A Clinical Lecture

ON THE

CAUSATION OF ANGINA PECTORIS.*

BY

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A PAROXYSM of angina pectoris is characterized by pain in the region of the heart. The pain is in many cases intense. During the attack the patient usually experiences a feeling of tightness and constriction in the chest; he may feel as if the chest were held in a vice; there may be a feeling of suffocation. The pain is usually accompanied by a feeling of alarm and dread, and in some cases by a terrible sensation of impending death.

The pain, which is usually first felt in the precordial or mid-sternal regions, usually radiates up to the left shoulder and down the left arm; in some cases the pain radiates through to the back; in some cases it extends up the left side of the neck; in exceptional cases the pain radiates still more widely. In some cases—but they are exceptional—the pain radiates down both arms, and in a few cases down the right arm alone, the left being unaffected.

After an attack of angina pectoris the skin over the parts in which the pain was felt during the attack is often hyperaesthetic and hyperalgesic. After a severe attack of angina pectoris the patient often for several days feels very much exhausted, and in some cases complains of special weakness—a certain degree of paresis—in the left arm, and, in the exceptional cases in which the pain extended to the right arm, it may be in the right arm. Dr. James Mackenzie thinks that the feeling of constriction in the chest is associated with and due to spasmodic contraction of the intercostal muscles.

Many different theories have from time to time been advanced to account for the manner in which the pain of angina pectoris is produced. In my book on the heart published twenty-five years ago (1884), I discussed the question in great detail. The theory which then seemed to me most satisfactorily to account for the mode of production of the pain I still believe the most satisfactory view.¹

The brilliant researches of Dr. Henry Head and Dr. James Mackenzie have thrown a flood of light upon our knowledge of pain in visceral disease, and have rendered clear many of the points which were formerly obscure with regard to the distribution of the pain in angina pectoris.

At the outset let me point out that any theory of the causation of angina pectoris, if it is to be satisfactory, must explain the facts: (1) *That in the great majority of cases of angina pectoris the pain radiates down the left arm;* (2) *that in some cases it radiates down both arms;* and (3) *that in exceptional cases, which are very rare, it is referred to the right arm only.*

The group of symptoms included under the term "angina pectoris" may be produced by, or perhaps it would be more correct to say associated with, a number of different organic lesions of the heart and the root and arch of the aorta; and in its typical and severe forms is apt to prove suddenly fatal.

In angina pectoris all degrees of cardiac pain are met with, though there seems to be a tendency in some men's minds to limit the term "angina pectoris" to those cases—comparatively rarely met with before the age of 40—in which the pain is intense. Cases in which "cardiac" pain occurs in young and neurotic subjects, and cases in which there is no discoverable organic disease of the heart or great blood vessels are often termed cases of pseudo-angina; but the term is not a good one; a better term is, I think, "functional angina pectoris."

The former variety, which very frequently proves fatal, is very often associated with "coarse" pathological changes in the coronary arteries, heart, or aorta, and very generally, I believe, with minute structural changes, usually degenerative in character, in the heart (cardiac muscle and

nerves) or blood vessels. The latter variety seldom, if ever, proves fatal, and often occurs without any "coarse" structural changes in the heart or vascular apparatus. The former may be appropriately termed the *organic* and the latter the *functional* form of angina pectoris.

This division, into a serious *organic* form and a comparatively trivial *functional* form, is of great practical and clinical utility, and may be safely adopted, provided that it is clearly understood that it is sometimes difficult or impossible to separate the two forms at the bedside—that is to say, whether the cardiac pain—the angina pectoris—is serious and due to organic disease, or whether it is, comparatively speaking, trivial and due to functional derangement.

In cases of true (organic) angina pectoris characterized by the occurrence of attacks of most severe heart pain, slight attacks of cardiac pain are of common occurrence; in some cases of organic angina pectoris the pain is never severe and widespread; in many cases of grave organic disease of the heart which prove fatal, slight angina pain is apt to occur, and in some of these cases the patients never have a typical and severe attack of angina pectoris.

