

Angina Pectoris With Special Reference to Its Mechanical Causes*

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Angina pectoris, while a definite clinical entity, is not a specific disease in the accepted sense. The name is applied to a syndrome, the most outstanding feature of which is paroxysmal attacks of severe pain, usually pectoral in location, accompanied by intense anxiety and apprehension.

The incidence of angina pectoris seems to be increasing. This may be due to improvement of diagnostic methods so that more obscure cases are now detected than previously, to the lengthening of the average length of life, and to the fast pace of modern civilization.

Some clue as to the part played by the nervous system in the etiology of the disease may be recognized from its high incidence in the intellectual groups. The disease is comparatively rare in the lower strata of society, especially among those individuals engaged in physical labor. Highly emotional races are especially prone to the disease. The highest incidence of angina pectoris is found in physicians, lawyers, and those holding responsible executive positions.

There is another indication that one type of angina pectoris may be purely a functional state resulting from deranged nerve control. It is the frequent disclosure at autopsy of angina victims that there exists no demonstrable pathology in the heart or the aorta. Although diseased coronary arteries are the most frequent post-mortem finding, there seems to be no constant relation between the severity of the attacks and the extent of vascular damage. Extensive sclerosis of the coronaries is often found without any history of precordial pain.

The syndrome of angina pectoris is also found with a considerable number of other pathological conditions. These include aortitis, insufficiency of the aortic valve resulting in decreased coronary circulation, coronary thrombosis or embolism, atherosclerosis and arteritis of the coronaries, the myocarditis of rheumatic fever, myocardial degeneration, and myocardial fibrosis. Cardiac pain may also result when a burden is thrown upon the heart in the presence of anemia or a rarefied atmosphere.

Several theories have been offered to explain the immediate cause of the anginal pain. Spasm of the myocardium was formerly believed to be the cause, but the paroxysm is not accompanied by any change in the rhythm of the heart which would indicate the existence of spasm. Recently there has been advanced and widely accepted the theory of myocardial anoxemia: that the oxygen supply to the heart is not sufficient to fulfill the requirements of the myocardium.

Wayne¹ has shown that when skeletal muscle is repeatedly contracted without sufficient circulation to supply the oxygen needed to meet its metabolic requirements, pain is soon felt. Wayne believes that muscular activity produces metabolic products normally removed by oxidation, which, when circulation is impeded, stimulate the sensory nerves with the resulting sensation of pain.

In coronary occlusion, which produces pain identical with that of angina, there is certainly an ischemia of a part of the myocardium. It has been noted that if a load is placed upon the heart when the oxygen carrying power of the blood is diminished by a severe anemia, angina-like pain results. Coronary thrombosis, coronary sclerosis, and aortic regurgitation, all of which have been found frequently when angina victims are autopsied, likewise interfere with a normal oxygen supply to the heart.

It is generally accepted that a spasm of the coronary arteries, by decreasing their blood carrying capacity, will likewise produce a myocardial ischemia with resulting pain. Such a vasospasm is considered to be the cause of angina pectoris in the absence of any cardiac pathology. In this connection it has long been known that anginal pain can be stopped during the paroxysm by the administration of vasodilators such as amyl nitrite and nitroglycerine. Fifty per cent of all angina pectoris cases are of this type, which we would expect to result reflexly from osteopathic lesions, and the type in which osteopathic manipulation is most useful².

The heart and the coronary arteries receive nerves from both the parasympathetic and the sympathetic nervous systems. Connector fibers arise in the dorsal nucleus of the vagus trunk to end in nerve cells in the SA and AV nodes. The vagus exerts a continuous restraining action upon the rate of the heart. Stimulation of the vagi results in a diminution in the force of myocardial contraction, conductivity through the bundle of His is impaired, and the output of the heart is reduced. As the blood pressure falls, the volume of blood entering the coronary arteries is decreased.

The sympathetic nerves to the heart belong to the thoracolumbar division of the involuntary nervous system. Fibers arising in the lateral horn of the first five thoracic segments enter the ganglionated cord and run to the inferior, middle, and superior cervical ganglia. The postganglionic fibers form the cardiac nerves which supply the SA node, the AV node, and the bundle of His. In addition to this, there are thoracic cardiac nerves which form direct connections between the upper four or five thoracic ganglia and the heart³. Stimulation of the sympathetic fibers to the heart causes a quickening of the rate and an increase in the force of contraction of both auricles and ventricles.

