Original Articles.

ANGINA CRURIS (INTERMITTENT CLAUDICATION), AND ALLIED CONDITIONS, INCLUDING PAINFUL CRAMPS, WITH REMARKS ON THE IMPORTANCE OF EXAMINING THE PEDAL ARTERIES.

BY G. L. WALTON, M.D., AND W. E. PAUL, M.D., BOSTON.

THE affection for which we venture to suggest the name "angina cruris" has been discussed under various titles since its introduction by Charcot in 1858, principally in neurological publications. It has not yet received, however, the general recognition to which it is entitled, and has practically eluded the textbooks, both those on general medicine and those on the diseases of the nervous system. It is in the hope of arousing general interest in the subject that we have chosen this comparatively simple, easily assimilable, and (as it seems to us) fairly appropriate name, in preference to such elaborate and yet inadequate terms as "myasthenia paroxysmalis angiosclerotica" (Higier), "angiosclerotische intermittierende dysbasia" (Erb), or "intermittierende Muskelparese" (Grassman). The "claudication intermittente" of Charcot and the "intermittirendes Hinken" of Erb are sufficiently simple for general use, but are misleading, in that they unduly emphasize an unessential feature of the disorder (lameness) while drawing no attention to the essential symptom, namely, intense paroxysmal pain, a symptom which takes, perhaps, the first place among the associative memories aroused by the word angina.

Most of the names previously presented, even if appropriate, are ill adapted for general use, as applied, not to a neurological curiosity, but to a set of symptoms found with more or less frequency, though not in their severest form, in the practice of every physician and surgeon.

The clinical picture of well-developed angina cruris consists of more or less frequent brief attacks of intense paroxysmal pain in the leg, affecting as a rule the same region, oftenest the calf, recurring at irregular intervals, generally when in the erect position, but not infrequently while the patient is in bed, often accompanied by local asphyxia and cyanosis, and usually in a limb in which pulsation is wanting in the dorsalis pedis or the posterior tibial artery, or both. The attack resembles in its severity that of painful facial tic. The underlying cause is probably a restricted circulation (Charcot,¹ Erb,² Hagelstam,³ Goldflam,⁴ Dutil and Lamy,⁵ Elzholtz,⁶ Higier,⁷ Oppenheim,⁸ Marinesco,⁹ and others), whether resulting from (a) the degenerative arterial changes incident to advancing years (atheroma), from (b) congenital predisposition to feeble circulation and to arteriosclerosis, or from (c) acquired arterial disease (obliterating arteritis, resulting, for example, from syphilis). Vasomotor spasm (common in persons of neuropathic inheritance) is generally regarded as the exciting cause of the attack, combined, per-

haps (Goldflam), with sudden accession of blood pressure. We shall not undertake to contribute to the already long list of theoretical considerations bearing on the pathology of the vascular occlusion, the pathogeny of the vasomotor spasm, or the exact seat of the pain, whether in vessel, nerve or muscle. We feel impelled, however, to confess our predilection for the view of Goldflam, that the pain is of vascular origin, conveyed, perhaps, as he suggests, from the sensory corpuscles in the vessel walls. It is true that simple pressure upon blood vessels is not productive of pain, and that simple vascular contraction and distention do not result in pain, but it is also true that simple pressure upon nerve trunks and nerve endings does not necessarily produce pain. This argument by no means precludes a susceptibility to pain resulting from a definite combination of mechanical conditions (for example, vasomotor spasm combined with increased blood pressure). Pain is primarily a cry of warning, contributing to our safety, and resulting from such conditions at the periphery as may do harm to the structures concerned. As instances of pain resulting from vascular rather than from nervous disturbance we need only name that resulting from embolus, from aneurism, from the continued application of the tourniquet, and perhaps that of migraine. Nor shall we dwell on the bearing of the study upon the paresthesias and pains of Raynaud's disease, erythromelalgia, and allied disorders. The reader who would pursue this line of investigation is referred to the articles of Charcot, Erb, Goldflam, Dutil and Lamy, Higier, Hagelstam, Mariensco, Ilaga,¹⁰ Sternberg,¹¹ Van Ordt,¹² Laveran,¹³ Panas,¹⁴ Lancereux,¹⁵ and others. Our main endeavor is to familiarize the practitioner with the clinical aspect of the disorder, to suggest that certain familiar but unclassified pains may represent a modified form of the malady.

Higier, Goldflam and Hagelstam have recently demonstrated the comparative frequency of the disorder by reporting eighteen, twenty-four and twenty-three cases respectively. These authors concluded that certain races (for example, the Semitic) were peculiarly subject to the complaint. We have collected in the past few months fifteen cases representing, as it seems to us, a modified type of the symptom, and one typical case, none of the individuals affected being included in the race mentioned; this tends to show that while the affection in its complete form is rare, allied conditions are of wide distribution, though the only previous cases reported under this category in American periodicals, as far as we can learn, are those of Putnam,¹⁶ of Gordon,¹⁷ of Riesman,¹⁸ and of Dana.¹⁹ * In twelve of the sixteen cases pulsation was lacking in one or more pedal arteries.