In the organic cases physical examination may fail to reveal any evidence of organic disease either in the heart or great blood vessels. This is, of course, only what one would expect when one remembers that it is in many cases extremely difficult or impossible to detect by means of physical examination the organic lesions and structural changes in the heart and great blood vessels with which the organic form of angina pectoris is apt to be associated. Atheroma of the coronary arteries, fatty and fibroid degeneration of the heart, small, deep seated aneurysms of the root of the aorta, may be unattended with any definite and characteristic physical signs. Though in some cases we may be able to diagnose these conditions with practical certainty, in other cases we cannot do so. I have met with more than one case of apparently slight angina pectoris in which I could detect no definite physical signs of organic disease of the heart or aorta in which the patient has died suddenly. Vice versa I have seen cases of angina in young subjects in which the pain was very severe, and in which the diagnosis of organic angina pectoris would undoubtedly have been made if the patient had been older, or if there had been any obvious cardiac or aortic lesion, but in which the subsequent course of the case—the complete recovery of the patient and the maintenance of good health for years after recovery—showed that the angina was functional. Some twenty years ago I met with a case in hospital practice which greatly impressed me, and which showed the impossibility of differentiating the functional and organic forms. The patient was a woman, aged 36; there was no evidence of organic disease either of the heart or great blood vessels, the kidneys were normal, and the radial arteries not thickened; the angina pain of which she complained was confined to the region of the heart, and seemed to be functional; the patient was a nervous woman, and had had a great deal of domestic worry, which appeared to be the exciting cause of the attack for which she consulted me; she had also had some fits which from her description appeared to be hysterical in character. I gave a favourable prognosis, thinking that the angina was functional. This diagnosis was based upon the facts that the patient was a nervous woman, that her age was only 36, that the exciting cause of the attack appeared to be worry, that the pain was not very severe, and that the most careful physical examination failed to detect any evidence of organic lesion or structural change in the heart, great blood vessels, peripheral arteries, or kidneys. Some weeks after I saw the patient she had another attack while sitting at dinner, and she died in it.

It must also be remembered that the functional form of angina pectoris may, and I believe not infrequently does, occur in later life, while the organic form may occur in young subjects.

In order to understand the phenomena of angina pectoris it is necessary to refer briefly to the construction, so to speak, of the afferent (? sensory) nerve apparatus of the heart.

In conditions of health, and when the heart is contracting quietly, we are unaware of the action of the organ. It is only, in fact, when the cardiac action is markedly

* The cases illustrative of the lecture have been omitted.

deranged, either as the result of temporary and functional or permanent and organic causes, that we are conscious of any cardiac sensations. We become conscious of the cardiac action when the afferent (? sensory) nerve terminations in the heart and adjacent structures are more powerfully stimulated than they are in normal tranquil action of the organ; under such circumstances an impression is conducted to, and registered by, the sensory perceptive centres in the cerebrum.

The exact course of the afferent (? sensory) nerve fibres, which convey impressions from the heart to the cerebrum, has not yet been fully determined. In some of the lower animals—the frog, for example—sensory impressions seem to be conducted upwards by a special branch of the vagus, which has been termed the sensitive nerve of the heart; but it seems certain that in man the channels of sensory conduction are by no means so limited. The phenomena of angina pectoris seem, in fact, to prove that the cardiac branches of the sympathetic contain sensory fibres—fibres stimulation of which, if excessive in degree, excites pain. It would appear that in man afferent (? sensory) impressions are carried from the heart by the sympathetic fibres connected with the cardiac plexus, probably also, as in the frog, by the cardiac branches of the vagus.

Afferent (? sensory) impressions, passing to the cerebrum from the heart through the sympathetic, pass via the spinal cord, while afferent impressions passing from the heart through the vagus join the nerve centres at the medulla oblongata.

Dr. Henry Head gives the following tabulated statement of the sensory supply of the heart:²

Transverse arch of aorta	...	Inferior laryngeal segment.
Ascending arch of aorta	...	3rd and 4th cervical, 1st, 2nd, and 3rd dorsal (? 4th dorsal).
Ventricle...	...	2nd, 3rd, 4th, and 5th dorsal (? 6th dorsal).
Auricle	...	5th, 6th, 7th, and 8th dorsal (? 9th dorsal).

Dr. Gaskell informs me that the afferent nerves of the heart have their nerve-cell stations in the posterior root ganglia of the thoracic nerves (2nd to 5th), largely in the 2nd thoracic, and that possibly there are a few in the 1st thoracic.

