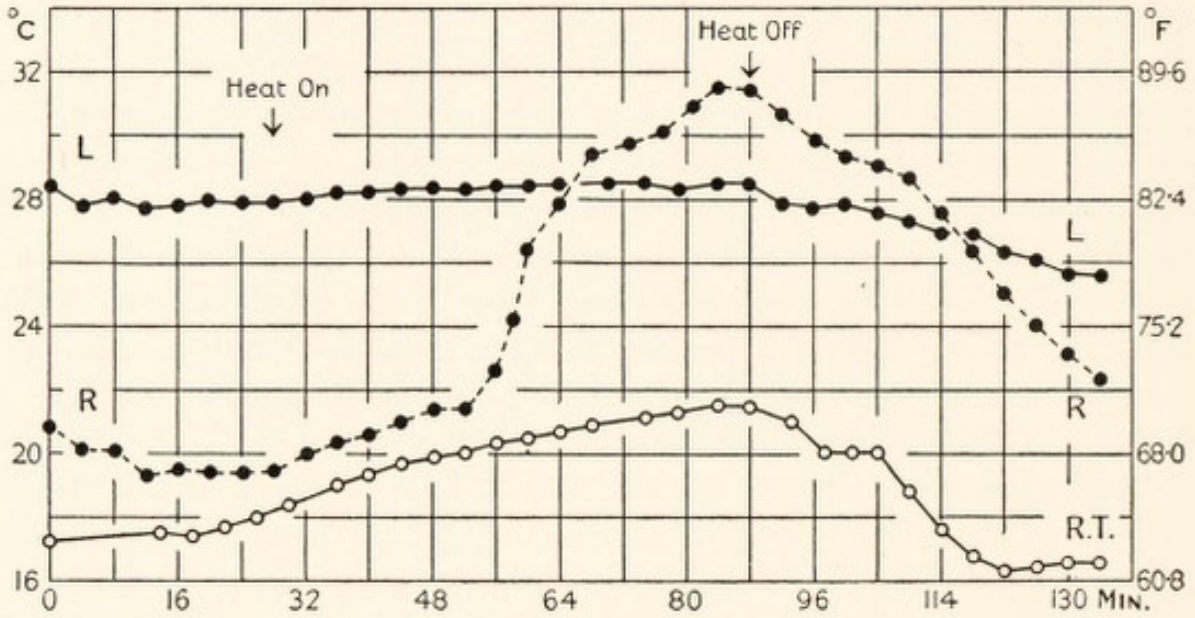


FIG. 5.—Continuous records of temperature from a finger of the right (R) and left (L) hand, taken from a case in which the left cervical sympathetic ganglia had been removed. The chart illustrates differences in the response of the fingers to change in the temperature of the room. The left (sympathectomised) finger was in the average warmer, but showed little change throughout. The temperature of the right finger was at first much the lower; later, as room temperature (R.T.) rose, vasodilatation set in, and the right finger became warmer than the left; as the room cooled again the right finger again became cooler than the left.

defects of the skin are likewise associated. There is very close correspondence in the cutaneous distribution of all these vasomotor, sudorific, pilomotor, and sensory, defects, a statement which may be given point by adding that in fresh peripheral nerve lesions reddening and loss of sensation are found in almost identical territories.

The increased depth of colour produced by sympathetic section does not continue; this is especially so in the case of arm and leg, hand and foot, where it is the rule for actual paleness to develop. Directly after sympathetic denervation the temperature of a limb is the same as that of the opposite limb under circumstances of full vasodilatation; this state also is temporary, the arteries and

arterioles regaining some of their lost tone after a period of days. Eventually a permanent condition becomes established in which the arterioles, though relaxed, are incompletely so. It is then the rule to find the limb, or the affected part of it, warmer than the symmetrical part; but if the body is heated sufficiently to produce general vasodilatation, the temperature of the normal part rises until it equals and ultimately exceeds that of the denervated part (Fig. 5). Thus, it is not exact to say that the sympathectomised part is the hotter; this is only true for most of the circumstances of ordinary life; it is the mean temperature which is higher. The characteristic of vasomotor palsy is stability of temperature; there are not the fluctuations of vasomotor tone, which keep the normal part usually at lower, but sometimes at higher, temperatures than the denervated part.

