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Haemodynamic Observations During Spontaneous Angina Pectoris

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In recent years a great deal of new information has been obtained concerning the general haemodynamic changes that occur during attacks of angina pectoris. Most of the reports on this subject are based upon studies during exertional angina pectoris. Haemodynamic observations during spontaneous angina pectoris are more scanty, and recordings of cardiac output during spontaneous angina pectoris do not seem to have been reported. The following case report includes such recordings.

Case Report

The patient was a 76-year-old man who had suffered from angina pectoris for 12 years. There were no signs or history of previous myocardial infarction. For the past 2 years he had experienced frequent attacks of spontaneous angina pectoris, some of which apparently were provoked by emotional reactions. Nitroglycerin brought prompt relief.

Physical examination of the heart revealed nothing abnormal. The blood pressure was 135–125/80–65 mm. Hg, haemoglobin 14·5 g./100 ml.

Electrocardiogram showed ST–T changes compatible with myocardial ischaemia. Chest *x*-ray films showed normal contours of the heart and a volume well within normal limits.

During 3 attacks of spontaneous angina pectoris the following investigations were recorded.

Cardiac output was recorded by dye dilution technique (Stewart–Hamilton principle): 5 mg. indocyanine green was injected through a polyethylene catheter introduced via a cubital vein to an intrathoracic position. Aspiration was accomplished through a polyethylene catheter in the brachial artery. *Brachial arterial pressure* was recorded through the same catheter. *Heart rate* was read from the electrocardiogram.

The patient was examined on two occasions one week apart.

Comments. The results of the recordings are set out in the Fig.

During the first and third attack of angina pectoris neither the patient nor the examiner could find any particular eliciting cause. The second attack was apparently provoked by conversing with the patient on a subject which caused an emotional response.

The recordings of cardiac output show that in this patient the attacks of spontaneous angina pectoris coincide with a marked hypokinesia of the left ventricle. Pulmonary arterial pressure, pulmonary wedge pressure, or any left-sided pressures were not recorded, but towards the end of the first attack, auscultation gave evidence of acute, left-sided backward failure, with physical signs of pulmonary congestion. At the same time the patient was very dyspnoeic. After nitroglycerin had been given and the angina pectoris had disappeared, the cardiac output increased to a level above that before the attack. This phenomenon was also observed after the subsequent attacks, and may be related to oxygen debt generated during the hypokinetic period.

The attacks of spontaneous angina pectoris were accompanied by increases of heart rate and brachial arterial pressure. The level of heart rate was fairly equal during the three observed attacks, while brachial arterial pressure and consequently the rate-pressure products varied considerably. The rate-pressure products have not been corrected for possible variations in the systolic ejection time as the incisura on the late systolic part of the brachial arterial pressure curve was faint.

Oxygen breathing apparently reduced the degree of left ventricular hypokinesia compared with the two other attacks of angina pectoris. It did not, however, abolish the pain, and the attack terminated only after the administration of nitroglycerin.

The recordings suggest that nitroglycerin has a strikingly advantageous effect on the abnormal haemodynamics during spontaneous angina pectoris.

