Intermittent claudication and allied syndromes due to angiosclerosis of the extremities / by J. Ramsay Hunt.

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Hunt, James Ramsay, 1872-1937.
Royal College of Surgeons of England

Publication/Creation

[New York] : William Wood, [1905]

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INTERMITTENT CLAUDICATION AND ALLIED SYNDROMES DUE TO ANGIOSCLEROSIS OF THE EXTREMITIES.*

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THE sclerosis of the arteries and veins of the extremities and the abnormal vasomotor states sometimes associated, form a subject which well illustrates the intimate relationship of neurology and the broad field of internal medicine, some of its phases even transporting us into the domain of surgery.

While the syndromes resulting from angiosclerosis and angioneurosis of the extremities are many and varied, the underlying pathological changes are those so familiar to us all—the arteriosclerosis, the obliterating endarteritis, and the senile calcification of the arterial tree.

Among the more or less clearly defined clinical pictures dependent upon vascular disease of the extremities may be mentioned: (1) The intermittent claudication (Charcot); (2) some forms of erythromelalgia (Weir Mitchell); (3) some types of symmetrical gangrene (Raynaud's disease); (4) the spontaneous gangrene of the extremities; (5)

*Read at a meeting of the New York Society of Internal Medicine, January 18, 1905.

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the arteriosclerotic neuritis from obliteration of the vasonervorum. For the production of these various clinical types special factors are essential, of which the more important are: The degree and nature of the vascular process; the localization and predominance in certain vascular areas; the neurotic element or the accompanying vasomotor neurosis.

Great importance must be attached to the associated vasomotor instability as a determining and often dominating factor in these cases. A simple obliteration of an artery would have as a result the so-called spontaneous gangrene. If such obliteration were symmetrically distributed, a picture similar to Raynaud's disease would follow. If, however, to the arterial disease a vasomotor instability is superadded, with a tendency to vasoconstriction or vasodilation, the symptoms of intermittent claudication or erythromelalgia would result. The so-called arteriosclerotic neuritis, a very rare manifestation, is merely an unusual predominance of the process in the vessels of the nerve trunks, inducing widespread degeneration of the nerve fibers and the symptoms of an irregular, progressive neuritis.

The kindred nature of these various clinical types receives abundant confirmation in a similar pathological anatomy, in their occasional combination, and a somewhat rarer alternation, *i. e.* the disappearance of one syndrome and its replacement by another, and, most important of all, in a very striking tendency to a common termination, *the dry gangrene.*

In fact, the important practical lesson taught by a consideration of this subject is to regard all these various manifestations in the light of a distinct warning and forerunner of this unhappy termination.

As the intermittent claudication has received but scant attention in this country, and even less in Great Britain, and as the subject is one of great practical importance, a somewhat detailed description is ventured upon, including four personal observations.

Historical.—For the name and the first description of this affection we are indebted to Charcot. His first publication appeared in 1858, and described in a most graphic manner a complex of symptoms, which he christened Claudication Intermittente; use was also made of the term Paralysie Douloureuse Intermittente. The autopsy revealed an aneurysm of the right iliac artery, the vessel having been occluded by a thrombus for a short distance below. Charcot also remarked the resemblance between this group of cases and a similar condition observed in horses by veterinarians. (Bouley in 1831, Rademacher in 1838, Böther in 1839, and others.) Animals suffering from this disease, which we term springhalt, after covering a short distance, are seized with sudden weakness and stiffness in the hind legs, which become absolutely rigid. After a short rest the spasm relaxes and the function of the extremities is restored. In these animals arteriosclerosis and calcification at the bifurcation of the aorta and in the iliac arteries was found interfering with the free circulation of the blood in the parts below.

Further contributions from the pen of Charcot appeared in 1886, 1887, 1891, and 1892, crystallizing and enlarging the clinical picture and adding materially to our knowledge of the etiology. Other communications from French observers appeared in 1873 by Sabourin; 1890, Delaunay; 1892, Magrez; 1894, Levet, and Bourgeois, in 1897.

Strangely enough, this very striking, and, as has been demonstrated by subsequent experience, not uncommon affection, received but little attention in other countries. It was not until 1892 that Elzholz, and in 1895 that Goldflam, recorded cases in Germany. In 1898 however, Erb, in a masterly exposition, comprising a complete analysis of the literature and a most able discussion of the whole subject in all its phases, inaugurated a new era for the intermittent claudication, and insured it a permanent and important niche in the pantheon of disease. While Charcot attached the greater importance to disease of the larger arterial trunks, as did also the veterinarians, Erb demonstrated in the clearest manner possible its dependence in the vast majority of cases upon sclerosis and obliteration of the smaller arterial branches and terminals.

Since Erb's publication many monographs have appeared in German literature showing the frequency and growing importance of the affection. Among these are Higier, in 1900, recording 23 cases; Hagelstam of Helsingfors, in 1901, recording 7 cases; Idelsohn, in 1903, recording 14 cases. In this country the first report of a case was made by Putnam of Boston in 1901. Subsequent case reports followed by Dana, in 1902; Riesman, 1902; Walton and Paul, 1902; Levy, 1902; Patek, 1904, and Burr, 1904. In the literature of Great Britain no contribution to this subject could be found. The natural inference is that in this country and Great Britain either the malady is rare, or it has not yet attained widespread recognition and importance.