Vasomotor palsy cannot be recognised by the colour of the skin, nor by isolated observations upon temperature, but only by observing that normal vasomotor responses to warming and cooling the body fail. The recognition of peripheral vasomotor palsy is often aided by the associated paralysis of sweat glands and of hairs. The skin of the denervated hand is dry, and may present fine desquamation.

### Disuse; Motor Palsy

*Simple Disuse.*—If a normal subject moves about in a cold room, using one hand to execute simple tasks, and allowing the other to hang passively at the side, the temperature of the fingers of the latter will fall several or many degrees in a half-hour. Use of the limb draws blood to it; use of muscles draws blood to them. If the hand is laid on a table and the muscles of the hypothenar eminence are alone exercised, the temperature of the ulnar side of the hand rises appreciably relative to the rest of the hand.

The effects of years of disuse can be studied in cases of injury in which joints or tendons become fixed, but in which the arteries and nerves are undamaged. A hand or even a single finger suffering from such disuse remains several degrees colder than its fellow in most circumstances; but it becomes normally warm when general cutaneous vasodilatation occurs, as it does when the body is warmed. As time goes by, the skin of the cold area gradually assumes a deeper and a more cyanotic colour than normal.

Very long disuse of the hand leads, in addition to colour changes, to atrophic changes. In the finger this displays itself by the disappear-

ance of natural wrinkles, such as those marking the flexures of joints, and the skin becomes as a whole smoother than before; this change is probably a relatively direct outcome of disuse. The finger also tends to become tapered, its pulp decreases and the nail becomes over-curved, both in its length and laterally; the bones become rarefied. These changes, which for brevity will be spoken of hereafter collectively as atrophic changes, are also found, though less strikingly, in cases of continuous obstruction or intermittent arrest of blood-flow to the fingers (see page 64). Occurring in association with disuse, they are probably due chiefly to disuse itself, and in part to deficient blood supply. Abrasions or other small injuries naturally heal more slowly on disused fingers than on those to which the circulation is normal.

*Progressive Muscular Atrophy.*—The disease runs a short course; when affected, the hand, though wasted and weakened, is still capable of use. The hand is colder than its fellow in cold rooms, but readily warms and displays full vasodilatation in response to body warming. The skin changes little in colour or texture, and atrophic changes are slight or absent. The changes are consistent with incomplete disuse of short duration and with the diminished circulation which this grade of disuse brings.

*Anterior Poliomyelitis.*—This disease provides examples of conspicuous disuse in the leg, lasting for many years. The limb, and especially the foot, is much colder than normal in cool and usually even in warm rooms; it is colder for most of the day and night. But if the body is heated, the vasomotor reaction is found to be intact, the affected limb becomes as hot as does its fellow; its arteries are found to pulsate fully and capillary pulsation is fully displayed by the skin, indicating that the vessels are free from disease. The skin of the affected limb, from the knee down, and especially of the foot, is deeply coloured and often cyanotic. The discoloration may be diffuse or patchy. The darker patches are often a little swollen or even nodular; sometimes larger nodules break and indolent ulcers form. It is clear that some of these local thickenings are chilblains and that less nodular patches are also responses to excessive cold, corresponding to the condition previously described as erythrocyanosis. The skin is worse in winter than in summer. The foot and leg may be puffy. The condition improves conspicuously after lumbar sympathectomy is performed, for this restores to the skin good circulation; ulcers rapidly heal, swelling and nodular thickenings subside, colour improves or becomes normal.

Atrophic changes are the rule, but, since the patients are young, these are confused by defects in growth.

The changes as a whole are consistent with conspicuous disuse of long duration and with the persistent malnutrition of and injury to the tissues which the reduced circulation brings.

*Syringomyelia.*—This disease provides examples of conspicuous and very long-lasting disuse of the hands. The affected hand is much colder and more deeply coloured than normal in most circumstances of everyday life; but once again, if the body is heated, the hand may display a full vasodilatation, and cyanosis previously present clears away. The hands may be puffy; they are prone to chilblain. Atrophic changes, as previously described in detail, are conspicuous and may be extreme. If we add to the effects of motor paralysis and of diminished circulation the consequences of injuries received owing to defective sensibility, a usual feature of the disease, the changes in the tissues of the hand are adequately explained.