The exact course of the fibres which conduct afferent cardiac impressions upwards through the spinal cord, and the exact position of the perceptive cerebral centre which receives such impressions, are not yet definitely known.

The pain of angina pectoris may theoretically result from excessive stimulation or irritation of any part of the sensory nerve apparatus connected with the heart; for, in accordance with the law of "eccentric projection," the pain which results from stimulation or irritation of a sensory centre is referred to that part of the periphery from which the sensory conductors or sensory centres are in the habit of receiving impressions, rather than to the point of irritation itself. Theoretically, therefore, we may suppose that it may be due to:

1. Irritation of the sensory nerve terminations in the wall of the heart itself (including under this term the pericardium, myocardium, and endocardium).
2. The sensory nerve-fibres comprised in the coronary and the superficial and deep cardiac plexuses.
3. The sensory conductors which connect these plexuses with the sensory perceptive centres in the cerebrum. (In the case of the sympathetic conductors the seat of irritation might theoretically be placed in (a) the branches connecting the cardiac plexuses with the spinal cord, including the posterior roots and posterior ganglia; (b) the spinal cord itself; (c) the conductors above the spinal cord—that is, between the spinal cord and the perceptive centre.)
4. The perceptive cerebral centres themselves.

Possibly the cardiac pain may be produced in most of these ways; but, so far as I know, there are no actual facts to show that angina pectoris is ever due to organic lesions in the spinal cord or brain. The so-called "cardiac crises" of tabes, in which angina-like attacks may occur, are perhaps an exception to this statement, but whether the cardiac crises of tabes are due to the lesion in the cord, or to a lesion in the posterior roots or the peripheral nerves connected with the heart, we do not definitely know. In some of the functional cases, the so-called "pseudo-angina"

attacks met with in hysterical patients, for example, the cerebrum is probably the seat of the disturbance.

Before proceeding to consider the exact manner in which the pain of angina pectoris is produced, let us turn for a moment to the records of *post-mortem* examinations, and see what lesions have been actually found after death in cases of angina pectoris.

In a large proportion of typical cases of angina pectoris (the form which I term organic, and which is apt to prove suddenly fatal) the coronary arteries are diseased (atheromatous). In some cases the heart muscle is degenerated (fatty, fibroid, etc.). In some cases the cardiac valves (and more especially the aortic valves) are diseased; in fact almost all forms of cardiac lesion have been found. In a considerable proportion of cases the base of the aorta is dilated or aneurysmal. In a few cases pericarditis has been observed. Very frequently the peripheral blood vessels, as well as the aorta, are atheromatous.

In some cases of angina pectoris changes have been shown to be present in the cardiac nerves or ganglia. Probably in some of the cases of atheroma of the coronary arteries in which angina pectoris does occur, the coronary nerves (which are so closely connected in their course on the exterior of the heart with the branches of the coronary arteries) are implicated, just as the cardiac nerves which ramify over the arch of the aorta are affected in some cases of atheroma and chronic arteritis of the base of the aorta.

In some cases of angina pectoris the heart has been said to be healthy, but structural changes are probably present, either in the heart or arteries or in both, in all cases of angina pectoris which prove fatal.

Let us now endeavour to determine the exact manner in which the pain is produced in cases of angina pectoris. In most cases it is, I believe, due to *irritation of the afferent (? sensory) nerve terminations in the wall of the left ventricle*—including under the term "wall" the pericardium, myocardium, and endocardium.

The exact manner in which the cardiac nerve terminations are irritated has not yet been definitely determined. The view which seemed to me most probable when I published my book on the heart was that the irritation is due to spasmodic contraction of the muscular fibres, or rather of some of the muscular fibres of the left ventricle—that the cardiac pain is, in fact, similar to the violent pain which is experienced in the calf muscles when they are spasmodically contracted as in ordinary cramp, and similar to the severe pain which is experienced in cases of renal, hepatic, and intestinal colic. We know that cramp in the calf is most apt to arise when the muscle is exhausted and fatigued; all football players must be well aware of this fact.