Several names, more or less descriptive in nature, have been suggested to supplant the original, as suggested by Charcot. Intermittent muscular paralysis (Grossman); myasthenia angiosclerotica (Hi-

gier); angina crusis (Walton and Paul); dysbasia angiosclerotica intermittens (Erb). The intermittent claudication seems, however, to have been firmly fixed by usage, and at present still finds most favor, although the dysbasia angiosclerotica, as proposed by Erb, is more descriptive and has a more general application.

Etiology.—The disease is much more frequent in men than in women. While the etiology is still obscure and somewhat speculative, a number of causative factors, both general and special, have come to be recognized. All those general causes favoring the development of angiosclerotic changes naturally assume a prominent place—advanced life, alcoholism, syphilis, gout, and nicotinism. Diabetes was present in a certain number of cases. Among the special causes having a more or less local influence may be mentioned exposure to excessive cold, flat foot (Idelsohn), arterial compression by a truss, and aneurism (cases of Charcot, Barth, Mannaury). Some observers, notably Oppenheim, attach great importance to the neurotic temperament, the so-called neuropathic diathesis. Others again would seek a cause in the hereditary or congenital insufficiency of the cardiovascular apparatus and its controlling nervous mechanism. Erb, who has had unusual opportunities of observation, recording 57 cases in all, attaches the greatest importance to excessive smoking and exposure to excessive cold as direct excitants of the vasomotor irritability and spasm.

Pathology.—Our knowledge of the pathological anatomy of this affection has been derived very largely from the study of amputated extremities. (Charcot, Erb, Marinesco, Goldflam, Laveran, Panas, Dutil and Lamy.) Only a meager number of

autopsies have been recorded. (Cases of Charcot, Elzholz, Magrez, and, recently, Erb.) The findings have been practically uniform, and may be summed up in the one word, angiosclerosis. All forms of arterial changes are met with, the obliterating endarteritis of Friedländer, the senile calcification, the arteriosclerosis of Gull and Sutton, the periarteritis nodosa of Kussmaul and Maier, with and without obliteration and thrombosis, but all tending to produce a common evil, a diminution in the caliber of the vessel, with consequent circulatory embarrassment. The veins not infrequently show analogous changes, but in a lesser degree, the phlebosclerosis and endophlebitis. These changes are all more marked in the smaller arterial branches and terminal vessels of the extremities, and they frequently show a symmetrical distribution on the two sides. It must be emphasized, however, that arteritis, aneurism, and thrombosis of the larger trunks have been found in quite typical cases.

In the more advanced stage of the obliterating process secondary changes of a trophic nature frequently occur. The muscles may become atrophied and show extensive areas of degeneration and sclerosis (Marinesco case). The nerves may present more or less diffuse degeneration from occlusion of the vasonervorum, this in turn leading to degenerative muscular atrophy (cases of Joffroy and Achard, Dutil and Lamy), or a portion of the extremity may be thrown off by the process of dry gangrene. It is this termination which unites these cases with the symmetrical, senile, and spontaneous forms of gangrene.

These are the structural changes found in the class of cases under discussion. But such alterations are very commonly present where not a trace

of intermittent claudication has been observed, so that another and most important factor deserves consideration, of a purely functional nature, and may be termed the vasomotor element. The variations in arterial tone, the alternate contraction and relaxation of the vessel wall under the influence of various internal and external stimuli is well known. The flushing of the face and sudden pallor, the coldness of the hands and feet under the influence of a sudden emotion is a common experience. It is this vasomotor irritability, this spasm of the arteries of the extremities which contributes so largely to the production of this peculiar syndrome, and accounts, in part, for the sudden restoration of function after a paroxysm. Some observers even (notably Oppenheim) suggest a purely functional form of the disease, or one in which the structural alterations of the vessels play an entirely subordinate rôle.

While the vasomotor element deserves all the importance accorded it, it should not be overlooked that a mechanical impediment to the circulation alone, perhaps sufficing during rest, but insufficient for the increased demands of activity, may of itself cause symptoms of claudication.

It is generally admitted that the symptoms are ischemic in origin, the manifestation of a temporary anemia of the muscles, nerve endings, and other structures. In this connection reference is often made to the experiments of Stenson and others who, by compression of the aorta, produced paralysis and anesthesia in the parts below.

The exact relation existing between the organic changes in the vessels and the vasomotor irritability is still speculative. Whether the tendency of the vessel wall to vasomotor spasm is the direct consequence of arterial disease from implication of the

intrinsic nervous mechanism, or merely an independent vasomotor neurosis superadded to the other condition. Three factors must be recognized in the production of the claudication ischemia. One, constant and organic in nature, diminishing the caliber of the vessel, and two factors, inconstant and functional, the tendency to vasomotor spasm and the increased demand upon the circulation of the part during activity.