*Hemiplegia.*—A rise of temperature on the affected side, shortly after the accident, is inconstant, and occurring remains unexplained. The ultimate effects of persistent hemiplegia are again those of disuse. The affected limbs are cold but capable of showing full vasodilatation on heating the body. The skin becomes discoloured, the hand and foot may become puffy. Atrophic changes occur.

*General Comment.*—The effects of disuse, whether produced by fixation, by muscular paralysis, or by painful states, are alike. A chief change is atrophy, a gradual process, which in fingers takes the characteristic form already described. An early change is a decline of blood-flow and fall of temperature, which by affecting nutrition contributes to atrophy. The decrease in blood-flow reduces the power of healing. The fall of temperature exposes the skin to injury by cold. The effects of such injury are well recognised. The minute vessels of the skin lose tone and dilate, becoming even telangiectatic. The nutrition of the skin and subcutaneous tissue suffers, the vessels become more permeable, the tissues tend to become oedematous. With further injury inflammatory processes assume prominence, perivascular exudates, nodular infiltrations, necrosis and ulceration appear. The inflammation is of chronic type, modified by the deficient circulation; healing is very slow or fails. This is the range of chief changes. The grade of change is governed by the defect of circulation and its duration, and by the severity and frequence of exposure. When account is taken of injuries brought about through lost sensibility, the changes seen are adequately explained. It is

unnecessary to introduce the idea of a "trophic" factor. The idea of a "vasomotor disturbance" can be entertained only in so far as to signify that disuse brings decreased blood-flow through changed innervation. This makes for very limited employment of the words, especially because it is still unknown if narrowing of the vessels in disuse is mainly a response to the low concentration of local metabolites or to change in vasomotor tone.

## Peripheral Nerve Section

Division of the peripheral nerves of the limbs, mixed in function as they are, may be exemplified by the median or the ulnar nerve; it leads at once to characteristic paralysis of movement and of sensation. The immediate effect on blood-flow to the skin is the result of division of the accompanying sympathetic nerve fibres; this consists as usual of full vasodilatation, indicated by free pulsation, reddening, and warmth of the affected regions of the hand. The reddening soon subsides and so does the temperature, which becomes relatively stable. These are the expected sequels of simple sympathectomy. But soon there is a new departure; from about the 14th to the 21st day the affected parts of the hand, and especially the fingers, become persistently and very decidedly colder than the corresponding parts of the normal limb. The change occurs at about the time when the sensory nerves degenerate and it is to their loss that cooling is attributed. It is difficult to attribute it to anything else; for a limb in which motor and sympathetic innervation is lost (as in anterior poliomyelitis plus surgical sympathectomy) is in general warmer, and not colder, than its fellow. The explanation seems to be as follows. With sensory nerve degeneration, the paths subserving the axon reflex, described on page 90, disappear; and, when this has happened, the vascular reaction of the skin becomes altered to a number of stimuli, some of which ordinarily help to maintain finger temperature. Thus, if a normal finger is placed in ice-cold water for 10 minutes and is withdrawn, in a short time the finger is found to be much hotter than its fellows; this reaction is dependent upon the axon reflex under consideration. The reaction to cold is a very important one in helping to maintain finger temperature and in protecting against the effects of cold; for the response occurs, not only to ice, but to cooling of much less degree (to 18° C. or about 64° F.), cooling such as is very frequently experienced by normal fingers in northern climates. It is noteworthy

too that this reaction is lost, not only to cold, but to all forms of injurious stimulation. Fingers that have lost their sensory nerves do not recover normal warmth until the sensory nerves regenerate, and until the axon reflex has become restored.

Fingers that have been denervated completely, like those that have lost their sympathetic nerve supply only, and unlike those that are cold from disuse, fail to respond when the body is warmed. Thus, not only are they unusually cold, but they are *persistently* cold. This form of coldness is characteristic. The effects of this malnutrition combine with the direct effects of disuse and lead to rapid and conspicuous atrophy of the fingers. In very long standing cases there is some evidence that the arteries of the fingers become thickened and obstructed by disease, the vascular defect thus becoming further exaggerated. It is in these conspicuously atrophic fingers that the skin is often so smooth and so thinned as to be rendered pink by increased transparency. But, in addition, in the affected parts of the hand, the minute vessels lose tone and increase in size, colour deepens, and cyanosis is frequent, as is usual in response to the action of prolonged cold.

The loss of sensibility combines with the gross defect of vascular response to render the fingers prone to injuries of many kinds, and defective in recovery.