Gaskell has shown that when the vitality of the cardiac muscle is impaired (by exhaustion, by injury, by malnutrition, etc.) it loses its power of rapid contraction, and contracts with a prolonged tonic contraction in the same way as unstriated muscle. Again, clinical experience has shown that muscular effort and conditions which cause increase of the blood pressure in the peripheral arteries—that is, *conditions which throw an increased strain on the left ventricle*—are in many cases the exciting cause of an attack of angina pectoris. In many cases the exciting cause of an attack is walking uphill, walking against a wind, etc.; in some cases, walking on the flat excites an attack; in some cases (in which we may presume the heart muscle is greatly degenerated or the blood supply to the heart very imperfect) the attack may come on without any apparent exciting cause.

An attack of angina pectoris—the pain of angina pectoris—is, in short, an external expression of the inability of a weakened or degenerated left ventricle to cope with the blood pressure in front of it; or, as Dr. Henry Head puts it: "Pain, whenever it occurs in organic

* In three cases examined by Lancereaux, Peter, and Bazy, a distinct change was found in the branches of the cardiac plexus passing over the root of the aorta. Peter, for example, found in one of his cases slight dilatation of the base of the aorta, increased vascularity and thickening of the aortic coats, and evidence of old pericarditis at the base of the heart. The branches of the cardiac plexus, which were carefully dissected out from the fibrous adhesions surrounding the root of the aorta, were found on microscopical examination to be in a condition of chronic inflammation, the nerve tubes were separated by masses of connective tissue containing numerous nuclei; some of the nerve tubes were strangled, as it were, by this fibrous tissue, their myelinated sheaths ruptured and transformed into an amorphous fatty mass (*Traité clinique et pratique des maladies du cœur*, p. 673).

heart disease, is a warning and an intimation that rest is required."³

It is difficult to conceive that all of the muscular fibres of the left ventricle could be spasmodically contracted without a fatal result; if spasmodic contraction of the muscular fibres of the left ventricle is the cause of the pain, it must be partial. This statement does not imply that spasm of the muscular fibres of the heart is *per se* attended with pain, but that it produces irritation of the afferent (sensory) nerves, which results in the production of pain.

The view that spasmodic contraction of the muscular fibres of the left ventricle is the cause of the irritation of the sensory cardiac nerves and of the pain in angina pectoris is not accepted by many authorities. Dr. James Mackenzie thinks that in angina pectoris the pain is not felt in the heart itself, but in the thoracic wall. He thinks that the pain in the region of the heart is a referred pain, exactly similar in character to the pain in the left arm which is such a characteristic feature of angina pectoris. Dr. Mackenzie does not agree with the view that spasmodic contraction of the cardiac muscle is the cause of the irritation. He says:

Such considerations lead to the conclusion that angina pectoris arises from certain conditions of the muscular substance of the heart, when the contraction meets with a resistance greater than it can easily and efficiently overcome, whether a fairly strong muscle struggles against an increased resistance (as when there is great peripheral resistance or a narrowed aortic orifice), or when a weak or degenerated muscle has opposed to its contraction a normal or even a lowered pressure, but a pressure greater than the weakened muscle can readily overcome. . . . From this line of reasoning I would suggest that angina pectoris is an evidence of exhaustion of the function of contractility. . . . The fundamental functions of the heart muscle correspond to those of other involuntary muscles that form the walls of hollow organs, these functions being modified to suit its special work. Like the other viscera, the heart is insensitive when stimulated in a manner that provokes pain when applied to the tissues of the external body wall. I may point out that a prolonged strong contraction of a hollow organ can produce pain, and that this is undoubtedly the cause of the severe pain associated with renal calculus, gall stones, spasm of the bowel, and uterine contractions. Can the heart give rise to pain in a similar manner? On account of the modification of its functions, the heart cannot pass into a prolonged state of contraction. Immediately it contracts, the function of contractility is abolished, and the muscle passes at once into a state of relaxation, and for this reason the pain cannot be produced by a "spasm of the heart." But I suggest that the heart muscle may produce pain when it is confronted with work greater than what it can readily overcome—a condition which produces strong peristalsis and pain in other hollow viscera. But the pain in the heart arises by a slightly different mechanism. A skeletal muscle will contract in obedience to stimulation of a sensory nerve going to the spinal centre of its nerve, if a stimulus of sufficient strength be applied. If the stimulus is too weak no contraction follows, but, if this weak stimulus be frequently and rapidly repeated, then the muscle contracts in accordance with the law of the summation of stimuli. I suggest that the heart muscle induces pain on the principle of summation of stimuli. If we minutely study our cases we shall find that the pain rarely arises at the first exposure of the heart to the effort that induces the pain; sometimes effort has been undertaken a few minutes before the pain comes on, and in certain cases it may not come on for hours after the causal exertion has ceased. From such observation we can infer that the heart muscle was exhausted by the exertion, and so great was the exhaustion of the reserve force that the heart was unable to regain its reserve with cessation of effort, so that the exhaustion persisted till it culminated in an attack of angina pectoris.⁴