It is significant that only five of these cases came to the Neurological Department of the hospital, and only one of these five for treatment of this particular symptom. Two cases were found

* The case published by Dana presents a picture of unilateral arterial obliteration in a woman of seventy-eight without paroxysmal symptoms but with pain and paresthesia.

in the Surgical Department, in a routine examination of the arteries (allowed by the courtesy of Dr. Scudder). The others were not hospital patients, but persons whose histories we investigated with this subject in mind, after learning that they were sufferers from so-called "cramps" of unusual persistence and severity and of constant seat.

Comparatively few, perhaps, of the many sufferers apply for treatment of this particular symptom, and when they so apply, the case is diagnosed as cramp, sciatica, the result of flat-foot, internal varicose veins, or, perhaps, if the history is somewhat bizarre, as hysteria, its true nature remaining unnoted, unless indeed, as is occasionally the case, evidence of malnutrition or even local gangrene makes its appearance, in which event the minor complaints receive scant attention unless the practitioner chances to be familiar with the subject. The muscles are generally hardened during the attack, whether this hardening is the result or one of the causes of the pain; but the malady is not to be confounded with ordinary cramps of occasional occurrence and varying location. The possibility should be borne in mind, however, that the ordinary cramp, though innocuous, may be of analogous production as far as the ischemia, the vasomotor spasm and the temporary increase of blood pressure are concerned. It is significant that such cramps are practically limited to the lower extremities, in which mechanical conditions render the circulation less free than in the upper. The essential distinction in this event would be the pathological basis for continued impairment of circulation present in well developed angina cruris. In point of fact not a few sufferers from so-called simple cramps will be found on careful study to fall into the class of modified angina cruris.

The details of the following case will serve to present the picture of this distressing malady in its severest form. The patient presented himself at the Neurological Clinic of the Massachusetts General Hospital, and was recommended to the house, where he entered the service of Dr. F. C. Shattuck, through whose courtesy we were enabled to study and report the case. Material aid was furnished by the careful records of Dr. Locke.

OBSERVATION I. R. L. C., 38, married; English; a weaver.

Family history.—His father died of pneumonia at 54. Two sisters died in infancy; one brother and two sisters are alive and well. His mother was always morose, nervous and despondent; is now in the insane asylum. Her brother is also insane. The family is neurotic.

Habits.—Has not taken alcohol for four years. Before that drank one or two glasses of beer in the day.

Patient's history.—He had mumps and measles in infancy, scarlet fever at fourteen. As a child was nervous and was subject to frequent pains in the legs. Since eighteen years of age he has been subject, once or twice a month, to severe frontal and occipital headaches, lasting one to twenty-four hours, accompanied by nausea and vomiting (migraine). Venereal disease is denied.

Four and one-half years ago he was obliged to give up work for six months. During this time there is

history of diarrhea with rectal and vesical incontinence. Three years ago discontinued work in the mill and was confined in bed seven weeks with girdle pain and pain extending into the legs, with tingling and numbness and distinct loss of power in the legs. Improvement followed, then he became worse, and two years ago entered the Fall River City Hospital. Marked improvement followed and the pain and disturbance in motion entirely disappeared. After two months he took up peddling, but this work was interrupted by sharp pains, which always began in the lower legs and feet when he was in the erect posture. He was confined to his bed for five months, after which he was up and down until he entered St. Joseph's Hospital in Rhode Island. He was there two or three months without relief.

For three years the urine has been passed frequently and he has been subject to dizzy spells. He has gained weight of late. His sleep is poor. His sexual power is unimpaired. For several years previous to the onset of these symptoms he was under great strain; his wife was very ill and two children died. He has worried for fear of becoming insane. For the past year he has taken morphia, often two or three grains daily.

There is no motor loss in the legs, but he is subject to sudden attacks of intense pain in the calf, the outer side of the thigh and back of the knee, finally involving the whole leg. The pain is accompanied by heat and prickly feeling and sometimes by changing color of the extremities. The pain is so intense that he is obliged to stand holding the leg or to sit down. The spasm passes off as rapidly as it comes, after which he can walk with freedom. The pain sometimes comes when he starts to walk, again if he has walked a distance. He may continue to walk after having an attack, for perhaps three minutes, when another will occur. Movement in bed often produces similar attacks. He has had similar pain in the left leg, but no motor or sensory trouble in the arms. Cold weather aggravates the disturbance.

Physical examination.—The patient is well developed and nourished. The pupils are alike and react normally. The tongue is moist and clean. There are small glands in the left axilla and in the groins. The lungs and abdomen are normal. The apex beat of the heart is in the fifth intercostal space just inside the nipple; the force of the heart is fair; no murmurs; the second aortic sound is accentuated, the first sound at the apex sharp and booming.