*Essay judged the best of those contributed by students at the Chicago College of Osteopathy in contest among all students in approved osteopathic colleges during 1937. [See p. 198 of THE JOURNAL for January containing announcement of prizes offered to both undergraduates and graduates in 1938.—Editor]

The coronary vessels are richly supplied with both vagal and sympathetic nerve fibers, the larger branches being innervated about equally by the two types of nerves. This would seem to indicate that proper operation of the coronary vasomotor apparatus is dependent upon a balance of sympathetic and parasympathetic fibers. Recent work indicates that the coronaries are dilated by sympathetic impulses and constricted by vagal⁴. Stimulation of the peripheral end of the cut vagus in an animal under the influence of atropine, which abolishes vagal action on the heart itself, reduces the flow from the coronary veins⁵.

The administration of adrenaline results in an increase in coronary flow of 85 per cent⁶. Anoxia increases the coronary flow greatly. Reduction of the oxygen saturation below 20 per cent causes a maximum dilation of the coronary vessels⁷. The obvious fact that this safety mechanism cannot be operating properly in cases of angina pectoris indicates the importance of proper balance of the two nerve supplies to the coronaries. Such a balance is readily upset in the presence of either visceral or somatic pathology.

The impulses giving rise to the pain of angina pectoris pass from the heart by way of the inferior and middle cardiac nerves and the thoracic cardiac nerves to the corresponding white rami and posterior nerve roots. Pain is apparently not transmitted through the vagus. That this pain is referred to the precordium and left arm in angina pectoris is explained by Mackenzie's theory of viscerosensory reflexes. Mackenzie conceived that afferent impulses carried from diseased viscera by sympathetic fibers set up an irritable focus in the segments of the cord which they entered, and thus excited cells which are accustomed to receive impulses from the somatic structures supplied by those segments⁸.

It has long been a challenge to osteopathic research workers to demonstrate the converse of Mackenzie's theory. If afferents from diseased viscera can set up an irritable focus in the cord which distorts normal sensations from somatic structures, it should be equally true that abnormalities of somatic structure give rise to a like focus of irritability capable of altering the efferent impulses to visceral structures. If this be true, its results should be most critical in those structures, of which the coronary arteries are only one of many, whose proper functioning is dependent upon a delicate balance of sympathetic and parasympathetic nerve impulses. To prove either theory conclusively by other than circumstantial evidence seems to be beyond the scope of methods so far devised. We know that the phenomenon of referred pain does exist, but it is more difficult to secure conclusive evidence of the reverse reflex, the somatovisceral reflex, in operation. Proof that somatic abnormalities can produce visceral pathology is a large and important step in the scientific proof of the osteopathic concept.

Recent studies of angina pectoris by several eminent cardiologists seem to offer a means of studying and measuring the part that somatic abnormalities in the form of osteopathic lesions may play in the production of the syndrome of angina pectoris.

Katz and Kissin⁹ and Levine and Levine¹⁰ have utilized lead IV of the electrocardiogram in studying a considerable number of cases of angina pectoris. From this work they find that absence of the Q wave in this lead or a Q wave of 2 mm. or less either with or without an accompanying inversion of the T wave (i.e. above the isoelectric line) is recorded in a large percentage of these patients. They conclude that while these findings are not absolutely diagnostic of angina pectoris, they are at least suggestive. In lead IV one electrode is placed over the left sternal border between the third rib and the apex and the second electrode on the left leg.

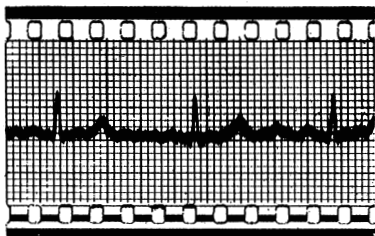
There seems to be no report in osteopathic literature upon the progress of any cardiac patients under osteopathic care as measured and recorded by electrocardiograms. If osteopathic lesions can reflexly decrease coronary flow, it would appear that the correction of such lesions should produce an immediate response in lead IV. If such is the case, it would indicate that there is a mechanical background to many cases of angina pectoris and that osteopathic corrective treatment is beneficial in these cases.