### RECOGNITION OF SENSORY NERVE DEGENERATION

The visible flare of the skin, which is produced through an axon reflex in cutaneous nerves belonging to the posterior root system (see page 90), may be used to decide whether these nerves have degenerated or not. The flare can be provoked by a number of different stimuli. A scratch with the point of a needle is a convenient way. The scratch should not draw blood, but merely ruffle the horny layer of the skin. The scratch is followed normally by a line of redness, and outside this on either side appears the flare, which is several centimetres broad and has irregular borders. When the nerves have degenerated, only the red line appears. Another way is to place a small drop of 1 per cent. morphine, 2 per cent. atropine, 5 per cent. acid or alkali, or 0·1 per cent. histamine, on the skin and drive a needle point lightly through it. At the point punctured a red spot and wheal appears and, when the sensory nerves are intact, the flare is seen surrounding it. The test proves useful clinically. For example, in spinal lesions it is useful in distinguishing

between those confined to the cord and those invading and destroying the posterior root ganglia, thus causing the nerve to degenerate; and again in distinguishing between the boundaries of hysterical anaesthesia and of that dependent on peripheral destruction of nerves.

### Peripheral Nerve Irritation. Causalgia

Instances of this disorder are rare. They are for the most part cases in which a peripheral nerve is injured in a wound, the nerve being bruised, or subsequently involved in inflammation, without being divided. Nearly always the median, occasionally the sciatic or another, is the nerve affected.

The first and predominant symptom is pain developing within the first few days or weeks, and increasing in intensity to become very severe. The pain is felt in the distal area, to which the nerve is distributed, and in the common case of the median injury is in the outer parts of the hand. The pain is burning in quality and because of this the malady has been called "causalgia"; the pain is associated with extreme tenderness of the corresponding area. The tenderness is superficial; pain is elicited by the slightest contacts, by friction rather than by firm contacts; it is very easily elicited by warmth, and also by real cold. The pain is always of the same burning quality; it is so easily provoked and so severe that the patient guards the limb closely, holding it flexed and covered, and shrinking at once from all threatened contacts. In severe cases tepid wet bandages, which keep the skin cool, are applied frequently by some patients, who thus obtain relief. The pain is intractable and untreated may continue for twelve months or longer.

When the malady has developed, the affected skin is pink or red, the discoloration being uniform or mottled; the skin is smooth to the degree of glossiness, devoid of wrinkle and hair, and usually wet with sweat. In a number of the cases a few blisters or a more definite herpetic eruption, and sometimes successive eruptions, have been reported. When the temperature of the skin has been noticed, it is reported to have been unusually warm or hot. In long-standing cases, the fingers not only lose their wrinkles but develop all the other signs of disuse atrophy already mentioned.

Closely related to cases of this kind are those in which a finger has been crushed, torn, or otherwise wounded, and in which similar symptoms subsequently develop in this finger and perhaps also in an adjoining finger supplied by the same nerve. Cases of similar

kind in which the finger is persistently cold, rather than warm or hot, probably belong to a distinct category.

*Treatment.*—The patient soon learns how, by keeping the skin cool and guarded, much of the pain can be avoided; but the skin condition is resistant to all forms of local treatment. It is said that periarterial stripping sometimes relieves the pain notably. Successful surgical interference has usually consisted in excision of the lesion with resuturing of the divided nerve. It has been stated emphatically by Tinel that section of the nerve distal to the lesion relieves pain, when section proximal to the lesion has already failed to do so.

### MECHANISM

When a cutaneous nerve, or the posterior nerve root, is cut and the distal end is excited electrically, the corresponding area of skin reddens and becomes hotter than previously. The effect has been called "antidromic" because it has been thought to be conveyed in sensory nerve fibres; it is certainly conveyed in fibres that have their cell stations in the posterior root ganglion. In experiment this vasodilation of skin to which the nerve is distributed has been shown to result from release in it of a histamine-like substance. In man the effects are the same, according to Professor Foerster of Breslau, to whom I am indebted for the additional and interesting observation that the vasodilatation of "antidromic" stimulation is frequently associated with local cutaneous itching, or with burning pain, sensations to be anticipated from the peripheral release of appropriate tissue substances. The released substance is supposed to affect the sensory endings of nerves that overlap and thus provide an intact sensory pathway to the central nervous system. It is difficult to avoid concluding that in causalgia, redness and heat of the skin in the distribution of the nerve is produced as a comparable "antidromic" effect. To explain the origin of the pain upon similar lines accords with the facts, for, as has been stated, pain can be produced by distal stimulation of a peripheral nerve. It is significant too that, in the malady now discussed, the skin is evidently in a state of erythralgia, which on separate grounds (see page 94) has been attributed to a local release of a pain-producing substance. Moreover, if pain arises from the release of a substance locally, then the fact would at once explain why section of the nerve distal to the point of its injury can relieve the pain, for it cuts off the source of nerve irritation. Herpetic eruption, the remaining but inconstant feature of causalgia, is not definitely known to be producible by