The pulses at the wrist are equal and synchronous, volume and tension good. The pulse can be felt in the posterior tibial arteries, though its character is thin and weak. No pulse can be felt in the dorsalis pedis of either foot. Examination of the blood shows 75% hemoglobin, white corpuscles 21,000 at the first examination, 25,000 at the second.

The blood pressure, taken in the finger and in the second toe (by Dr. James Jackson), varies from 95 to 120 mm. of mercury. The blood pressure taken in the right toe during an attack increased from 95 mm. to 140 mm. This attack, which was a typical one, lasted thirty-four seconds.

Examination is negative as regards sensation and motion. The hip and other joints are free and normal. The knee jerk is exaggerated on both sides. No continuous ankle clonus is elicited, but the Achilles tendon reflex is increased. There is Babinski reflex on both sides, more marked on the left. The cremaster reflex is sluggish, the abdominal and epigastric reflexes wanting. Gluteus clonus frequently appears on the right side when he is standing, sometimes spontaneously, generally started by a tap.

There was marked edema in the right ankle and shin when the patient first appeared at the Out-Patient Department, and a slight edema on the left. This symptom disappeared after he had remained in bed twenty-four hours. In the Out-patient Department the whole right leg was distinctly colder to the upper thigh; both legs were somewhat cool after entering

the house (temperature of dorsum of right foot 82°, of right hand 87.2°).

Urine was of normal color, acid reaction, specific gravity 1.020, with no albumin and no sugar. The sediment was slight, containing no casts.

During his stay in the hospital there has been no diminution of the severity or frequency of the attacks, even when he has remained constantly in bed. The administration in turn of strophanthus and nitroglycerin gave no relief.

This case is noteworthy in presenting a neuropathic and psychopathic tendency, a tendency which has been regarded by most students of this subject an essential factor in the production of vasomotor spasm (Erb, Hagelstam, Van Ordt, Goldflam, Oppenheim, Higier and others). It is also of interest that the reflexes were exaggerated, a condition reported in one or two previous cases, and interpreted (Erb, Goldflam) as pointing perhaps to a defective circulation in the spinal cord, allied to that in the lower extremities. The thought naturally suggests itself that the paralytic seizure through which the patient passed three years ago may have been due to spinal syphilis. This diagnosis would in no way alter the status of his present attacks, but would merely point to specific disease as an important etiological factor in the arterial changes. It should be also noted that the patient was under the usual age of atheroma, and that no tortuosity or hardening or visible pulsation was present in the arteries, either at the wrist or temple. In the early study of the disorder the arterial degeneration incident to advancing years was deemed the essential factor (except in cases of recognized local or general cause of obliteration, as aneurism or syphilis), but many cases have now been reported varying from twenty to forty years of age, and a congenital tendency to arterial fibrosis is regarded as equally important. Snger²⁰ has demonstrated, by the Rntgen rays, chalky deposit in the arteries in three cases, and emphasizes the importance of utilizing this aid in diagnosis.

The histopathology of the arterial occlusion in these cases is not definitely established. In each of the conditions mentioned all the arterial coats are liable to become thickened, in whichever coat the process may commence; and in all the varieties the lumen is liable to become narrowed and eventually obliterated. Upon this purely pathological branch of the subject we shall not venture to encroach.

Another point of interest is the increase of blood pressure demonstrated by Dr. Jackson. This is a branch of investigation to which little or no attention has been paid, but the result of Dr. Jackson's observation would tend to show that increased blood pressure is a factor in the production of the paroxysm. Further study in this direction is desirable.

In view of the question whether the increased blood pressure was merely a result of the movement made by the patient on account of the pain, Mr. Quinby, at the instance of Dr. Jackson, has tested the blood pressure during short periods of violent exercise and has found no appreciable increase.

With regard to the *etiology* of claudication, diabetes, syphilis, misuse of tobacco (and perhaps alcohol), gout, and exposure to wet and cold have been in turn emphasized, but further contributions have shown that none are essential. Two factors, however, appear with great frequency throughout the literature, namely, tendency to defective circulation and constitutional neuropathic taint.

The cases hitherto reported have been very largely males, and this sex has been regarded as peculiarly susceptible to the affection (Dutil and Lamy, Goldflam, Hagelstam, *et al.*). Our experience shows that the modified form to which we call attention is quite as common among females, for out of the sixteen cases which have come under our observation, nine occurred in women. In two of these cases the attacks first appeared during the latter half of pregnancy, tending to show that the increased pressure of the fetus may be added to the mechanical causes, and the question naturally presents itself whether certain cases of so-called "cramps" occurring in pregnancy and labor may not be allied to the symptom under consideration.

The following case will serve to illustrate this phase of the subject and to demonstrate the benefit of treatment directed towards aiding the circulation. The history is also suggestive of a neuropathic tendency such as might favor vasomotor spasm, and perhaps of an inherited circulatory equipment not quite adequate to withstand the strain imposed by childbirth and other conditions.

