The Lumleian Lectures
ON
ANGINA PECTORIS.

Delivered before the Royal College of Physicians of London

BY WILLIAM OSLER, M.D., F.R.S.,
REGIUS PROFESSOR OF MEDICINE IN THE UNIVERSITY OF OXFORD.

LECTURE II.[1]

Delivered on March 15th.

PATHOLOGY.

MR. PRESIDENT AND FELLOWS,—Had Heberden listened to my first lecture he could have remarked very justly: "Well! they have not got much ahead since my day." In descriptive symptomatology we have not, and among 100 cases of angina pectoris there is no reason why Heberden should not have met all the important anomalies and complications. He had the good sense not to say much about the cause of the disease, and the good fortune to get very close to the truth in what he did say. I do not propose to weary you in a vain repetition of the scores of explanations which have been offered since his day. The older ones are to be found in the monographs of Parry and Jurine, the more recent in the Traité of Huchard, in the writings of our President, and in those of Allbutt, Bramwell, Gibson, Morrison, Mackenzie, and others. At the outset let us frankly face the main obscurities which have not yet been cleared up. Why is it more common in the upper classes? Why do we not see it more often in hospital practice? Why are the lot and portion of the poor, among whom degeneration is more widespread. It is as though only a special strain of tissue reacted *anginally*, so to speak, a type evolved amid special surroundings or which existed in certain families. Or there may be a perverted internal secretion which favours spasm of the arteries, as Harvey at Cambridge has shown to be the case with pituitary extract and the coronary vessels. And a case of aortic valve disease is reported in which the use of this extract caused anginal attacks. This suggestion is supported by the fact that in myxœdema anginal attacks may be caused by thyroid extract. I saw last year a patient of Dr. Lafleur's of Montreal with this most distressing peculiarity, which was mentioned to me also by Dr. Allan Starr of New York. The disease may occur in three generations, as in the Arnolds, and a father and four children have been affected. In three instances of my series father and son were attacked; in two, brothers; and in one, a brother and sister. It is not the delicate neurotic person who is prone to angina, but the robust, the vigorous in mind and body, the keen and ambitious man, the indicator of whose engines is always at "full speed ahead." There is, indeed, a frame and facies at once suggestive of angina—the well "set" man of from 45 to 55 years of age, with military bearing, iron-grey hair, and florid complexion. More than once as such a man entered my consulting-room the suggested diagnosis of angina has flashed through my mind. Still more extraordinary and inexplicable is an imitative feature, if one may so speak of it, by which the repeated witnessing of attacks may induce one in the observer. The case of Senator Sumner attracted widespread interest on account of his distinguished public position. Two weeks after his death Dr. Hitchcock, his physician, died in an attack with coronary artery disease and acute infarct of the myocardium. Tabor Johnson, his other physician, at that time a young man, had two attacks, diagnosed by Brown-Sequard as angina, and he had seen some twenty cases of what may be called the manufactured variety. Straus died not long after his friend Charcot. A young man, aged 28, whose father, a very vigorous planter, had through the spring and summer of 1900 severe attacks and died in one Sept. 28th, consulted me the following January for angina. I had seen the father, and had been a witness to the devotion of the son during the terrible paroxysms. Within a month of the death of the father he began to have severe pain in the chest, with pallor, sweating, the pains down the left arm, which became numb and tingled. The sister said the paroxysms were identical with those of the father, and naturally the family were greatly distressed. The patient was a healthy, robust fellow, very neurotic, and almost frightened to death. A reassuring prognosis was all the treatment he required. He has had no further attacks. A woman, aged 38, after her father's death from angina, had severe pains about the heart, and attacks which she insisted were of the same character, but she, too, got quite well. A still more remarkable illustration of the imitative, emotional influence was seen in the outbreak of angina-like attacks among the sailors of the French corvette *L'Embuscade* reported by Gelineau.

There are two primary features of the disease, pain and sudden death—pain, paroxysmal, intense, peculiar, usually pectoral, and with the well-known lines of radiation—death in a higher percentage than any known disorder, and usually sudden. Often, indeed, it is, as the poet says, "Life struck sharp on death." The problems for solution are: What is the cause of the pain? Why the sudden death? The secondary features of the attack, the vasc-motor phenomena, the radiation of the pain, the cardiac, respiratory, and gastric symptoms are of subsidiary interest.

