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# The Diagnosis and Treatment of Impending Coronary Thrombosis

BY

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For many years doctors have been aware of symptoms of cardiac pain which were followed after a short interval of time by myocardial infarction. Indeed, it appears that as early as 1912 this was recognised, when Herrick<sup>1</sup> described transitory chest pain preceding myocardial infarction. Wearn<sup>2</sup> (1923) noted that cardiac infarction could occur after a number of anginal attacks, constituting the only previous warning of coronary artery involvement. In 1937, Sampson and Eliaser,<sup>3</sup> in an article entitled "The Diagnosis of Impending Acute Coronary Artery Occlusion," described 29 cases of prolonged praecordial pain preceding characteristic coronary artery occlusion. Feil,<sup>4</sup> describing some sixteen cases of this syndrome, named it "preliminary pain in coronary thrombosis." Mounsey,<sup>5</sup> in his article "Prodromal Symptoms in Myocardial Infarction," describes 49 cases.

Aware of this entity, the doctor will find that on taking a careful history of the events before an attack of acute myocardial infarction, a proportion will be found with preliminary cardiac pain. The reported incidence of this proportion varies from 29 per cent. to 50 per cent., the latter figure being a more correct reflection of the actual state of affairs. These symptoms may precede acute coronary occlusion by twelve weeks.

There being ample warning of an impending cardiac infarction, the question immediately arises as to whether occlusion of the coronary arteries can be prevented. Wood<sup>6</sup> (1949) treated ten cases with anticoagulants of impending infarction, eight recovering without any evidence of infarction, and two dying four weeks after therapy was stopped. Smith and Papp<sup>7</sup> (1951) treated 14 patients, six recovering without the development of myocardial infarction.

## PATHOLOGY

An understanding of the underlying pathology is essential for the appreciation of therapy and for the failure of therapy in a certain percentage of cases. For practical purposes the pathological changes are those of coronary artery atherosclerosis. Briefly, there is thickening of the intima, with fibrosis, cellular hyperplasia and deposition of lipoids, leading to the formation of

atheromatous plaques. Should the process continue, further lipoid deposition gives rise to atheromatous "abscesses," with ingrowing capillaries from the lumen and the vasa vasorum. These may rupture, giving rise to intimal haematomata, which encroach upon the lumen. It would appear that it is at this stage that the patient develops the symptoms of impending infarction.

The subsequent pathological process leading to acute coronary artery occlusion is by one or more of the following mechanisms:—

(1) Rupture of the intimal haematoma into the lumen of the vessel, with secondary thrombosis of the artery. The thrombus forms at the site of rupture of the intimal haematoma, and gradually enlarges, finally leading to an acute occlusion. This is the pathological process underlying the majority of cases of acute coronary artery occlusion. Anticoagulant therapy would be indicated for these cases.

(2) Secondary thrombus formation on arteriosclerotic intimal plaques with damaged and "sticky" endothelial lining. It was formerly held that this was the chief mode of coronary thrombosis. However, in 100 autopsies with acute arteriosclerotic coronary artery occlusion, closure was due to thrombus formation on arteriosclerotic plaques in only one-third of the cases, and in two-thirds to intimal haematoma with or without subsequent thrombus formation (Horn and Finkelstein<sup>8</sup>). Here, also, anticoagulant therapy could be effective in preventing thrombus formation.

(3) Intimal haemorrhage with the formation of a large intimal haematoma encroaching upon the lumen and occluding the artery. In this group, the least frequent, occlusion takes place without thrombus formation. It is unlikely that these cases would respond to previous anticoagulant therapy. There is at present no method of diagnosing these cases. It is partly for this reason that anticoagulant therapy will not always succeed in preventing acute occlusion.

## DIAGNOSIS OF IMPENDING CORONARY ARTERY OCCLUSION

Patients presenting with symptoms of impending myocardial infarction, which may simply be referred to as prethrombotic angina, can be divided into two groups—those with and those without a previous history of angina.

(1) *Previous History of Angina Pectoris of Over Three Months' Duration.*—In this group

there will be evidence of sudden or gradual increase in the duration of anginal pain, of pain appearing at rest, and of a lack of response to nitroglycerine and rest. The pain usually increases in intensity.

Examination generally reveals no change from the previous state of affairs. The electrocardiogram may reflect increased myocardial ischaemia.