OBSERVATION II. Patient a widow, 67; American. Her mother was subject to severe cramps at night on account of which she always wrapped the limbs in a blanket; two sisters have been subject to attacks similar to those to be described in her own case, and one sister has been through an attack of phlebitis.

She has been subject to so-called rheumatic pains, such as lumbago. She has had two children, and early in the first pregnancy had dropsy. In the latter part of each pregnancy she was subject to severe cramps of short duration, without continuous pain or disturbance of gait. After the birth of the first child, thirty-five years ago, there was considerable pain and swelling of the legs, face, hands and whole body, and loss of vision. She was confined in bed for a number of months. On trying to reach an armchair she felt something snap in the calf of the left leg and suddenly lost power in all four extremities. The paralysis lasted several days and gradually disappeared. For a month previous she had been nervous, depressed and hysterical. In the course of a week after the onset of paralysis an abscess appeared on the outside of the left shin which was later opened. Both legs remained swollen, the left discolored, flexed and rigid. Gradual convalescence followed, and in nine months she was able to walk without crutches; in a few years she could use the leg as well as ever. The legs have been liable to swelling at intervals since that time, especially if tired. She has been subject to cramps for a number of years, recurring many times in each year and always confined to the right leg, sometimes in the calf, sometimes in the groin. The pain, especially when in the latter situation, has been so severe as to cause her to faint, but has been of brief duration (about half a minute). When the pain has appeared in the calf and foot it has been less severe and has lasted perhaps five or ten minutes. The attacks have invariably come at night. She was advised by one physician to tie a cord round the leg, by another to exercise.

Fifteen months ago the legs had been swollen for several weeks and there was distress in the precordium. On starting to rise a moderate cramp and pain appeared in the back of the right leg; this pain continued and increased and two days later she was confined to the bed. The diagnosis was made of phlebitis and venous thrombosis, the clot being plainly seen and felt on the inside of the right shin. She remained in bed two weeks; gradual recovery followed. During the past year she has worn an elastic stocking and avoided excessive use of the limbs. During this time there has been no recurrence of the cramp.

There are moderate varicose veins. The heart sounds are weak. The physical examination is negative as regards motion, sensation, reflexes and other functions. No pulse is to be felt in the posterior tibial on either side, and the dorsalis pedis pulsation is faint.

No further comment is required in presenting the salient points in the following:

OBSERVATION III. Female, 40, married, of Irish parentage. Has had nine children. With each pregnancy has been subject to severe cramps with pain sufficient to make her cry out. They have appeared about the middle of the term and increased in frequency until the child was born, appearing practically every day toward the end. The severity of the attacks remained unchanged throughout, pain being extreme from the first. The attack would appear while walking or on rising from the bed. She would be compelled to stand still, to hold and rub the leg, and be unable to take another step until the pain had passed. The last child was born two years ago, since which time the same attacks have appeared at intervals of perhaps a month. She has had no miscarriages, no eruption and no loss of hair. She is rather easily startled, worries and has various phobias. Her mother was rather high strung and of a worrying disposition, but there is no history of definite nervous or mental disease in the family. Between the attacks there is no limp, no paresthesia, no pain, no numbness or other disability. The physical examination is negative excepting that the posterior tibial artery is not felt on either leg. The dorsalis pedis is felt on both sides.

The following history is of interest in connection with the study of local malnutrition. It is true that evidences of malnutrition in the extremities may, and often do, appear without the least suggestion of angina. In certain instances, however, it would seem that the right combination of circumstances may produce the characteristic pains, and the study of these cases may aid in the solution of the difficult problems connected with the obscure general subject of nutritional disturbances.

The patient came to the Neurological Clinic with a probable diagnosis of mal perforans accompanying locomotor ataxia, but it appeared that the acute attacks of pain were always limited to the left leg below the knee. This history led to the examination of the arteries of the foot, which showed absence of pulsation. As the result of this study the diagnosis of angina cruris was established without precluding the possibility of early tabes.

OBSERVATION IV. Female. For twelve years has had pain in legs and back, with swelling of the legs. She has had occasional vertigo and some headache, but no vomiting. The bladder is irritable, but there has been no incontinence of urine. An ulcer appeared under the left great toe about six months ago and has persisted. The pain has always been limited to the left leg, never extending above the knee. Sometimes

she has paroxysms of intense pain, of brief duration, preventing walking, though sometimes the pain will persist several hours. She has had no children; no miscarriages; no eruption. She has lost her hair steadily for three years.

The left foot is cold and cyanotic, especially the great toe. There is no pulsation in the dorsalis pedis on either side. There is no loss of muscle sense or other form of sensation, and no loss of motion. The pupils are irregular, the right slightly larger than the left, of rather small size; reaction sluggish. The knee-jerk on the left is increased, on the right very slight. There is a dry, black ulcer under the left great toe. There is no ataxia. There is very slight Romberg.

The patient was transferred to the Surgical Department, to which she was to report the next day for further examination and treatment, but has failed to reappear, so that we are limited to the above brief report of the case.