MORBID ANATOMY.

Naturally, in the presence of a disease with such startling characters, men have sought an explanation in the bodies of its victims. And angina pectoris has a very definite morbid anatomy, few affections more so, since in practically all cases vascular disease exists. With Morgagni, Jenner, Fothergill, and Parry, a majority of authors have correlated the fatal symptoms with the arterial disease; others have reached the less satisfactory, if more philosophical, position of Rougnon, who, taking all the circumstances into consideration, concluded, "Monsieur Charles est mort parce qu'il est mort." Not a hospital disease, one naturally does not see many necropsies. I have notes of 17 post-mortem examinations, all in men, 8 of them in men under 40 and 4 of them with a history of syphilis, and dying at the ages of 34, 38, 37, 39. They fall in three groups—aortitis, coronary artery disease, and a negative case.

A. Aortitis.—From the publication of Morgagni's famous case writers have recognised the importance of aortic changes at its root. The special importance of this has been dwelt upon by my brother regius of Cambridge, whose many publications upon the subject, dating from his remarkable study of syphilitic arteritis[2] in 1868, have edified his colleague and students. For our purposes here there is but one aortitis—the syphilitic. Occasionally a fairly acute process occurs at the root of the aorta in the specific fevers, but this is very uncommon, except in connexion with endocarditis. Chronic atheromatous changes in the aorta of the aged are very rarely associated with angina unless the coronary arteries are involved. Syphilitic aortitis is a most distinctive lesion. I pass round the beautiful plate of Corrigan's paper, in which he brings out for the first time I think, and with great clearness, the connexion of the disease with this lesion. The frontispiece of Balfour's book on "The Senile Heart" gives an equally good representation. Upon its anatomical features I need not dwell further than to refer to its predilection for the supra-sigmoidal region, the sectional limitation, and the great frequency of its association with aneurysm.

Of the post mortem examinations of my series only one offered a good illustration of the supra-sigmoidal type; a negro, aged 38, who had had syphilis about a year before. The attacks of angina began in December, 1904; they lasted for from 15 minutes to half an hour, with very characteristic distribution of the pain; in severer paroxysms he had fallen unconscious. The attacks recurred even when he was in bed and quiet. There was diffuse cardiac impulse, the area of flatness was increased, but there were no murmurs; the blood pressure was 188 mm. Hg. On the evening of admission he had a very sharp attack, and another at 1.30 A.M., in which the pain was chiefly epigastric; he sweated profusely and became very weak, and at 2.30 was found unconscious, and died at 4 A.M. The heart weighed 490 grammes; the free edges of the valves were a little thickened; the only important lesion was an extensive fresh-looking aortitis, involving the root of the vessel and narrowing the orifice of the left coronary. The right coronary orifice was normal; the coronary arteries themselves were not affected.

[1] Lecture I. was published in THE LANCET of March 12th, p. 697

[2] St. George's Hospital Reports, 1868.

Another syphilitic patient, W. A. M., aged 38, admitted Feb. 20th, 1895, had very severe paroxysms of angina, with aortic insufficiency. The aortic segments were thickened and curled; the coronary arteries were small but healthy; there was the characteristic sclerotic aortitis not confined to the root. The smaller arteries of the body, particularly the splanchnic, were tortuous and thickened.

In a third syphilitic case, J. W., negro, aged 34, admitted May 25th, 1897, the paroxysms were most characteristic, and had recurred since March; in several attacks he had become unconscious, and following them he had transient weakness of the left arm. During the fortnight he was in hospital he had several severe attacks; the left arm was distinctly weaker than the right, particularly the grasp of the hand; the heart appeared to be normal. On June 8th he complained of a great deal of coldness of the hands and feet; at 6.10 in the evening he threw up his hands suddenly and died within a few minutes. Widespread aortitis of the sclerotic type, with here and there plaques of atheroma, were the only lesions. The coronary arteries were not involved; they looked small, the walls thin, but there was no occlusion.

B. *Coronary arteries.*—We are all united in the acceptance of the Jennerian view of the close connexion of lesions of the coronary arteries with the disease. As shown in the extensive analysis by Huchard, a very large proportion of all the cases show changes in these vessels. Of the 17 necropsies of my list, 13 illustrated all the varieties of the lesions.