(2) *Recent Onset of Angina (within three months).*—It is in this group that a most careful history is required in order to arrive at the correct conclusion. Cardiac symptoms may precede cardiac infarction by a period of up to three months, with an average period of three and a half weeks.

Generally, the patient has enjoyed good health before, and may even claim that he has never been to a doctor before. He may state that he has a mild, short-lived sensation of pressure, or burning under the sternum, and when appearing at its inferior portion may be referred to as indigestion. Indeed, many will have already treated themselves with alkaline mixtures. The sensation may be vicelike, gripping, squeezing, burning or aching. There may be radiation to the back, shoulders, down the arms, or up into the neck. Alternatively, the sensation may begin peripherally as a burning or ache in the wrist, forearm, jaw or neck.

The duration of this discomfort may vary from a few minutes to over an hour. It may be brought on by effort, by emotional extremes or appear at rest. There may be an interval of a week before the first and second bout of discomfort. However, the attacks gradually increase in frequency, duration and intensity, and tend to become localised in the substernal region.

There is no nausea or vomiting. The complexion is normal and the pulse rate, blood pressure, temperature, white cell count and blood sedimentation rate fall within normal limits. The electrocardiogram is either normal or may show slight ischaemic alterations in the form of RS-T segment and T wave changes, which may only appear after an exercise tolerance test.

It will be seen that the diagnosis rests almost entirely upon an accurate analysis of the symptoms, the clinical and instrumental examination serving to exclude other disorders.

The following conditions are commonly associated with coronary atherosclerosis and may serve to indicate the true state of affairs: Hyper-

tension, diabetes, a family history of cardiac infarction or of hypercholesterolaemia. The majority of these patients fall in the age group of 40 to 70 years, the proportion of males to females being three to one.

#### DIFFERENTIAL DIAGNOSIS

This covers a wide range of diseases, which can, for the greater part, be readily excluded.

(1) Referred pain from cervical and dorsal vertebral lesions are generally worse at night, are aggravated by spinal manipulation and percussion, and the affected vertebrae are tender. Areas of hyperaesthesia and muscle wasting may be found.

(2) Joint lesions are excluded by local tenderness and increase of pain on movement.

(3) Chest wall lesions are aggravated by deep respiration, by compression of the thorax, and are tender locally.

(4) Pleural and pulmonary diseases are readily excluded on clinical examination.

(5) Pericardial inflammatory lesions are associated with fever, persistent pain, friction rub, signs of fluid and electrocardiographic changes.

(6) Endocardial lesions, such as mitral and aortic stenosis, may lead to anginal attacks.

(7) Gastrointestinal disorders, such as oesophagospasm, oesophageal and para-oesophageal herniae, peptic ulcer, gaseous distension of the fundus of the stomach or of the splenic flexure of the colon, may give rise to transient substernal or praecordial discomfort. These will, however, be related to the intake of food, posture, relief with expulsion of air or by alkalis. With biliary tract disorders the pain has a specific radiation, there is localised tenderness and a history of fatty dyspepsia.

(8) Cardiac neurosis is commonly associated with left mammary pain, described vividly as stabbing, sticking, piercing, with praecordial tenderness, sighing respirations, absence of palatal reflexes, dermatographia and various phobias.

#### PROGNOSIS

The outlook in patients with symptoms of impending acute coronary artery occlusion is that of myocardial infarction. This may be severe, in which case the mortality rate is in the neighbourhood of 50 to 60 per cent., or it may be mild, when the lowest reported mortality

rate is about 2 per cent. With adequate treatment, occlusion can be prevented in about 50 per cent.; and in the remainder, in whom an intravascular thrombus has formed, this tendency would be lessened. In patients in whom vascular occlusion is occurring without thrombus formation, anticoagulant therapy is unlikely to be of benefit.

#### TREATMENT

It has been repeatedly demonstrated that intravascular thrombus formation in a damaged vessel can be prevented by anticoagulants (Solandt and Best<sup>9</sup>). It has been shown that the majority of acute coronary artery occlusions are due to intravascular thrombus formation. It therefore follows that anticoagulant therapy would be indicated in patients who are about to develop coronary artery thrombosis, more so than in those who have already developed thrombosis, in whom the object of this therapy is to prevent further thrombotic complications. It has been urged that this is a hazardous form of treatment. With adequate care and control the mortality rate from haemorrhage does not exceed 1 per cent., and is probably less with the newer anticoagulants. Furthermore, the outlook in patients with prethrombotic angina is uncertain, and a calculated risk has to be accepted. The duration of anticoagulant therapy should be a minimum of three weeks, and preferably six weeks. Logically, this treatment should be carried on indefinitely, as the background to thrombosis is ever-present.