Symptomatology.—After a consideration of the various clinical features and peculiarities of dysbasia angiosclerotica, that which is perhaps most striking and most characteristic is the intermittent character of the symptoms. An absence of pulsation in the pedal arteries and evidences of circulatory disturbances in the lower extremities are of scarcely less importance. The patient suffering from this affection, while at rest, either in the sitting or recumbent position, is unconscious of any disability; the extremities are freely movable and are free from any abnormal sensory manifestations. He feels that he could rise and walk indefinitely. In fact, it is usually possible for him to rise and start off at the usual pace without any ill effects, but gradually, in from five to ten, twenty or thirty minutes, or even longer, varying with the severity of the case, the symptoms of claudication appear, and increase in severity with the effort to proceed. At last further progress becomes distressing, or even quite impossible, and an enforced rest results. Gradually the symptoms of distress grow less, the legs resume their natural feeling and power, and in a very few minutes the pedestrian is able to pursue his way, only to halt again as the time limit of his toleration is reached.

The symptoms induced during the period of activity, while differing somewhat in individual cases,

are strikingly similar. Usually the first indications of beginning trouble as the paroxysm approaches is a sense of weight and heaviness, a feeling of fatigue and pain in the leg. This is soon followed by coldness and pricking sensations, and the etremity becomes numb and weak. If activity is maintained the muscles stiffen and become painfully cramped, and the characteristic limping results, or locomotion may even become quite impossible from the intense, marble-like rigidity which follows (abasia). Thus weakness, painful cramps, paresthesia, and stiffness may be regarded as the essential accompaniments of the paroxysm, although other and often very curious sensations are sometimes graphically described by the patient, as: "A sensation of water rushing through the leg"; "as if the leg were being sundered from the body"; "a bursting feeling in the calf"; "as though I dragged a cannon shot about in my leg." Sometimes scalding or burning sensations are complained of, or the hot sensation of very intense cold.

If the feet and legs are inspected after a prolonged effort, or even in moré severe cases in the dependent position alone, evidences of circulatory disturbance are very obvious. They are congested and swollen, with evidences of mottling and cyanosis, and are palpably colder than normal. In cases with marked vasomotor irritability the feet and toes may even assume a "dead" or waxy hue from the high degree of arterial spasm. In the more advanced cases the intense burning pain in the toes, which may be a glossy red or cold and purple, indicate the beginning of total arterial obliteration, with consequent gangrene.

Objective changes in the pulsation of the pedal arteries are of great importance in these cases. In the vast majority of cases the posterior tibial and

dorsalis pedis arteries, one or both, are either quite pulseless or weaker than normal. To determine their constancy under other conditions Erb examined 750 cases, at all ages and suffering from various ailments, with a positive result of 99 per cent. With so small a proportion of negative cases, some of which may be accounted for on the ground of anomalies of distribution, the foot pulses may be practically regarded as fixed and constant, and their absence as evidence of a pathological state. Goldflam and Walton and Paul have carried out similar investigations on a smaller series of cases (200), with practically the same results (Goldflam, 99 per cent., Walton and Paul, 96 per cent.).

Changes are not infrequently met with in the superficial veins of the extremities, minute dilatations and varicosities, and an increase in the number of visible venules. In some cases thrombosis of the veins has been observed.

Wasting and atrophy of the muscles occasionally occur. This is often only the atrophy of inactivity, but true muscular degeneration and sclerosis may result from obliteration of the vessels. While the symptoms are often confined to a single extremity, in the majority of instances both limbs are involved, but rarely in the same degree. No adequate explanation has been offered for the greater frequency of the affection on the left side.

While the typical picture of intermittent claudication is as outlined above, the paroxysm during activity and complete freedom during rest, certain exceptions may be mentioned. In the more severe cases, in which the obliterating process is well advanced, or the patient has abused the "time limit" for a considerable period, sensory symptoms and weakness may be present more or less constantly,

and may give rise to errors in diagnosis. In this group of cases sharp lancinating pains shoot through the legs and the calves may be constantly stiff, swollen, and tender.

As might be inferred from the general nature of the underlying process, the symptoms of intermittent claudication may occur in other arterial distributions of the body. One or both arms may present rarely the characteristic symptoms of the disease, usually associated with objective changes in the pulsation of the arteries at the wrist. Cases presenting this distribution have been recorded by Nothnagel, Hagelstam, Dana, Burr, and Massaut. An unusual localization was observed in a case recorded by Brissaud. A severe burning pain would regularly appear on the anterolateral aspect of the left thigh after walking twenty minutes, always disappearing after a short rest. Autopsy revealed an exquisite arteriosclerosis of the branches of the femoral artery in Scarpa's triangle. In two cases, in addition to the other symptoms, the pudendal artery was found occluded, and had produced during life rectal disturbances and impotence in the case of Luxemburg, and incontinence of urine in Burr's case.

The syndrome of intermittent claudication, viz., function perfect at rest, but impaired during activity, is by no means confined to the vessels of the extremities. It has long been thought that the paroxysms of angina pectoris were referable to a similar state of the coronary arteries. In recent years cases with autopsy are recorded showing the occurrence of this syndrome in the brain, kidney, retina (Wagenmann), and intestines (Schnitzler and Ortner).