(*a*) Narrowing of the orifices is a very common occurrence, particularly in the syphilitic aortitis, but not often met with without some involvement of the branches. In the case of a man who died suddenly in my wards after recurring attacks the sclerosis of the ascending part of the arch was marked and the orifices of the coronary arteries were extensively contracted; as the post-mortem report states, "they admitted only a bristle." The arteries beyond were nearly normal, showing only slight sclerotic change.

(*b*) Blocking of a branch with a fresh thrombus is very common in cases of sudden death in angina. In my post-mortem experience this has been more frequent in the medico-legal cases of sudden death without symptoms of angina. One of the main stems or a small branch may be plugged with the formation of fresh infarct. In patients who live some time the infarct may soften and pericarditis may be excited. A specimen in McGill College, from a man who died suddenly the day after an attack, shows the left coronary artery blocked by the thrombus and perforation of the softened anterior wall of the ventricle.

(*c*) Obliterative endarteritis, if we may judge from the reports of fatal cases collected by Huchard and others, is *the* lesion of the disease; it was present in nine cases of my series. The most remarkable peculiarity is the variation in the extent of involvement. The angina may be associated with obliteration of a comparatively small branch, or with a most widespread involvement of all the vessels. In the younger subjects the process is a gradual endarteritis with narrowing, and even complete occlusion of the vessel. In older subjects, the arteries may be converted, as in John Hunter and in William Pepper, into "open bony tubes." In one instance of my series the vessels were calcified to their smallest branches. Four cases showed disease of the coronary arteries alone; five in connexion with aortitis. In looking over these notes one is astonished at the comparatively small extent of coronary tubing which is sufficient to carry on the myocardial circulation. Mr. G., aged 39 years, an extraordinarily vigorous, muscular man, after a day full of effort and strain, had read an important paper at a college society and died the following night in an attack. Not more than a third of his coronary vessels were in use. It has long been known that advanced coronary artery disease may be present without much disturbance of the function of the heart. There is not a clinician among us who could not furnish from his notes a dozen cases of this kind. A man may get on very comfortably with only the main branch of one coronary, practically a fourth of the whole system. A heart once in my possession showed almost complete obliteration of the left coronary, only a pin-point channel could be traced for a short distance. Of the right branch, the main division passing between the auricle and the ventricle was completely obliterated, so that the only one of full size passed in the posterior interventricular groove. The heart came from a large, very muscular imbecile, aged 36, an inmate of the Institution for the Feeble-Minded, at Elwin. I knew him well; a good-natured, helpful fellow, constantly employed in carrying about, and attending to, the more helpless children. He died suddenly one day in a fit. The coronaries are not endarteries in the sense of Cohnheim, and disease of their branches is not necessarily associated with angina.

(*d*) And in a few fatal cases no lesions whatever are found; we must accept the fact that angina pectoris may kill without signs of obvious disease in heart or blood-vessels. Such an instance has been reported by Dr. Bullard and myself.[3] The case was regarded by all who saw it as one of so-called functional angina. The patient, aged 26, was very strong and robust, devoted to athletics, and a heavy smoker. He had served in the United States Army, but was discharged in the spring of 1896 for attacks of angina. The chief feature was pain in the heart, and "awful cramps," as he described them, in his arms. The attacks were so severe that at times he became unconscious, and after one he was thought to be dead, and was about to be removed to the dead-house! The attacks were brought on by cold and exertion. The pain was evidently very severe, and in the major paroxsyms respiration would cease, and his pulse would become so feeble that he seemed to be dead. Only chloroform and morphia were of of any avail in the attacks. He had an extraordinary number of attacks in 1896–7; Dr. Bullard had notes of 105. In 1898 he was better and had not nearly so many attacks, and was able to be at work. On Nov. 27th at 11.30 he had an attack of great severity; at 12.55 the doctor gave him chloroform; the attack was very prolonged, and the muscles of the chest became fixed, and remained so; he had a series of paroxysms and died at 6.40 in the morning. Except a few pleural adhesions, there was nothing special to be noted. The heart weighed 14 ounces; the muscle and the valves were normal. Just above the ring the aorta measured not quite 6 centimetres, a small vess for a man of 5 feet 10 inches, weighing just over 13 stones. There was no disease except a flake here and there of atheroma. There was no thickening about the pericardium, and the sections showed no changes in the cardiac nerves.