We append brief histories of a number of cases obtained with so little effort as to indicate that they represent nothing unusual. The frequent absence of pulsation in one or more of the arteries of the foot gives point to the observations.

OBSERVATION V. Female, married, 40, Irish. Two years ago she had an attack of unconsciousness lasting one hour, followed by right hemiplegia with aphasia. The condition persisted with athetoid movements and astereognosis. She has had five or six miscarriages, occurring at about the sixth month. She has had no living children. She has had no rash or sore throat. Her father had diabetes, her mother is in good health. Patient has had various paresthesias in the whole right side with loss of pain, touch, spacing and position senses. The Babinski reflex is present with increased knee jerk. Since the paralytic stroke she has been subject (up to two weeks ago) every morning, on waking and moving, to a sharp pain in the right calf lasting a few minutes without hardening of muscles, but with pain sufficiently intense to cause her to cry out, the foot becoming cold and white. No dorsalis pedis artery is to be felt on the right, the posterior tibial is normal, as also are both arteries of the left foot.

OBSERVATION VI. Female, 64, widow, American; has had three children. As long as she can remember has had cramps in both legs in bed, especially the latter half of the night; also cramps in the hands with strong flexion of the fingers. In the past year has noted in both legs, more in the left, on starting to walk that the leg feels weak from the hip down, causing her to stop for a few seconds. This trouble may occur in crossing the street or in playing the pianola. The left leg feels cold and is cold even on hot days. A crawling feeling occurs in the legs at times, and going-to-sleep feeling in both legs, more on the left. The right dorsalis pedis artery is prominent, the left is faintly perceptible; both posterior tibials are present. The knee jerk is normal. Her father was alcoholic and suffered from cramps. Her mother was subject to cramps so sudden and so severe as to cause her to drop objects from the hand.

OBSERVATION VII. Male, 60, American. Has suffered from cramps in both legs for years at night in bed. He has a weak, dilated heart. Edema of the legs is present. Dorsalis pedis arteries not perceptible. Pulsation is perceptible in both posterior tibials.

OBSERVATION VIII. Female, 30, American, of Irish parentage. Married. One brother and sister nervous. Self nervous (epileptic since sixteen years). Subject to cramps since girlhood for two or three weeks; always at night, especially if feet exposed, and always worse in cold weather. Attacks are of short duration, pain very severe. The dorsalis pedis is felt on both sides, the posterior tibial on neither. The knee jerk is active.

OBSERVATION IX. Female, 13, American, seen in Dr. Minot's clinic. Is subject to severe attacks of painful

cramps, sometimes at night, sometimes on walking. In latter event has to stop. Both feet affected, the left worse. Has these attacks several times a day. The cramp is generally in the calf. Is subject to similar attacks in the arm. The fingers are blue and cold, the right foot is very cold, the left warmer. The arteries are easily felt on the left foot, but on the right the dorsalis pedis is not felt, and the posterior tibial only faintly. The arteries of the upper extremity are rather small.

OBSERVATION X. Male, 48, American. Subject to pains at night on moving in bed. Sometimes has them a number of times in the night, especially during the latter part. Pain is extreme, generally in the calf, and lasts two or three minutes. Disappears rapidly if he straightens the leg. Dorsalis pedis on the left is very faintly felt. The other arteries are normal.

This patient complains of practically continuous fibrillary twitching in both legs, a symptom which has persisted for about three years without other objective sign (myokymia). It is suggestive that fibrillary twitching has been noted in several cases of claudication, and further study may show the connection between them. In the only other case of myokymia coming under our observation, however, the pedal arteries were apparently normal.

The following case was observed before our attention was called to the question of pulseless arteries, so that we are unable to complete the history in this respect, but the impairment of circulation is beyond question.

OBSERVATION XI. Male, American, married, died at 58 years of age. In the last months of his life general edema developed, due to a dilated heart and arteriosclerosis. The urine showed a trace of albumin and was of low specific gravity. Six years before death he first complained of cramps in the right leg, lasting from one to five minutes and subsiding slowly. These cramps always occurred in the early morning when the patient was in bed. At first the cramps occurred at intervals of some months and became more frequent though never oftener than every two weeks. Within a year of his death he was observed in one attack; he was pale, cold perspiration stood on his face and he seemed to be in agony. The pain in the calf of the left leg disappeared slowly after five or ten minutes' duration.

In addition it is only fair to insert a brief statement of several cases in which we found a history of painful cramps of varying frequency and severity, without definite signs of circulatory impairment. It is a question whether we should regard such cases as militating against the views advanced, or whether we should regard them as mild types of a similar disturbance, in some of which the functional element alone is present, while in others a physical basis perhaps exists, but is not sufficiently advanced to present marked signs at the points available for palpation.