deliberate "antidromic" stimulation; but it is not improbable that it can be, for it is well known that herpes zoster is associated with lesions of the posterior root ganglia.

The suggestion is that chronic irritation of a peripheral nerve stimulates nerve fibres of the posterior root system to distal action, thereby releasing substances in the skin which induce a state of local erythralgia, with or without herpes, and that it is from the skin so affected rather than from the nerve at the seat of injury that much or all of the pain arises, the pain impulses being conveyed back along the same nerve, or along a nerve supplying overlapping branches to the common territory.

### LOCAL AND REFERRED TENDERNESS

Further observation may quite firmly establish what is already a sound working rule. Tenderness arising out of local disturbance in the skin and that which is merely referred to the periphery from a nerve lesion, such as neuritis, can be distinguished by differences in reaction. In the local disturbance the affected skin is abnormally sensitive to heat, and friction produces pain, not only at the time, but for long afterwards; neither of these phenomena appear when tenderness is merely referred.

## Bedsores

To understand the common bedsore it is necessary to know how ischaemia affects the skin and how nature protects against injury. When the flow of blood to a tissue is arrested for a certain time the tissue dies; as stated in Chapter II, ischaemia of 6 or 12 hours' duration damages skin to the extent of causing it to wheal subsequently, ischaemia for a continuous period of 24 or 48 hours, or for frequent shorter periods, results in death and necrosis of the skin. It is unnecessary that a main artery should be stopped for the skin to lose its blood supply; direct pressure is more efficacious. The blood supply is lost to the soles of the feet when standing still on them, is lost to the skin of the buttocks while sitting; but no one stands or sits quite still for periods of hours. Changes of posture, small or considerable, remove the pressure, and the affected skin at once displays reactive hyperaemia. Blood now floods the skin in unusual quantity and rapidly revives the tissues. The effects of ischaemic periods are not additive, if these are separated by adequate intervals of relief; a fully adequate interval is half the length of the ischaemic period or a little longer. The amount of pressure required to arrest blood-flow to

skin is apt to be overestimated; when arterial pressure is normal and there is no venous stasis, 50 or 60 mm. Hg is enough. Such a pressure is felt when a subject lies supine in bed, by most of the skin pressed upon, and especially by the scapular and sacral skin, and by that of the heels. It is usual for the supply of blood to be lost to areas of skin during sleep; but the skin is rarely hurt in this way, because it is not normal to sleep lying like a log; posture is changed many times in a night. But if a patient is bedridden, is under avertin anaesthesia, is paralysed, or is too weak from illness or age to move sufficiently and often, then damage to the skin results. This damage at first takes the form of redness and soreness with a little swelling, later of simple or haemorrhagic blisters, later still of necrosis and ulceration. The different grades appear according to the length of the ischaemic period and the number of repetitions. Ulceration of the skin following a single period of arrest is illustrated by splint pressure, already referred to on page 37. Where ischaemia is repeated, damage will be carried over from one period to the next, if the interval is insufficient to complete the reactive hyperaemia, or if other changes in the tissues established during the first period are not quickly reversed. Once injury is established, the chances of recovery from it lessen, according to the degree in which the circulation fails subsequently to be restored and to be maintained.

It is easy to see how necrosis develops from persistent or intermittent pressure, and why an ulcer once formed is usually intractable. If the skin is anaesthetic, there will be no discomfort to suggest movement. If the patient is enfeebled by disease or by age, movement will be infrequent or impossible, and vascular reaction and resistance to infection impaired. These factors fully account for bedsores and their intractability.