Indeed, so general would its application seem at present that *dyspragia intermittens* (Ortner) has

been proposed as a generic term, indicating the intermittent disturbance of function.

Perhaps the most circumscribed type of the affection is found in an isolated muscular localization, associated with recurring cramps and pains in certain definite areas. The frequency of these muscular spasms in old people, and their dependence upon arterial disease, has been emphasized by Walton and Paul. Thoma and Erb likewise refer to many of the so-called rheumatic pains of middle life and their possible dependence upon vascular conditions.

In discussing the pathological anatomy mention was made of secondary nerve degenerations, arteriosclerotic in origin, due to obliteration of the vasonervorum. In rare instances, notably in the cases of Schlesinger, Joffroy and Achard, the localization of the obliterating process in the neural arteries was so extreme, and the resulting degeneration of nerve tissue so great as to give a distinctly neuritic coloring to the clinical picture. These cases presented the picture of a slowly progressive multiple neuritis, of very irregular course, with occasional acute exacerbations and associated with paralyses, atrophies, objective sensory disturbances and electrical changes.

Before leaving the subject of symptomatology reference must be made to the rare occurrence of certain atypical, or even paradoxical forms described by Erb. In these weakness and fatigue in the legs may occur, without the characteristic intermission or even the necessity of resting, or activity, instead of aggravating, may cause an amelioration of the symptoms.

Diagnosis.—The diagnosis in well-marked and typical cases is easy. The nature of the motor and sensory manifestations, their intermittent character

dependent upon activity and rest; the accompanying evidences of circulatory disturbances and absence or feebleness of pulsation in the pedal arteries should render the recognition of this affection comparatively easy. Early and atypical cases may present greater difficulties and suggest disease of the central or peripheral nervous systems, or even some purely local condition, such as flat foot, tarsalgia, meta tarsalgia, and podalgias of gouty origin. It is needless to insist that in every case a thorough examination of the nervous system is absolutely essential. It should also be an established routine practice in all nervous cases of doubtful nature involving the extremities to examine carefully the pedal arteries and note the condition of the circulation in these parts.

Prognosis.—The prognosis as regards cure is unfavorable. Many cases, under appropriate treatment, have shown considerable improvement, while others resist every known method, and continue their downward course, terminating in gangrene. Those cases in which the vasomotor element is well marked, and those due to obliterating endarteritis of syphilitic origin, are most amenable to therapy. In general, it may be said that, under appropriate measures much may be done towards arresting the process and warding off indefinitely the threatened gangrene.

Treatment.—One of the most important elements in treatment is rest. These patients should never be allowed to transgress their time limit, *i.e.* the period of time elapsing while walking before the appearance of the claudication. They should, as Charcot advised, walk watch in hand, with slow, measured step, and frequent intervals of rest.

From time to time prolonged periods of rest in bed are also advisable, as allowing a better collateral circulation to be established in areas poorly nour-

ished, and also favoring the establishment of a more stable vasomotor tone, so important an element in these cases.

Of the internal remedies, potassium iodide in moderate doses finds great favor, as in other manifestations of arteriosclerosis. Where syphilis is suspected it should naturally be pushed to the point of toleration. Also cardiac tonics, especially those with little action on the vasomotor system, such as strophanthus. Drugs bearing a special influence on vasoconstriction, such as digitalis and ergot, should be studiously avoided. Nitroglycerine has been recommended in these cases for its effect on the arterial walls, but without very brilliant results. Warm foot baths and galvanic foot baths are highly recommended by Erb for their vasodilator effect, and should be given a trial in all cases. On the contrary, very hot baths and naturally cold baths should never be employed.

Idelsohn, in his series of 22 cases, found an associated flat foot in eight cases. He is inclined to regard this as an etiological factor of importance and recommends its careful correction. Its exact relation to the affection is not clear, but his suggestion that the circulation in the foot may be impeded by sinking of the arch, or that possibly by stretching of the nerves a state of reflex vasomotor irritability is induced, well merits consideration.

Report of Personal Observations.—Case I. (Referred by Dr. Henry Russell.) The patient is a widow, 58 years of age, of unusually strong constitution and hale and hearty appearance. She has never borne any children. Her parentage is English, and on both sides of her family for several generations gout has existed in its most exquisite form. The patient herself has never suffered from this affection. So far as her memory goes no member of

her family has suffered from any of the graver mental or nervous diseases. It is interesting to note in this connection that both the patient and a brother have been sleepwalkers from an early age. Two sons of this brother were also somnambulistic. This tendency was never outgrown in the patient's case, and even up to the present time a change of surroundings or any unusual stress or worry is sufficient to excite a night ramble. In her younger days these somnambulistic episodes were frequent and of some gravity. It was not uncommon to find her in this state walking the streets. Objects were frequently hidden, letters written and other complicated acts performed. She has always enjoyed exceptional health, and her mode of life has been one of unusual care and moderation. In her diet she has been rather addicted to red meats, and for many years she has taken daily six large cups of strong tea, of alcoholic beverages only on the rarest occasions. During the latter years of her life she has had many trials and anxieties, culminating about five years ago in a distressing episode, at which period her present malady began. She had always been fond of walking, took a very quick step, but rarely exceeded from three to five miles a day. Until the beginning of her present trouble she had never suffered with cold hands or cold feet or other evidences of vasomotor instability. In 1885 she abandoned the use of the circular garter.