OBSERVATION XII. Male, 41. A patient with ataxic paraplegia, seven years ago had an attack of so-called nervous prostration, since which time there has been numbness and unsteadiness of gait, with spastic tendency and increased reflexes with Babinski and clonus. He is subject to cramps of brief duration accompanied by intense pain every time he straightens the legs in bed. The pulse is felt in all four arteries of the feet, but the left radial artery is hard and tortuous.

OBSERVATION XIII. Female, 70, American, married, has been subject for some years to very severe cramps

with intense pain recurring every night for long periods. Has never suffered from phlebitis. There is much lameness and stiffness of the left leg especially about the knee. All arteries are felt plainly. There is flatfoot on the left side.

OBSERVATION XIV. Female, married, 40, American. Suffered from phlebitis in the left leg 18 years ago, since which time has been subject to occasional nocturnal cramps, accompanied by intense pain. The cramps always appear in the left leg. Pulsation is felt in all arteries.

OBSERVATION XV. Male, 44, American, a physician. Has been subject for several years to cramps in the calf occurring at night, accompanied by intense pain. Flatfoot is present. Since wearing a plate for the flatfoot, has been free from cramps, excepting on one occasion, after leaving off the plate the cramps occurred with such frequency and severity as to cause him to renew its use. Pulsation is normal in all arteries.

A review of these cases suggests that there is no sharp dividing line between (a) simple cramps with occasional occurrence and with moderate pain, and (b) severe and frequent cramps of constant seat, and (c) the classical type of angina cruris. In Observation XV it is noteworthy that the painful cramps disappeared after the use of the footplate, and that in Observation XIII flatfoot was present on the side affected with cramps, and that in both these cases the pulse was good in all the arteries of the foot.

The question presents itself, therefore, whether the cramps in such cases are of a different kind from those accompanying signs of poor circulation. The subject of one of these observations (a physician) suggests that possibly the cramps of flatfoot are due, in part at least, to alterations in the circulation of the foot brought about by the abnormal mechanical conditions. Dr. Goldthwait assures us, in this connection, that evidence of impaired circulation, as well as hyperidrosis, not infrequently accompanies flatfoot, and that these symptoms are often relieved by the use of the plate and of appropriate exercises.

The subject of cramps in general seems obscure, and it is to be hoped that the line of thought here offered may serve as a stimulus for their future study.

If it were found an invariable rule that painful cramps are accompanied by pulseless arteries, the proposition might be regarded as practically established, but this is by no means the case. We have already cited cases in which cramps appeared without pulseless arteries, and the following observation illustrates the converse relation. In this case no pulsation was felt in the dorsalis pedis, posterior tibial or popliteal arteries, but no pain was present severe enough to suggest angina. The patient did, however, complain of troublesome paresthesia, a symptom often present in sufferers from this disorder.

OBSERVATION XVI. J. D. S. (patient of Dr. Fitz), 42, laborer, Irish; had typhoid fever two and one-half years ago, complicated by gangrene of the left leg, which was amputated above the knee. Since that time he has suffered from burning and prickling of the thighs extending up the back to the waist line. His sleep is impaired and he frequently has to change his position at night to seek comfort. The right leg has been losing power during the past year. The ra-

dial pulse is weak, the artery small. No pulsation is felt in the dorsalis pedis, the posterior tibial, or the popliteal artery of the right leg.

With a view to determining the frequency of pulseless pedal arteries in health, and in persons not complaining of painful cramps, we have examined 200 individuals of whom the majority were in good health. These individuals have varied from old age to early infancy. Our thanks are due to Dr. Morse for allowing us the privilege of his clinic at the West End Nursery; also to Miss Amy Morris Homans, the director of the Normal School of Gymnastics, for the opportunity of examining a large number of healthy young women; also to Mr. Roberts, who has kindly aided us by the examination of healthy young men in gymnasiums. The results are as follows:

Pulsation felt in all four arteries	181	
Pulsation absent in both dorsalis pedis arteries	10	
Pulsation absent in one dorsalis pedis	4	
Pulsation absent in both posterior tibials	3	
Pulsation absent in one posterior tibial	1	
Pulsation absent in all four arteries	1	infant.
	200	

It should be noted that in the majority of healthy individuals in whom certain arteries were not felt, the other arteries were well developed and pulsated strongly, showing that we had to do with a physiological anomaly. It is common, on the contrary, in angina cruris and allied conditions, with pulseless arteries, to find the pulsation in other arteries feeble. In certain cases, again, we had to do with an artery neither wanting nor obliterated, but obscured by thickened tissues. Our results are less uniform in this connection than those of Erb and of Goldflam, but they serve none the less to indicate that the occurrence of painful cramps in combination with pulseless arteries is too striking to be a mere coincidence.

Those examined were generally questioned with regard to cramps, and it is noteworthy that the only individuals (two) giving a history of cramps at all severe, frequent or fixed in location were among those in whom the pulse was not to be felt in either dorsalis pedis artery, and in one of these persons the feet showed marked signs of impoverished circulation. These two cases might equally well have been included among those probably allied to angina cruris.