Dr. Henry Head believes that the cause of the irritation of the sensory nerves of the heart and of angina pectoris is increased tension in the cavity of the left ventricle. He says:

Now, it is this distension at the moment when the walls of the cavity are at rest that is most pernicious, and it is this condition above all others that is most liable to lead to referred pain.⁵

And again:

Thus, in conclusion, it would seem that the most potent conditions for the production of referred pain are: First, the maintenance of considerable tension within a cavity of the heart, accompanied by, secondly, a sudden accession of tension (owing to regurgitation) at the moment when the walls of the cavity are dilating after systole.⁶

Possibly in some of the cases of angina pectoris in which the coronary arteries are atheromatous the degenerative process, which commenced in the inner coat of the coronary arteries (endarteritis deformans), has extended

to the outer coat and implicated the coronary nerves. Under such circumstances the terminal nerve fibres in the wall of the heart would, we may theoretically suppose, be in an unusually irritable condition.

Now, taking all these facts in connexion, we may, I think, with some probability theorize that in many cases of angina pectoris the sequence of events is as follows:

First, a sudden strain is thrown upon the left ventricle, either as the result of muscular effort or an increase of the peripheral resistance due to contraction of the peripheral arteries, the result of some external condition (sudden effort, exposure to cold, mental agitation, etc.), some reflex impulse arising within the body, or changes arising in the central nervous system (vasomotor centre). The strain or increased blood pressure may be actual or merely relative in proportion to the weakened left ventricle. In some cases in which the cardiac muscle or cardiac nerves are diseased, the attack arises independently of any muscular effort or sudden increase of the peripheral resistance. Under such circumstances, a strain on the left ventricle, which under ordinary circumstances would not be attended with cardiac pain, would probably be sufficient to produce the condition.

Secondly, in consequence of the strain (either actual or relative) which is thrown on the cavity of the left ventricle, or rather those fibres of the left ventricle which are degenerated, either as the result of imperfect blood supply (disease of the coronary arteries) or of degenerative changes in the cardiac muscle, irritation of the sensory cardiac nerves is produced. This irritation is attended with severe pain (angina pectoris).

The exact cause of the irritation of the sensory cardiac nerves is not clear, and this seems to me to be the point with regard to the pathology of angina pectoris which needs to be cleared up; perhaps it is spasmodic contraction of some of the muscular fibres, perhaps increased tension in the cavity of the ventricle.

Thirdly, this irritation of the terminal branches of the afferent (? sensory) nerves in the wall of the left ventricle (including under this term the pericardium, myocardium, and endocardium), is reflected, via the sympathetic branches of the cardiac plexus, distributed to the left ventricle and the spinal cord (left side of the upper dorsal segments), in the form of pain, to the periphery.

Now we would naturally expect that the reflex impression would be first reflected and referred to those parts of the periphery which are more immediately connected with the spinal centres to which the cardiac branches of the sympathetic distributed in the wall of the left ventricle pass; and that when the irritation was extreme, the stimulus might extend to other and more distant masses of spinal grey matter, and be reflected to other and more distant parts of the periphery. And such is in fact the case. In most cases of organic angina pectoris the pain radiates up to the left shoulder and down the left arm, sometimes to the tips of the fingers; it not infrequently passes up the left side of the neck; in exceptional cases it may pass down the right arm.