In the winter of 1900, after walking from a quarter to a half mile, she first noticed that a slight stiffness and numbness would develop in the calf of the left leg. She frequently rested on this account, and found that after a few minutes it passed away, so that it became her habit to break a journey or a shopping expedition by these short waits. These symptoms gradually grew worse, so that in time it

became necessary to shorten the distance, and for any longer effort to increase the period of rest. About the same time the right leg presented similar symptoms, but of a trifling nature and slight in comparison. It sometimes happened that when no good opportunity was offered for a rest she would try to brave it out and push on, but to her consternation the left leg would become painfully cramped, absolutely stiff and immovable, and she would have to remain standing where she was. Several times while in this condition she simply collapsed helplessly on the sidewalk, but after a short rest was able to proceed without difficulty. Her condition continued to grow worse, so that eventually only a very short distance could be traversed (from five to ten minutes) before the appearance of painful cramp, numbness, and rigidity in the affected leg. At times very sharp lancinating pains would shoot through the legs, especially at night. The feet were almost constantly cold, and in a dependent position would assume a bluish, mottled hue. In December, 1902, she slipped on the ice and fractured the external malleolus of the left leg, and while under treatment for this passed through a severe attack of pleuropneumonia. The convalescence was complicated by thrombosis of the veins in the lower extremities, first in the right leg, a day or two later in the left, preceded by severe pain and the development of tender lumps along the inner side of the thighs, and followed by a considerable edema of both legs. After recovery from this illness, in May, 1903, the legs were much worse, the feet and ankles would become puffed and edematous, and she complained of almost constant pains, numb feelings, and cramps in both legs, but much more severe on the left side. It was at this time that she first came under my care. and I am

much indebted to her physician, Dr. Henry Russell, for his observation of the case previous to this.

Status præsens, May, 1903.—Complains of constant numbness and aching pain in the left calf, especially along the perineal side. In this region there is a painful throbbing sensation, "as if a boil were about to burst." She also experiences at times a sense of great weight and heaviness in the calf, as if a cannon-ball were embedded in the muscle. Sharp pains dart through the legs, often of such severity as to cause a scream, and the muscles of the leg become knotted in painful cramps. The feet and ankles are swollen and cold, and on standing they assume an angry red discoloration. After walking about three minutes the steps become slow and tottering, the muscles of the calf stiffen, the outer side of the leg, sole of foot, and toes become numb and dead, and it is only with the greatest effort, associated with dyspnea, that the left leg can be moved any further. The same disturbance, but much milder, is present in the right leg. The knee jerks and ankle jerks are present on the two sides and exaggerated. Deep pressure on the calves causes the same pain as is observed in neuritic affections. The examination of the nervous system, however, is entirely negative. Sensation over the lower extremities is normal. No pes planus. The heart is free from murmurs and regular, the first sound of good muscular tone, and the second aortic moderately accentuated. The radial pulse is exceptionally small, in view of the large physique and florid appearance of the patient, but the walls are without a trace of thickening. The urine is free from albumin and sugar.

The condition noted in the pedal arteries is as follows: Right posterior tibial, good pulsation; right dorsalis pedis, not palpable; left posterior tib-

ial, feeble pulsation; left dorsalis pedis, good pulsation. No difference in the pulsation of the femoral and popliteal arteries could be noted on the two sides. After walking five minutes no appreciable change was noted in the character of the pulsation in these vessels. No muscular atrophy and no muscle quivering (myokymia) was observed in either leg, and the calves measured the same on the two sides. The thighs escape entirely, and the complaint is of the leg below the knees. At times, however, when seated in a hard straight-back chair, a painful rigidity develops in the gluteal muscles, especially of the left side, analogous to that described in the legs, and disappearing quickly in the recumbent posture. At times, while seated at meals and not obeying the first warnings of the gluteal spasm, the buttocks become so painfully stiff that she is unable to rise alone and has to be literally pulled out of the chair. This localization of the painful spasm never occurs in walking or in standing, nor in the recumbent position, but only while sitting.

She was removed to a sanatorium and placed at absolute rest in bed for one month. At the end of the first week nearly all the distressing subjective symptoms had disappeared, the swelling of the feet and legs had subsided, but the dorsal aspect of the toes was still reddish, paling on pressure.