With regard to the prognosis and treatment of angina cruris, the prognosis resolves itself into an estimate of the competency of the vascular system in the extremities. In some of the severe cases the nutrition of the part will become seriously compromised, but in the large majority the outlook, according to Erb and others, is fairly good if the conditions are favorable.

Prophylaxis is of signal importance. Persons with tendency to arteriosclerosis should avoid violent, continued exercise, should avoid extremes of temperature, and if tendency to flatfoot exists this should of course receive skilled attention. If angina cruris is well developed the patient should be kept in bed for a certain period, and when he is allowed to walk should do so, as Erb expresses it, with the watch in the hand, endeavoring

always to keep within the amount of exercise which would produce a cramp. Strophanthus and nitro-glycerin should be tried. Iodide of potassium has been advised by all authorities and should, perhaps, be used, especially if there is reason to suspect syphilis. Bandaging the affected limb has appeared useful, and in one of the cases of modified angina reported in this paper (Observation II) the elastic stocking combined with avoidance of exercise seemed of value, since no attack occurred during the year of these precautions. Erb has recommended a galvanic foot-bath, the details of which may be found in his article. Riesman¹⁸ recommends vegetable diet, avoidance of red meat, alcohol, tea and coffee, and of rich foods.

This investigation has impressed us with the importance of examining the dorsalis pedis and posterior tibial, as well as the radial and temporal arteries in all cases in which an impaired circulation may play a part in the pathological process, whether of the brain, spinal cord or other organs. It is well known that the finding of healthy arteries in one region does not preclude serious changes in the vessel walls elsewhere. In case symptoms point, for example, to vascular occlusion, or hemorrhage of the brain, we cannot assume that cerebral atheroma is present, because we find a rigid radial artery, nor can the converse be established. Our only recourse is to examine such arteries as are available for palpation, and to draw what inferences we may. It behooves us, then, in all doubtful cases, to include the pedal arteries in the examination. This is the more important, inasmuch as these arteries may well be the first to suffer, partly from their distal location, and partly from the mechanical disadvantages under which they labor. We select one from many instances showing the advantage of such examination:

OBSERVATION XVII. A lady of 61, seen in consultation with Dr. Phippen of Salem, has suffered from progressive paralysis of insidious onset, commencing in the right hand and gradually spreading during the past two months so that the entire arm is now helpless and the right leg partially so. There is no headache, no vomiting, no optic neuritis. The arteries at the wrist and temple present no notable variation from the normal, but no pulsation is felt in either posterior tibial, and none is felt in the left dorsalis pedis, though the pulsation is normal in the right dorsalis pedis. There is no history of painful cramps.

The examination of the pedal arteries was of material aid in establishing the diagnosis, already suggested by the clinical history, namely, progressive softening due to vascular occlusion. This case further shows that pulseless pedal arteries do not necessarily produce angina.

Though it leads us somewhat afield, we cannot resist alluding in this connection to the indications of spinal disease sometimes accompanying angina cruris. A review of the present pathological status of the so-called primary degenerative cord diseases shows that while the degeneration is recognized as peculiarly prone to attack those portions of the cord whose blood supply is

normally the feeblest (that is, the lateral and posterior columns), the exact part played by the circulation is not determined by those who have given the subject the most study (Marie,²¹ Putnam²² and Taylor,²³ Dana,²⁴ Russell, Batten and Collier,²⁵ and others). Even Marie, who lays special stress on the vascular origin of these degenerations, practically subscribes to the views of Ribbert, who assumes that some *irritant* must be present, thickening the vessel walls and producing diapedesis at the spots he regards as foci of inflammation. Mere local ischemia in these regions (regions whose blood supply is already naturally poor) seems nowhere accepted as sufficient cause for degeneration, though thickened arteries, collapsed arteries, arteries showing hyaline degeneration, and even signs of arterial obliteration, have appeared with more or less constancy in the pathological reports and though Osler states that the *cerebral* symptoms of arteriosclerosis embrace those of many degenerative processes, acute and chronic. If a *toxin* must be invoked for these changes, it seems remarkable that such toxin should invariably attack individuals from forty-five to sixty years of age, and sometimes more than one individual in a family, whereas this is just the age when the effects of a congenitally feeble circulation might be expected to become apparent, and the occurrence of the symptoms at this age in more than one member of a family could be explained, without stretch of imagination, by a family tendency to an inadequate circulatory system.

The following case suggests the possibility that some of the paroxysmal pains of the degenerative spinal diseases (for example, tabes) may be allied to angina cruris and have their origin at the periphery rather than in the spinal roots from which they are ordinarily supposed to be referred.

OBSERVATION XVIII. An American, 53 years old, gives a history of primary sore followed by eruption, twenty years ago. During the past year his disposition has changed, and his memory failed. He is now mentally incapacitated for his work. The speech is somewhat impaired, the pupils are unlike and the right fails to react to light. The gait is unsteady, and the ground feels unnatural to him, though no objective anesthesia is found. The knee jerks are exaggerated, without ankle clonus or Babinski reflex. There is painless enlargement of the right knee joint, with great increase of fluid.