PUMP AND PIPES.

The circulation as a whole may be compared to a vast irrigation system, with innumerable sub-districts of varied extent, under the control of local officers, but all under one central bureau, with which they are connected by telephone and an automatic signalling apparatus. The engine, pumping night and day, keeps a steady, uniform supply in the mains. The efficiency of the system depends upon the care with which the managers of the sub-stations regulate the flow to different plantations as occasion demands; the slightest disturbance in the most distant district is at once indicated by telephone to the central office, or in some instances automatically to the pump itself. Into certain vast irrigation areas with large sluice-ways all the water of the system can be diverted; and through carelessness of the men in control or through misinterpretation of a message from the head office, it sometimes happens that these sluice-ways are left wide open and the whole system is wrecked. Or strikes arise in local, outlying districts, the distributing mains are closed, and the pumping reservoir is flooded and permanently disabled. Or things go wrong in the central bureau—supplies are not forthcoming to keep up the plant, or there is litigation with neighbours, and the works are shut down, sometimes abruptly and without warning. What happens in a great irrigation plant happens also in the vascular system of the animal body, the mechanism of which, pump (heart), mains (aorta), sluices (arteries), and lakes (capillaries), is very much the same. Take two illustrations of its working. In Hill's experiment—hold a tame rabbit up with the forelimbs spread, and the gates of its splanchnic sluices will open so wide that the head office, pumping station, and whole irrigation system are wrecked in a few minutes. Try the same with the wild rabbit, whose splanchnic dam is under the control of trained officials—nothing happens. The pumping-engine itself is as sensitive as a galvanometer and has a marvellous mechanism for relieving and preventing any strain or tension on its machinery. Irritate with a probe, as in Stewart's experiment, the inner surface of the left ventricle, just enough to suggest or imitate tension, and automatically messages are sent, opening wide the most distant sluice-gates to prevent any strain on the pump itself. Or damage the main valve of

[3] Medical News, vol. lxxvii., p. 974.

the pump so that there is a leak with increased central strain, as in aortic insufficiency, and all the outlying territories open their sluice-ways to relieve the pressure. The circulation is maintained, equalised, and regulated by one working element—the muscle in the walls of its system, a peculiar, indeed a unique, type in the pump, ordinary unstriped fibre in the distributing channels. Both constituents, heart and arteries, are elaborately "wired" with nerves, which end about possibly in the muscle fibres, and there are peculiar end organs widely distributed (Paccinian bodies). Just as in the irrigation fields, pump and channels are connected by wires with the local and central offices of control, so the arteries and the heart are connected with centres, local and general, which act directly upon their muscular elements, by which the whole system is worked and regulated—an automatic set of fibres which keeps the head office constantly informed as to pressure conditions in the engine, a set which slows, and a set which hastens its action. Moreover, a complicated, subsidiary system coördinates the different parts of the heart ministered to by a tissue of special type, unlike the fibres of the heart itself or of the arteries. Not only does the muscular element maintain the circulation, but it keeps the vascular walls in a state of tension, a tonus or tautness which has an all-important influence in relieving the strain on the non-muscular elements. As Harry Campbell remarks in his recent Study of the Circulation,[4] "The greater the tonus of the muscular elements the more exclusively does the vascular strain caused by the blood pressure fall on them."

INVOLUNTARY MUSCLE PAIN.

Involuntary muscle pain has its peculiarities, and whether in artery, bowel, ureter, gall-duct, or uterus, comes in crises, storms, and outbursts. I have recently taken advantage of an unpleasant experience in my own person to observe the phenomena of these paroxysms in a ureter struggling with a calculus. Periods of complete freedom, extending from two to three, to eight or ten hours, attenuated with three types of disturbance of sensation—a dull, steady, localised pain, the situation of which could be covered with a penny. It could be imitated exactly by firm pressure with the handle of a knife, or, indeed, with a finger upon a bone, particularly upon that tender spot on the sternum just a little above the ensiform cartilage. Lasting for hours and unmoved it was fairly bearable. Now and then, when free from pain, there were remarkable flashes, an explosive sort of sensation, not actually unpleasant, and accompanied by a glow-like wave along the course of the ureter and out through the flank, as it were through the muscles. And then abruptly, or working out of the steady pain, came the paroxysm, like a twisting, tearing hurricane, with its well-known radiation, followed by the vaso-vagal features, the pallor, cold extremities, feeble pulse, sweating, nausea, vomiting, and in two attacks, a final, not altogether unpleasant period, when unconsciousness and the pain seemed wrestling for a victory reached only with the help of God's own medicine—morphia.