*Management.*—In guarding against bedsores, and in treating them when formed, the essential measure is to change the site of pressure. Ideally, pressure should not be maintained continuously on a given area of skin for more than a half to one hour, and the same area should not be called upon again to support weight until an equal period of time has elapsed. To distribute pressure by means of pneumatic or water mattresses, which mould themselves to the body, is less certainly effective than frequent changes of posture. When the patient is moved the skin should be cleansed and dried with spirit, and dusted with a simple, mildly antiseptic, absorbent powder, such as zinc oxide, boracic acid, and starch.

# INDEX

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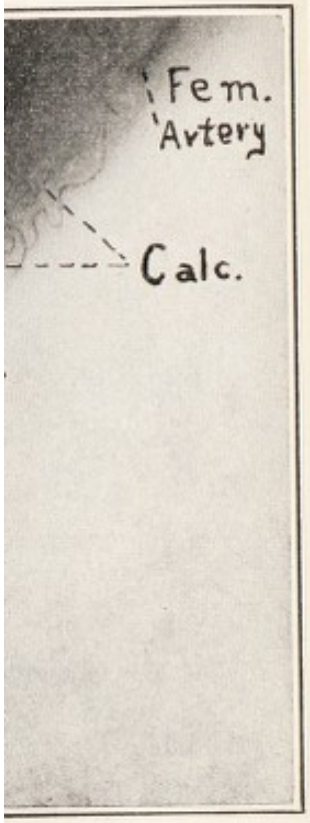


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sclerosis but is occasionally important in Buerger's disease. Since the reflex is mediated by the sympathetic nervous system, the vasoconstriction can be tested by measuring the surface temperature of the limb before and after anesthesia or other inhibition of the sympathetic tone. If much vasoconstriction is thus demonstrated, some improvement can be expected from surgical attack on the sympathetic nerves. The occasions when such operative treatment is necessary in these organic diseases are, however, infrequent, and this matter will therefore not be discussed further in this paper.

In arterial insufficiency from any cause, the closure of the major arteries makes it necessary for the blood to flow through the smaller communicating vessels, which we term the collaterals (Figs. 6 and 7). As these collaterals increase in size, the aggregate of their openings becomes equal to, or even exceeds, the caliber of the blocked arteries. However, this does not ensure the delivery of an equivalent amount of blood, since the flow in small vessels is not directly proportionate to their size, but rather to the square of their diameters. In these small arteries the pulse wave is damped and the blood oozes through under a lowered pressure (Fig. 9). In effect, then, while it is desirable and necessary to increase the size of the collaterals, in no case can these vessels transmit the quantity of blood passing through the previously normal arteries.

At present, at least, we have no means of restoring the major arteries involved in arteriosclerosis or Buerger's disease. It is thus obvious that the patient can never expect a return of perfect function in the affected limb, as the new non-pulsatile circulation cannot support an active metabolism.

This thesis acquires verification when we examine a group of animals whose circulation is normally similar to this abnormal flow in the human subject. Reference is made to sloths, anteaters and lemurs. In these animals, the main artery to the limb breaks up into a great group of fine vessels at the very root of the extremity (Fig. 8). This vascular arrangement can be correlated with the sluggish muscular habits of these animals.[4]

## DIAGNOSIS

Clinically, before one can accurately diagnose the specific nature of the background disease he must establish the presence of arterial insufficiency. As a basis for judging the adequacy of the cir-

nutrition of the peripheral nerves ensures normal sensation; (4) there is efficient defense against in-

FIGURE 7. *Collateral Circulation after Organic Obstruction of the Major Arteries.*

*This sketch is drawn in accordance with arteriograms. Compare with Figure 8.*

FIGURE 8. *Normal*

*The major lim great number of by corresponding*

jury of various types; (5) the skin color is a characteristic pink; (6) the pulsations are adequate.

*Muscle action.* In high-grade obstruction, even occasional muscular action is weak. The muscles are apt to be atrophied, partly from malnutrition, partly from disuse. If the obstruction is moderate, a single muscular contraction may be powerful, but continued use is impossible. This occasions the syndrome called "intermittent claudication," a symptom of arterial insufficiency from any cause. Typically, after walking one or two blocks the patient is seized by a severe cramping pain in the calf or sole of the foot. He recovers on resting a few minutes, and again can walk the same distance as before, again to be seized by the cramp, and again recovering after his rest. Moreover, the

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