A very slight thread-like pulsation became apparent in the right dorsalis pedis artery. Treatment consisted essentially of sodium iodide in ascending doses, nitroglycerin, alkaline waters, anti-gout diet and daily hot salt sitz baths at 100° F. At the end of a month the beneficial results of these measures became apparent. She could walk from ten to fifteen minutes with only a moderate sense of stiffness and numbness; if, however, the exercise was prolonged beyond this time painful cramps and rigidity

would develop. After an additional two weeks she was allowed to return home where practically the same measures were instituted. She was advised never to transgress her time limit of fifteen minutes, to take frequent rests and for at least one-half of the day to maintain the recumbent posture. For six weeks after leaving the sanatorium she complained bitterly of painful cramps and stiffness in the gluteal region while sitting, but these gradually disappeared. The patient has been under continuous observation up to the present date. Her condition varies from time to time, any exacerbation can usually be traced to prolonged over-exertion and inattention to the warning pains and numbness. If the law of fifteen minutes is disobeyed, the legs ache and feel almost continuously numb, the ankles puff up and the feet become cold and cyanotic. She dreads the cold of winter and always rests with the hot water bag at the feet. Quite recently her condition was aggravated by the worry and exertion attending a change of residence. The old symptoms returned in all their intensity, and in addition, there appeared two swollen areas, very tender to the touch, one just above the internal malleolus and another over the upper third of the tibialis anticus muscle. Over these the skin was quite hyperesthetic. The swelling in the tibialis anticus muscle was tender only on direct pressure, movement of the muscle as in flexing the foot was unattended by pain. These tender swellings as well as the distressing subjective symptoms disappeared after an enforced rest.

December 12, 1904. Pulsation in the pedal arteries is as follows: Right dorsalis pedis, very faint and thread like; right posterior tibial, strong pulsation; left dorsalis pedis, full, strong pulsation; left, posterior tibial, fair pulsation but distinctly weaker and of smaller volume than the right.

Good pulsation in both femorals and popliteals. Repeated examination of the urine was negative. No change in the cardiac condition, the radial pulse is exceptionally small and soft. Dyspnea on exertion.

April 1, 1905. During the past three months patient has received treatment as follows: Moderate doses of the iodides and bromides, tincture of strophanthus, galvanism to the lower extremities, and hot salt foot-baths, under this regime some improvement has been effected, she sits with greater ease and walks with more freedom, but I cannot say that her time-limit (15 minutes), has been materially lengthened.

Case II.—Referred by Dr. C. E. Campbell. The patient is a Hebrew, forty-two years of age, a storekeeper by occupation. Family history is negative. The man himself is of a quick, nervous temperament, but had enjoyed excellent health up to the onset of his "leg trouble," in the winter of 1902. He is careful in his habits, very moderate in the use of alcohol and tobacco, and denies syphilitic infection. The urine was examined repeatedly and was always normal.

Early in the winter of 1902 he observed that the sole of the left foot and the left leg would become numb during a long walk. The muscles of the calf would stiffen and the foot would become swollen. At such times it was his custom to sit down, unlace his shoe and rub the foot, all trace of the trouble disappearing in about five minutes. He would then resume his journey, repeating this procedure as often as necessary. If, however, he continued without resting, the leg would become more and more stiff, and the numbness extended up the thigh. A very unpleasant sensation would then develop, as if the whole left leg were being thrown off or separated

from the body. Paresthenia and stiffness would also develop after long-continued standing in his shop, but never so severe as in walking. Quick relief was always obtained in the sitting or recumbent position. The right leg was unaffected. On awakening in the morning, the leg always felt perfectly well. He feels the cold in both feet, but the left foot is always the colder, both subjectively and to the feel. The left foot sometimes swells about the ankle, but he has never remarked any bluish discoloration. It is possible for him to walk from one-quarter to one-half mile with comparative comfort.

Status præsens.—July 31, 1903. The lower extremities are normal on inspection, no atrophy, no fibrillation, no discoloration. There is no appreciable difference in the temperature of the two sides. The knee jerks and the Achilles jerks are exaggerated and equal on the two sides. No flat foot.

There is no evidence of any involvement of the central or peripheral nervous systems. Heart action is good, no murmurs; the aortic sound is sharp. The radials are not thickened.

Pedal Arteries.—Dorsalis pedis: a full pulsation on both sides; posterior tibials; good pulsation in both posterior tibials, but the right is appreciably weaker than the left; popliteals, pulsate equally on the two sides; femorals, also pulsate well, rather fuller on the left side; the difference, however, is not marked.

The patient was cautioned about over-exertion, and the same measures taken as in CASE I. He returned to his home in Corning, N. Y., where he continued this treatment for several months, with some amelioration in the symptoms, and has not come under my personal observation since. In response to an inquiry dated December, 1904, I received the following reply: "The trouble is still

entirely in the left leg, especially the sole of the foot and the calf of the leg. I can walk about half a mile without much annoyance; then the prickling, electricity-sensation, pain and stiffness develops, especially in the calf of the leg. Standing for a time also brings it on. The foot does not swell or change color. If I rest about five minutes, all the symptoms disappear. There is no material difference in my condition summer or winter. My physician advised me to wear a plate in the left shoe, which seemed to giv me some relief but my condition remains about the same."

CASE III.—(From Prof. Dana's Neurological Clinic, Cornell University Medical College.) A bartender aged sixty years, no venereal history, excessive use of alcohol. For the past twenty years averages ten to twenty drinks a day, in addition to this frequent sprees lasting one or two weeks. For twenty years has smoked ten cigars a day, also chews tobacco (half an ounce in a day). His occupation behind the bar and in damp cellars, has entailed prolonged exposure of the feet to cold and wet for many years.