The second factor to be stressed is that of moderately restricted activity. It has been noted that rest, sedatives and the use of vasodilators has not succeeded in preventing an impending infarction. Indeed, it would appear that better results are to be obtained by avoiding bed rest and by merely moderately restricting the patient's activities. Phipps,<sup>10</sup> analysing 137 cases of acute coronary artery occlusion, observed that the majority had developed the occlusion at rest, whilst asleep or after an operation. It is accepted that a sluggish circulation is apt to be associated with thrombus formation (e.g., post-operative venous thrombosis, thrombosis in calf muscles of cardiac patients confined to bed), and that this complication may be avoided by exercise of the legs. Obviously, however, harm may result if myocardial anoxaemia is accentuated by increased activity. Moderately restricted activity, such as being confined to the house and garden, is therefore recommended.

Mild sedatives are useful for allaying the patient's anxiety. The use of such vasodilators as aminophyllin or papaverine is recommended by some, but their value has not been established. A low fat and cholesterol diet is advisable, with the addition later of the lipotropic amino-acids, methionine or choline.

#### ILLUSTRATIVE CASES

*Case 1.*—A retired farmer, aged 64 years, previously well, developed an aching and burning pain in the back between the shoulder blades, whilst at rest, of about five minutes' duration. Further attacks occurred, of increased duration and intensity, brought on by walking and relieved by rest. This was regarded as a fibrositic pain. Four weeks after the commencement of symptoms he developed a severe substernal, crushing pain, with sweating, collapse, fall in blood pressure and cardiographic changes of a recent extensive anterior myocardial infarction.

*Case 2.* A European shopkeeper, aged 42 years, developed a burning lower substernal discomfort, of short duration, at rest, which he thought was indigestion. Over the course of the next three weeks he had repeated attacks of increased severity, unrelated to effort and unrelieved by alkalis. He then developed symptoms of an acute coronary artery occlusion, with cardiographic changes of a recent infarction.

*Case 3.*—A retired business man, aged 55 years, developed, whilst sitting indoors, sudden lower substernal pressure, with dyspnoea, of 15 minutes' duration. A week later he had a similar attack. Eight days later he had a third attack of an hour's duration. There was no nausea or vomiting. Clinical examination was negative, the blood pressure being 130/80, respirations 20, pulse rate 75 per minute, temperature 98° F., and the E.S.R. 10 mm. (Westgren). Screening of chest and barium swallow was normal. Resting cardiogram normal, but after exercise mild myocardial ischaemia was shown by sagging of the R-S-T segment in leads I, II and V6, depression of this segment in V2-V5 of 1 mm., and diminution in the height of the T waves in V3 and V4 by 3 mm.

The patient was placed on anticoagulant therapy, advised to restrict his activities to the home and not to remain in bed. He was given a mild sedative. He had one further attack four days later. There was no further change in the cardiogram two weeks later. Anticoagulants were stopped after four weeks, and the patient was well three months later.

## SUMMARY

The symptoms and signs produced by the narrowing of the lumen of coronary artery preliminary to its occlusion are described. This set of symptoms has been referred to as pre-thrombotic angina. The pathology is briefly discussed, and the rationale for anticoagulant therapy and modified activity mentioned. Three cases are described illustrating the syndrome.

## REFERENCES

1. Herrick, J. B. (1912), *J. Am. Med. Ass.*, 59, 2015.
2. Wearn, J. J. (1923), quoted by Feil.
3. Sampson, J. J., and Eliaser, M. (1937), *Am. Heart J.*, 13, 675.
4. Feil, H. S. (1937), *Am. J. Med. Sci.*, 194, 42.
5. Mounsey, P. (1951), *Br. Heart J.*, 13, 215.
6. Wood, P. (1949), *Br. Med. J.*, 1, 26.
7. Smith, K. S., and Pappa, C. (1951), *Br. Heart J.*, 19, 655.
8. Horn, H., and Finkelstein, L. E. (1940), *Am. Heart J.*, 19, 655.
9. Solandt, D. Y., and Best, C. H. (1938), *Lancet*, 2, 130.
10. Phipps, C. (1936), *J. Am. Med. Ass.*, 106, 761.





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