The radiation to the left shoulder and left arm is so constantly observed in typical cases of angina pectoris that it has been by some writers supposed that the primary lesion is situated in the spinal cord itself. Anstie, for example, thought that angina pectoris was probably due to "a mainly unilateral morbid condition of the lower cervical and upper dorsal portion of the cord, liable, of course, to be seriously aggravated by such peripheral sources of irritation as would be furnished by diseases of the heart, and especially by diseases of the coronary arteries." But with this opinion I cannot agree. In no case of angina pectoris, so far as I am aware, has a lesion of the spinal cord been found, and it is extremely difficult to believe that any spinal lesion could be so constantly unilateral—the very fact, in short, which Anstie advanced against the radiation of the pain outwards from the heart, namely, the unilateral character of the brachial pain, seems to me strongly opposed to his view.

This explanation is in accord with, and is in fact corroborated by, the facts (1) that the exciting causes of angina pectoris are increased muscular effort and conditions which produce increased pressure in the peripheral arterial circulation—that is, conditions which throw an increased strain (either actual or relative) on the left ventricle; (2) that in

many cases of angina pectoris the paroxysm is at once relieved by rest (stopping to rest while going uphill) and remedies (nitrite of amyl, etc.) which rapidly reduce arterial tension and take off the strain from the left ventricle; and (3) that in some cases of angina pectoris the attacks of angina disappear with the development of mitral regurgitation.

But further—and this is a very important point which for many years I have emphasized in my teaching—in the exceptional cases of angina pectoris in which the pain radiates to both arms or to the right arm alone, the “coarse” lesions, which are associated with and which are apparently the cause of the pain, often (? always) involve the root of the aorta or the aortic arch. I have met with a number of clinical cases which seem to corroborate this view.

A number of different theories may be advanced to account for the fact that in some exceptional cases of angina pectoris the pain, instead of radiating to the left arm, passes down both arms or down the right arm only.

The most satisfactory explanation is, in my opinion, that which I have just stated—namely, that in the exceptional cases of angina pectoris in which the pain radiates to both arms or to the right arm only, the irritation which causes the pain has its point of origin outside the heart—that is, that the irritation is due to a lesion, for example, of the root or arch of the aorta, or of the afferent (? sensory) nerves in the outer coat of the aorta, and not, as in the ordinary run of cases of angina pectoris, in the wall of the left ventricle. This view is corroborated by the fact that in many of the exceptional cases of angina pectoris in which the pain radiates to both arms or to the right arm only, a lesion either at the root of the aorta or of the aortic arch (aneurysm, atheroma of the root of the aorta, aortitis or periaortitis, aortic regurgitation, pericarditis) is actually present. I have seen quite a number of cases of this kind, and from time to time brought them before my clinic, and explained to the students their great interest and importance.

At the last annual meeting of the British Medical Association an important discussion on angina pectoris took place. Sir T. Clifford Allbutt, who opened the discussion, advanced the theory, which he had first propounded in the year 1894, that “the origin of angina pectoris was not in the heart but in the supra sigmoid portion of the aorta and its investments.”¹ In the article on angina pectoris in my *Diseases of the Heart* I stated that disease at the root of the aorta and of the nerves ramifying over the root of the aorta seems in some cases of angina pectoris to be the cause of the pain. But that disease at the root of the aorta, or stretching of the root of the aorta, is the usual cause of angina pectoris, as Sir T. Clifford Allbutt supposes, seems to me to be contradicted by the fact that in the great majority of typical cases of angina pectoris the pain is reflected to the left shoulder and left arm. It is difficult, I think, to conceive that irritation arising in the aorta above the sigmoid valves in the way Sir T. Clifford Allbutt supposes could be so constantly reflected to one (the left) side of the body; while the fact that in many of the exceptional cases of angina pectoris in which the pain is reflected to both arms or to the right arm only, my clinical and pathological observations show that the root or arch of the aorta is diseased is, I think, of great significance. It is easy to conceive that when disease at the root of the aorta is the cause of angina pectoris the pain would be reflected to both arms, since in these cases the afferent nerves from both ventricles or from the aorta itself (passing to both sides of the spinal cord) would be likely to be implicated; and that in some cases of aneurysm of the aortic arch or local disease of the aortic arch the nerves passing from the right side of the heart or the right side of the aorta to the right side of the spinal cord might alone be implicated; this would explain the reflection of the pain to the right arm alone.