The first indication of his present trouble was a numbness and coldness appearing in the right great toe about two hours after beginning work in the morning. This was first noted about five years ago and has persisted ever since, gradually involving the whole foot to the ankle. The feeling is one of stiffness and weakness in the right foot and toes, associated with stinging pains, numbness, and prickling sensations. The foot also feels cold and is cold to the touch. In the morning on awaking there is not a vestige of any disturbance, but after walking one-quarter to one-half a mile the stiffness and numbness are induced, varying in intensity with the temperature, and season of the year. In winter all his

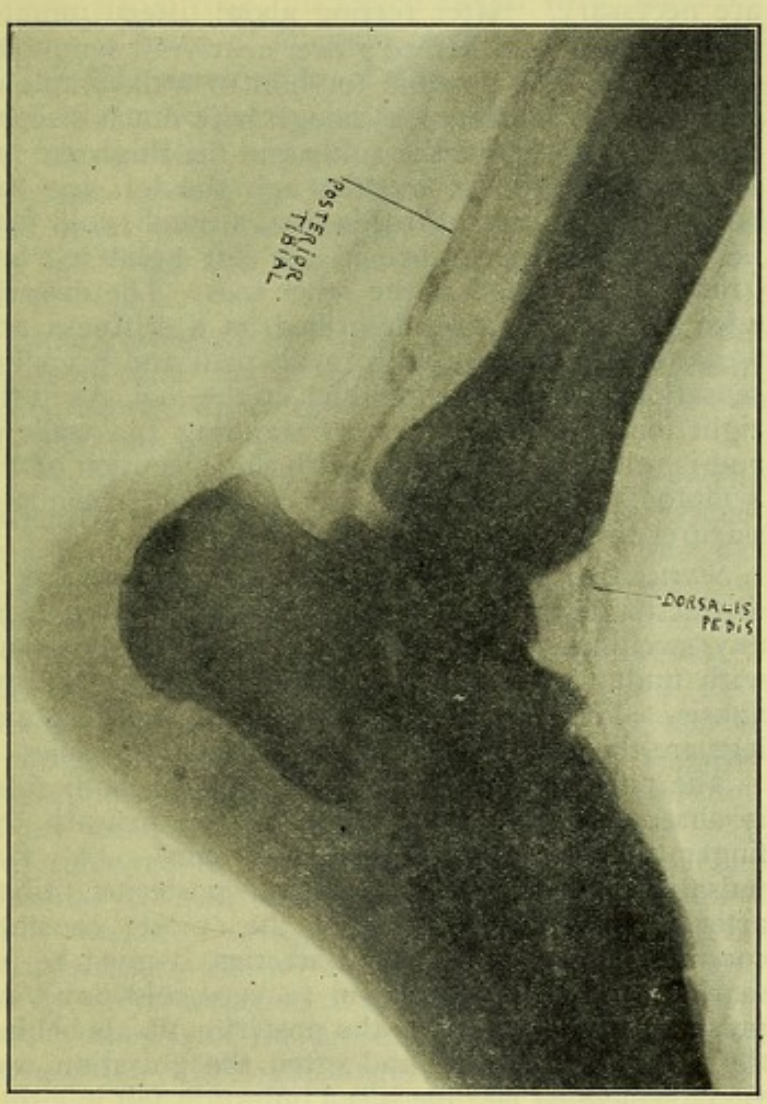

Fig. 1. Skiagram of leg in intermittent claudication, showing deposits of lime and calcareous plates in the walls of the dorsalis pedis and posterior tibial arteries.

symptoms are much aggravated and frequent rests are necessary. After resting about fifteen minutes with the feet held before a fire, nearly all symptoms disappear. It is possible for him to walk a mile or even a greater distance, although with much discomfort. The leg above the ankle and the thigh are not involved. Until six months ago the left leg had been perfectly free. At this time a numb, cold feeling made its appearance in the left great toe and gradually extended to the other toes. The disturbance here is likewise described as a stiffness and weakness in the toes with much pain and prickling sensations gradually extending up the foot. As in the right foot it is brought on by standing and walking and is relieved by resting. With the exception of the symptoms just described and occasional rheumatic seizures this patient enjoys fair health.

Status Præsens.—The temperature of both feet is distinctly lower than normal. They are free from any swelling, but on standing the veins stand out with undue prominence and there is moderate cyanosis. The venules of the skin of the feet and legs are the seat of minute dilatations and ectasies.