The patient complains of frequent attacks, twenty or more daily, of paroxysmal pain in the calf, generally brought on by walking, of brief duration, and so intense that he cannot proceed till the spasm has passed. On the left foot no pulse is felt in either the dorsalis pedis or the posterior tibial artery; in the right foot pulsation is felt only in the dorsalis pedis. He complains also of sharp darting pains in various localities.

The diagnosis is obviously general paralysis, with characteristic spinal involvement, but the paroxysmal pains of which he chiefly complains conform much more readily to the type under discussion than to the type of pain ordinarily attributed to irritation of nerve roots resulting from disease of the spinal cord.

CONCLUSIONS.

(1) The concurrence of the paroxysmal pains of angina cruris with pulseless pedal arteries is

too constant to be explained by coincidence, though it is true that pulseless arteries may be found without the pains, and, conversely, that such pains may appear with apparently normal arteries.

(2) The painful paroxysms are probably of vascular origin, and result from vascular spasm coupled, perhaps, with increased blood pressure acting on vessels already partially occluded, whether from local or general disease (aneurism, syphilis), from senile changes (atheroma), or from congenital tendency to angiofibrosis.

(3) Recurring painful cramps of constant seat probably represent a modified form of angina cruris.

(4) It is important to examine the dorsalis pedis and posterior tibial, as well as the radial and temporal arteries, in all cases in which it is desirable to estimate the bearing of the vascular condition upon disease in the central nervous system or elsewhere.

REFERENCES.

1. Charcot. Leçon du, Mai 31, 1886; *Progressive Medicine*, 1887, No. 32, p. 99, No. 33, p. 115; *Leçons du Mardi*, Dec. 13, 1887; *Leçons du Mardi*, 1892, I, 44; *Compte-rendus et Memoirs de la Société de Biologie*, 1858, S. 2, xii, 225; *Gaz. méd. de Paris*, 1859, No. 19, p. 282.
2. Erb. *Deutsch. Zeitschr. f. Nervenheilk.*, 1898, Bd. xiii, H. 1 and 2.
3. Hagelstam. *Loc. cit.*, Nr. 20, p. 65.
4. Goldflam. *Neurol. Centrbl.*, March 1, 1901; *Deutsch. Med. Woch.*, Sept. 5, 1895, Nr. 36, p. 587.
5. Dutil and Lamy. *Arch. de méd. exp. et d'anat. pathol.*, 1893, v, 102.
6. Elzholtz. *Wein. Med. Woch.*, 1892, Nrs. 49 and 50, S. 1893, et seq., S. 1919, et seq.
7. Higier. *Deutsch. Zeitschr. f. Nervenheilk.*, Nr. 19, p. 438.
8. Oppenheim. *Loc. cit.*, 1898, Bd. xvii, p. 317.
9. Marinesco. *Semaine méd.*, Fevr. 15, 1896, No. 9.
10. Haga. *Virchow's Arch.*, April 19, 1898, Bd. ciii, p. 26.
11. Sternberg. *Wien. klin. Woch.*, 1895.
12. Van Ordt. *Neurol. Centrbl.*, Sept. 1, 1901.
13. Leveran. *Semaine méd.*, 1894, p. 100.
14. Panas. *Loc. cit.*, p. 255.
15. Lancereaux. *Loc. cit.*, p. 261.
16. Putnam. *Boston Medical and Surgical Journal*, Feb. 21, 1901, p. 182.
17. Gordon. *New York Medical Journal*, Nov. 10, 1900, p. 806.
18. Riesman. *American Medicine*, May 25, 1901, p. 343.
19. Dana. *New York Medical Record*, Feb. 22, 1902.
20. Sanger. *Monats. f. Psych. u. Neurol.*, December, 1901, Bd. x, H. 6, p. 465.
21. Marie. *Leçons sur les mal. de la moelle*, Paris, 1892, p. 158.
22. Putnam. *Journal Nervous and Mental Disease*, February, 1891.
23. Putnam and Taylor. *Loc. cit.*, January and February, 1901.
24. Dana. *Loc. cit.*, April, 1891, and January, 1899.
25. Russell, Batten and Collier. *Brain*, Spring, 1900.

PHYSIOLOGICAL HEART MURMURS PRODUCED BY THE ELECTRIC-LIGHT BATH.

BY THOMAS HOWELL, M.D., WORCESTER, MASS.,
Assistant Physician, Worcester Insane Asylum.

THE readiness with which murmurs, or something which answers for them, can be produced in the heart and blood vessels was strongly impressed upon me while engaged recently in conducting a series of experiments to ascertain the physiological effects of the electric bath upon the human system. These experiments demonstrated conclusively to me how little weight should be attached to the mere presence of a murmur in making a diagnosis of heart disease. In all, fifty-two persons were examined, with special reference to the effects of the bath on the heart, and murmurs were heard in the heart or arteries, gen-