Another explanation of the radiation of the pain to both arms is that in the cases of angina pectoris in which this occurs the peripheral irritation has been sufficiently severe to pass over to the opposite (right) side of the spinal cord, and to be reflexly directed (referred) to the right as well as to the left side of the body (right as well as the left arm).

Another explanation of the radiation of the pain to the

right arm is to suppose that the nerve terminations in the wall of the right ventricle have become irritated.

It has been suggested that the paroxysm of angina pectoris can be produced by a sudden diminution of the blood supply to the heart itself. According to this view, which I mentioned in my *Diseases of the Heart*, the coronary arteries become spasmodically contracted and cause a sudden diminution of the blood supply to the heart.

The intermittent claudication view, which is a modification of the anaemic theory (but which does not necessitate the supposition that there is spasmodic contraction of the coronary arteries), is more probable.

The phenomena which characterize the remarkable condition which has been termed “intermittent claudication,” or intermittent limping (angina cruris), suggest that angina pectoris may be produced when increased muscular effort is demanded of a left ventricle which is imperfectly supplied with blood (perhaps as the result of spasmodic contraction of some of the muscular fibres).²

In intermittent claudication the arterial blood supply to one or both of the lower extremities is interfered with by obliterative arteritis, etc.; so long as the parts are at rest there are, or may be, no symptoms; but muscular efforts, which demand a copious supply of arterial blood, are attended with loss of motor power, stiffness, cramps and spasms, and, it may be, *acute pain* in the muscles of the affected part. If the patient rests for a time these symptoms pass off, and so long as muscular effort is not demanded the patient remains (apparently) well.

Now the most constant pathological change which is met with in fatal cases of angina pectoris is atheroma of the coronary arteries. In many of these cases the blood supply to the heart is defective, and degenerative changes are present in the muscular tissue of the heart. Under such circumstances it seems not unreasonable to suppose that when a sudden strain (whether actual or relative) is put upon the left ventricle, weakness and overdistension or spasmodic contraction of the muscular fibres or some of the muscular fibres of the left ventricle may occur, and an attack of angina pectoris may be developed, just as spasmodic contraction of the muscles and pain in the leg may occur in cases of intermittent claudication as the result of muscular effort. This seems to be a plausible explanation of the usual (typical) cases of angina pectoris. It accounts for the frequent association of angina pectoris with disease of the coronary arteries, for the occurrence of the angina pains after muscular effort and other things which throw a strain (either actual or relative) on the left ventricle, and which demand increased effort from the muscular fibres of that cavity.

The organic form of angina pectoris is often met with in gouty subjects, and is popularly included with other conditions under the term “gout of the stomach.” Gout, it will be remembered, is one of the conditions which produce atheroma, and therefore disease of the aorta and coronary arteries. Further, in gouty subjects increased blood pressure, whether actual or relative to the strength of the left ventricle, which is such an important exciting cause of an attack of angina pectoris, is, of course, extremely common, more particularly when the kidneys are cirrhotic.

REFERENCES.

- ¹ *Diseases of the Heart*, page 672, et seq. ² *Brain*, vol. xix, p. 225.
- ³ *Ibid.*, p. 226. ⁴ *Diseases of the Heart*, pp. 39 and 40. ⁵ *Brain*, vol. xix, p. 215. ⁶ *Ibid.*, p. 224. ⁷ *Lancet*, 1909, vol. ii, p. 337. Sir T. Clifford Allbutt's remarks are reported in full in the *BRITISH MEDICAL JOURNAL*, 1909, vol. ii, p. 1122.

* I have discussed the subject of intermittent claudication and published cases illustrative of this condition in my *Clinical Studies*, vol. vi, p. 317.

THE Pasteur Institute of Paris has presented to the Rockefeller Institute for Medical Research of New York a replica of the bronze bust of Louis Pasteur, in recognition of assistance rendered during the recent epidemic of cerebro-spinal meningitis which prevailed in France.

DR. PARTROU has founded a vegetarian society in Roumania, with branches all over the country. The society issues a journal, the aim of which is to make the advantages of vegetarianism known to the people. It has also established at Bucharest a restaurant which is managed on vegetarian principles.