Any portion of the arterial system taken as a unit may present the phenomenon of involuntary muscle pain, and herein, I think, lies the key to the explanation of the anginal attack. The intermittency, the suddenness of onset, the steady, dull, enduring pain, and then the paroxysm, with its associated vaso-motor features, sometimes unconsciousness, and the radiations are paralleled in other involuntary muscle crises. Paralleled, but not equalled, and not often associated with the dangerous collapse symptoms, and rarely causing sudden death. And yet a man may die in renal or biliary colic, borne down in a vaso-motor storm, as happened in the only case of the kind I have seen.

CARDIO-VASCULAR PAIN.

What do we know about cardio-vascular involuntary muscle pain, and under what circumstances do we meet it? Like other viscera, the heart itself is insensitive to ordinary stimuli. You remember how this so amazed Harvey when handling the apex of the heart of the young Viscount Montgomery. Even his Most excellent Majesty, who studied the case with him, "acknowledged that the heart was without the sense of touch; for the youth never knew when we touched his heart except by a sight or the sensation he had through the external integument."

[4] THE LANCET, Jan. 15th, 1910, p. 193.

In most affections of the heart pain is conspicuous by its absence, particularly in the more serious maladies, so that it has almost become an axiom that "not much is the matter when a patient complains of his heart." Pericarditis may pass through all its phases without pain. Occasionally it is present in a marked degree, and it may be a special feature in the chronic mediastino-pericarditis.

In acute endocarditis pain is rarely present, and ulceration of valves or of the wall may proceed to a most extreme degree without any sensory disturbances. Of valvular lesions mitral disease is often associated with slight pain, particularly in children with greatly enlarged heart. And sometimes in women the pain is of great severity and persistence, but it rarely has the characters of true angina. There are a number of cases on my list with mitral lesion, stenotic or regurgitant, but, curiously enough, the only instance of attacks which I could call genuine angina pectoris in the stage of cardiac insufficiency occurred in a young girl of 11. And in this point I see that my experience coincides exactly with that of Nothnagel. On the other hand, lesions of the aortic ring are often painful, and attacks of true angina are common, particularly when the root of the aorta is involved.

Arterial pain is met with under many different circumstances, and may present all the features of angina. In the first place, external pressure directly upon the wall is associated with agonising pain. Those of us who as students took our turn in digital compression of the femoral artery for popliteal aneurysm have a lively recollection of the misery suffered by the poor patient.

Secondly, pressure from within; the pain caused by an embolus may be of the most terrific character. A man admitted to the Radcliffe Infirmary under Dr. Brooks—an old examination case of aortic insufficiency, with a loud, musical, diastolic murmur—had a sudden pain in his right leg, just below the popliteal space, and for days was in such agony that he had to have repeated hypodermics of morphia. As the swelling and pain subsided signs of an aneurysm became evident, and it was noted that the loud, musical murmur had disappeared. A calcified fragment whipped off from the aortic valve had torn the wall of the artery. Not only sharp emboli but the soft ones of ulcerative endocarditis cause intense pain. As I was going up the steps of the house of a patient, the diagnosis of whose trouble had wavered between typhoid fever and ulcerative endocarditis, I heard loud screams and found a young fellow in great agony, and he pointed to a spot below Poupart's ligament which he would not allow us to touch. He had embolism of the femoral artery, with subsequent gangrene of the leg. The intense colic of mesenteric embolism, such as we see in aneurysm, and occasionally in endocarditis, is of the same character, and it is diagnostic point between thrombosis and embolism of the cerebral arteries.

Thirdly, spontaneous tear of the arterial coats is associated with atrocious pain, with symptoms, indeed, in the case of the aorta of angina pectoris, and many instances have been mistaken for it. In this remarkable drawing which I pass round, of a split, fissured, and healed rupture of the internal coats of the aorta just above the valve, the patient was thought to have angina pectoris, and in the second attack, from which he died a year or more subsequently, a fresh split of the internal coats was found, which had ruptured into the pericardium.