The problem presents such interesting possibilities that W. G. Richmond, D.O., and the writer very recently decided to experiment with it in the clinic of the Chicago College of Osteopathy. So far there has been opportunity to little more than start the work and the mere mention of it at this time is certainly open to criticism. However, since early results indicate that the field may be such a fertile one, and since there seems to be no mention in osteopathic literature that manipulative treatment has ever been observed to change an abnormal electrocardiogram at all, the work is mentioned here. It is hoped that in this way others may be interested in studying the problem.

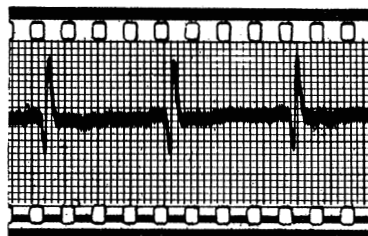
Electrocardiograms were first run on several students in the college and they showed average normal tracings. Any upper thoracic and cervical joint lesions found were then corrected. After the subject rested for a half hour, the electrodes were reapplied and another electrocardiogram was run. No difference except minor changes in rate could be detected between the record made before and that made after the treatment.

A patient, (A), entered the clinic with a complaint of attacks of pectoral pain. An electrocardiogram was made before any treatment was given. An osteopathic examination revealed joint lesions of the third cervical and first and fourth thoracic segments. These lesions were corrected and after a half hour rest a second electrocardiogram was run. Lead IV before treatment showed a Q wave varying from a depth of 1 mm. to complete absence. The T wave was above the isoelectric line. Lead IV after treatment showed a relatively normal tracing with a Q wave of 4 to 5 mm. and a T wave flattened or slightly below the isoelectric line.

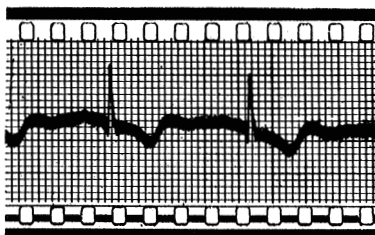
Another patient, (B), reentered the clinic after an absence of a year. At that time the diagnosis was essential hypertension. An electrocardiogram was made because his complaint suggested anginal pain. The Q wave was found to be less than 2 mm. in depth. An osteopathic examination revealed



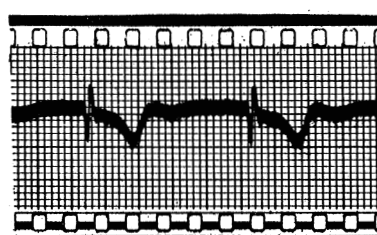
Electrocardiogram, Lead IV,
Patient A, before treatment.



Electrocardiogram, Lead IV,
Patient A, after treatment.



Electrocardiogram, Lead IV,
Patient B, before treatment.



Electrocardiogram, Lead IV,
Patient B, after treatment.

joint lesions of the first cervical and the first, second, third, fifth, and sixth thoracic vertebrae. Correction was made, the patient rested a half hour and a second lead IV run. The Q wave was found to be increased to an average of 5 mm.

Several other similar cases have been handled by the same procedure, and while the results are less outstanding, the EKG run after treatment more nearly approaches the normal.

SUMMARY

Angina pectoris is a syndrome which can be caused by any pathological change which decreases the oxygen supply of the myocardium, or by a spasm of the coronary arteries without other changes.

The pain of angina pectoris is the result of an accumulation of unoxidized products of metabolism in the heart muscle and is relieved by the improvement of coronary circulation.

Coronary circulation is under nervous control, sympathetic fibers causing vasodilatation and vagus fibers causing vasoconstriction.

In EKG studies of angina pectoris, lead IV evidences an absence of the Q wave or its reduction to a depth of 2 mm. or less, with or without an accompanying inversion of the T wave.

In a small series of cases studied at the Chicago College of Osteopathy, these characteristic changes in lead IV were confirmed.

Correction of upper thoracic and cervical joint lesions in these cases results in a change towards normal in lead IV EKG findings.

CONCLUSION

In the absence of more direct experimental means of relating angina pectoris to osteopathic lesion pathology, the electrocardiogram seems to provide an accurate measurement of comparing the efficiency of coronary circulation before and after correction of the lesions found. A large number of cases should be studied by this method and their progress under continued osteopathic treatment recorded.

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