The pulsation in the dorsalis pedis on both sides is absent and the artery may be felt beneath the finger as a minute rounded cord. A fair pulsation is present in the posterior tibial arteries. While this was the most constant condition noted in the pedal arteries, it must be remarked that on one occasion (a very cold day), no pulsation could be felt in the posterior tibials behind the internal malleolus and often the pulsation was very feeble. At no time was a pulsation felt in either dorsalis pedis artery. The pulsation of the femoral arteries and the popliteals was of normal volume and equal on the two sides. The general indications of arteriosclerosis are manifest. Arcus senilis, hyper-

trophy of the left ventricle, accentuation of the second aortic tone, thickened radial with increased tension, a trace of albumin in the urine, no sugar. A careful examination of the nervous system, including motility, sensation and reflexes fails to show any abnormality whatever. The calves of the legs show no atrophy or fibrillary twitchings and present equal measurements on the two sides. He is not flat-footed and there is no tenderness along the nerve trunks.

Radiographs of the right foot and leg show deposits of lime in the posterior tibial and dorsalis pedis arteries delicately outlining their cause. See the illustration.

Case IV.—(Cornell Neurological Clinic).—The patient is forty-nine years old and has been an iceman for thirty-two years, an occupation which exposes the hands and feet to the influence of cold and wet almost continuously. He has indulged excessively in alcohol for many years, as a result of which had gastritis and hemorrhages two years ago. Smokes six cigars a day and chews tobacco almost without intermission. In 1879 acquired syphilis, ulceration on the right leg in 1880 and a perforation of the nasal septum in 1885. In September, 1903, was removed to the alcoholic pavilion of Bellevue Hospital in delirium tremens, where he remained eight weeks. During a part of this time restraint was necessary and he was bound in the usual manner by sheets attached to the wrists and ankles. The first symptoms of the present affection were noticed immediately after discharge from the hospital and were attributed by the patient to the restraints used. He complains of a numbness and "dead" feeling in the toes and dorsum of both feet which become stiff and weak in walking. The left side is worse than the right. The stiffness extends as high as the mid-

dle of the calf and is produced and aggravated as are the sensory manifestations by standing and walking.

Last summer he could walk a mile without resting, with discomfort to be sure, but at present (winter), a quarter of a mile renders a rest necessary preferably before a fire, when the more distressing symptoms disappear in about fifteen minutes. The feet feel cold and numb most of the day and swell considerably after standing and walking. They also become blue and mottled in appearance. At times sharp, shooting pains are felt in the feet and legs. On rising in the morning the legs feel perfectly well and natural but after standing and a short walk, the numbness, coldness, and stiffness reappear.

Status Præsens.—Both feet and ankles are swollen and cyanosed. The dorsal surface of the feet and the front of the legs present numerous small venous dilatations and varicosities. The toe-nails of the left foot are marked with transverse ridges. A full pulsation in the dorsalis pedis is present on both sides. A faint pulsation is apparent in both posterior tibial arteries. Good full pulsation in both femorals and in both popliteals. Not infrequently on a cold day it was quite impossible to detect any pulsation in the posterior tibials.

The general signs of arteriosclerosis are present, and the urine contains a faint trace of albumin but no sugar. The motility, sensations and reflexes are normal. No tenderness over the nerve trunks, no wasting or fibrillary twitchings in the muscles of the leg. Radiographs of the left foot and leg show an exquisite deposit of lime salts in the walls of the posterior tibial and dorsalis pedis arteries distinctly outlining their course.

Remarks.—The cases just narrated present the typical pictures of the intermittent limping in vary-

ing degrees of severity. The ages of the patients were fifty-eight, forty-two, sixty, and forty-nine years respectively. The etiological factors noted were. CASE I.—Neurotic temperament, gouty heredity, anxiety, and tea. CASE II.—A moderately nervous temperament. CASE III.—Excesses in alcohol and tobacco and prolonged exposure of the feet to cold and wet for many years. CASE IV.—Syphilis, excesses in alcohol and tobacco, and constant exposure of feet to cold and wet for years. Exciting cause: Restraint in alcoholic delirium.

Both legs were affected in CASES I, III and IV, but of unequal intensity. In CASE II the left leg alone was involved. Pulsation was absent or diminished in one or more of the pedal arteries in CASES I, III and IV. In CASE II the anomalous condition was found of a diminished posterior tibial pulsation on the unaffected side. The *x*-ray photographs taken of CASES III and IV, the walls of the posterior tibial and dorsalis pedis arteries are distinctly outlined by calcareous deposits. According to Sänger this method has a certain prognostic importance, for, as was pointed out by Charot, cases dependent upon calcareous deposits are less amenable to treatment, than the arteriosclerosis and the endarteritis. In all the cases something has been gained by treatment, in the amelioration of symptoms and the lengthening of the "time-limit," but in none was a very definite improvement noted. On the other hand, since the institution of a proper regime and treatment, the malady does not appear to have progressed materially.

To Summarize Very Briefly.—Angiosclerosis of the extremities in its gravest form, uncomplicated by a vasomotor nerousis leads to spontaneous gangrene. If the angiosclerosis occurs in combination with a vasomotor instability and a tendency to

vasomotor spasm, the syndrome of intermittent claudication results. This is characterized by the development of sensory (pains and paresthesia), and motor (weakness and rigidity) manifestations during functional activity with a rapid and permanent restoration to the normal during rest. The syndrome, however, has a wider and more general application to the whole circulatory mechanism and has been observed in relation to various organs of the body (heart, intestines, brain, kidney, and eyes).

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