Fourthly, as a result of extreme dilatation, distension, and stretching. Following the application of an Esmarch bandage, the arteries of the limbs dilate and throb, and there may be pain of a very intense character. In chilblains and in erythromelalgia the pain is probably arterial, and may be greatly aggravated with each systolic distension. In the excessive dilatation of the vessels following frost-bite the more rapid the dilatation the more intense the pain. As boys we had to give practical recognition to this point; if after a snowball fight anyone was foolish enough to put his cold hands into warm water he would be sure to suffer agonies of pain. An every-day cause of arterial pain is met with in aneurysm. In 132 cases of thoracic aneurysm the histories of which were carefully revised for this symptom pain was present in 104, and in 62 the trouble began with it. A feature of special interest to which attention has been called by many writers is the occurrence of attacks of angina pectoris as the first symptom. This happened in four cases in my series, and in every one of

them the anginal attacks disappeared with the increase in the size of the aneurysm. There are other mechanical causes of pain in aneurysm, but I think we all accept the fact that pain is a very constant feature in the early formation and growth of the sac. Stretching of the aorta without disease of its coats, as seen in the dynamic dilatation of aortic insufficiency and certain neurotic states, is not necessarily painful.

Fifthly, spasm of the arteries may cause severe pain. Slow, gradual contraction of the peripheral vessels due to cold is associated with a sense of numbness but not of actual pain. Scores of everyday vascular actions illustrate the same thing, and the radials may be contracted to obliteration of the pulse without any abnormal local sensations. On the other hand, there are types of arterial spasm accompanied with acute pain. Dubois, you may remember, referred the pain of hemicrania to angio-spasm. And our distinguished emeritus registrar, in his classical monograph on the disease, notes a number of instances and discusses this theory very fully. One does occasionally meet with an extraordinary degree of contraction of the temporal arteries during a paroxysm, but I have never seen an arterial distribution of the pain, nor are the vessels themselves sensitive.

The painful extremities in the various pathological states described as Raynaud's disease afford the best illustrations of disturbance of sensation as a direct result of angio-spasm. In a great many of the cases there is either obliterative endarteritis or the thrombo-angiitis of Buerger. But numerous observations show that spasm alone may account for all the symptoms. The paroxysmal character of the attacks, the intensity of the pain, the direct association with angio-spasm, suggest its vascular origin. It is an interesting point, too, that angina pectoris has been met as a complication of Raynaud's disease. In the case reported by Cleeman, a man, aged 62 years, had from his fiftieth year severe attacks of Raynaud's disease, chiefly in the hands, and usually in the winter season. Following several pronounced attacks of local asphyxia, and local syncope in the hands, he had one day a very severe paroxysm of angina pectoris; the pain lasted for two hours and was of such intensity that he was greatly prostrated. The association of migraine with angina pectoris, particularly the vaso-motor type, has long been recognised, and is discussed by Dr. E. Liveing; two of my patients had been great sufferers with typical migraine.

ARTERIAL SPASM.

Let us now consider in what conditions we actually see spasm of the arteries; and by spasm I mean a persistent contraction leading to ischæmia, with disturbance of function of the parts supplied. Raynaud's disease is, of course, the type of an angio-spastic affection. One does not actually see the arteries contract, but one may feel the gradual reduction in volume of the pulse, even to obliteration. One may feel a full, easily palpable radial contracted to a narrow cord, followed by a gradual blanching of the skin of the hands. The spasm may affect the smallest twigs, such as those distributed to the extreme tips of the fingers, or it may be the tip of one finger only. The spasm is not always painful, but it may be associated with intense pain, and I have noted in one or two instances that there is greater pain with the local syncope, and the reactionary intense hyperæmia, than with the cyanosis. Of late in so many instances of so-called Raynaud's disease arteritis has been discovered that is well to insist upon the fact that the most advanced necrosis may occur as a consequence of spasm in vessels apparently healthy. Russian Jews are subject to a very remarkable malady studied by Buerger of the Mount Sinai Hospital, New York. While similar in some features to Raynaud's disease, it differs anatomically in having widespread obliterative endarteritis, with thrombosis of the veins; indeed, the disease may begin in the veins, so that Buerger calls it "thrombo-angiitis." The same condition has been shown by Parkes Weber in several Russian Jews at the Clinical Section of the Royal Society of Medicine. Buerger writes me that in two cases of typical Raynaud's disease in which he performed amputation of the leg the arteries were found normal; evidence of exceptional value, as this observer has made a special study of the condition of the vessels in some 30 or 40 cases of thrombo-angiitis of the leg.

There is one place in which we can actually see spasm of the arteries associated with loss of function. In numbers of instances of amaurosis spasm of the papillary arteries has been noted by Priestley Smith and others. I have seen but one case myself—a man with small contracted kidneys and the usual associated vascular changes, became blind while walking from the out-patient department to the ward, and was unable to see for some hours. The retinal arteries on both sides were strongly contracted and I had the advantage of the confirmation of the observation by my colleague, Buller, the well-known ophthalmic surgeon. In Raynaud's disease a similar contraction of these vessels has been seen, originally by Raynaud himself, since then by a number of observers, and quite recently in an interesting case reported by Friedman.[5] In none of these cases has pain been mentioned as a symptom. I know of no other conditions in which we actually see angio-spasm with disturbance of the function.

And now let us leave the solid ground of observation for a few minutes. As I mentioned in my first lecture, the term "vascular crises" was introduced by James Collier in discussing the features of erythromelalgia, and it is a most useful term which admirably expresses the state of affairs in the recurring paroxysms of Raynaud's disease. It has been used with great effect by recent authors, particularly by Pal of Vienna, in whose monograph, "Gefässkrisen" (Leipsic, 1905), the whole question is exhaustively considered, more particularly in reference to its association with high tension and arteriosclerosis. The profession is at present riding on the top of a cardio-vascular wave, and it is impossible to approach questions without considering blood pressure and sclerosis. In Pal's hand the vascular crisis is a key to unlock many of the mysteries of disease in head, chest, and abdomen. Paroxysmal high tension we know with its remarkable phenomena—cardiac dyspnœa, cardiac pain, headache, uræmic symptoms, nausea, vomiting, and convulsions. No one who has seen much of blood pressure work can doubt that in patients with arterio-sclerosis these paroxysms play a very important part; but when we come to conditions of *local* high tension associated with contraction of the arteries, I confess that we are a little bit in the spray of the wave, and yet it may be used as a working hypothesis to explain a whole group of obscure conditions. As briefly stated, Pal's contention is: "Where the tension is produced by contraction in a definite vascular area, local consequences follow and dominate the picture. These are manifest chiefly by a peculiar painful sensation and local disturbance of function. General phenomena to a greater or less extent are manifest at the height of the tension." One is a bit staggered at the very free use which many writers make of the vascular crises, but it is a seductive theory and only the name is new. We have, I think, evidence that sclerotic arteries are specially prone to spasm. In many of Buerger's cases of thrombo-angiitis the symptoms were in part due to spastic contraction; in intermittent claudication vascular spasm plays a part, and one may actually see the foot get pale, as the patient begins to complain of pain and stops walking. We have really very little positive evidence of angio-spasm of the internal vessels. In a few remarkable cases of Raynaud's disease transient cerebral symptoms have occurred—aphasia, monoplegia, epilepsy, either at the time of or alternating with peripheral attacks. So transient has been the disturbance of function that it could be scarcely any other condition than angio-spasm. This was the view I took of the two remarkable cases which I reported some years ago. Now we have in arterio-sclerosis identical transient cerebral attacks for which it is scarcely possible to offer any other explanation. The condition, familiar to me for more than 25 years, was brought to my notice by the illness of a warm personal friend, who before his forty-fifth year was the subject of the most advanced sclerosis, with high tension. He had literally scores of attacks of transient paralysis, of monoplegia, asphasia, occasionally hemiplegia for 24 hours; and once as he got off the steamer after a trip to England he became paraplegic and remained so for nearly two days. The attacks are not always associated with very high tension. The cases are by no means uncommon, and a peculiarity is the extraordinary frequency of the attacks and their transient character. The question has recently been reopened in an interesting discussion on intermittent closure

[5] Friedman: American Journal of Medical Sciences, February, 1910.