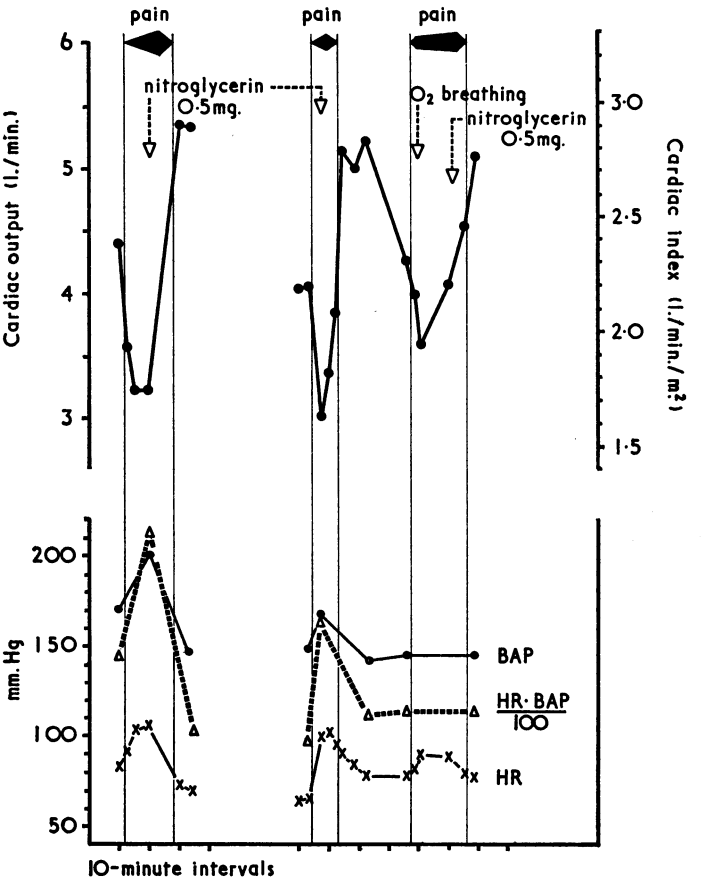

FIG.—Each point on the cardiac output curve represents one cardiac output determination. The figures illustrating pain are drawn to give approximative information of the time at which the patient sensed the start of, the diminution of, and the disappearance of pain.

Discussion

It is commonly believed that angina pectoris is caused by transitory myocardial ischaemia which may be the effect of reduced coronary blood perfusion (in the left ventricular myocardium secondary to increase of heart rate and shortening of diastole and possibly vasospasm) or increased oxygen consumption secondary to increased myocardial work.

Robinson (1967), using rate-pressure product (1/100 × heart rate × systolic arterial pressure) as a parameter of cardiac work, observed the relation between the rate-pressure product and angina pectoris, and found that in the individual patient pain was elicited at a constant level of raised rate-pressure product. The same author also observed one patient with spontaneous angina pectoris. In this patient an increase in heart rate and systolic arterial pressure was followed by angina pectoris which started at the same level of rate-pressure product which was followed by angina pectoris during exercise. This corresponds well with the observations of Roughgarden (1966) recording arterial blood pressure and heart rate during spontaneous angina pectoris.

On the 2 different days of observation the rate-pressure products of our patient show great variation. Our recordings are not frequent enough to show whether the increases in heart rate and brachial arterial pressure initiate the attack of angina pectoris or if these phenomena are secondary in the sequence of ischaemia and hypokinesia of the left ventricle.

Robinson interprets his observations to indicate that vasospasm is usually of little importance as a cause of angina pectoris. On the other hand, it has

been commonly believed that the effect of nitroglycerin on angina pectoris is based on coronary vasodilatation and subsequent increase of coronary perfusion. Bernstein *et al.* (1966) noted increased coronary perfusion secondary to the injection of nitroglycerin into the coronary arteries, while no significant change of perfusion was seen after sublingual administration of the drug. There are, however, reasons to believe that nitroglycerin may affect angina pectoris by reducing cardiac work secondary to its effect on heart rate and blood pressure (Bernstein *et al.*, 1966; Hoeschen *et al.*, 1966; Müller and Rørvik, 1958; Najmi *et al.*, 1967; Parker, Di Giorgi, and West, 1965).

Some observers have shown that angina pectoris induced by exercise is accompanied by a relative hypokinesia of the left ventricle, and that nitroglycerin prevents this haemodynamic event when the drug is able to prevent development of angina pectoris (Najmi *et al.*, 1967; Parker *et al.*, 1965).

In accordance with these changes in cardiac output, increases in pulmonary arterial pressure and pulmonary wedge pressure have been observed during angina pectoris, but the pressures remained normal during exercise when nitroglycerin prevented angina pectoris (Müller and Rørvik, 1958; Najmi *et al.*, 1967; Parker *et al.*, 1965). Ross *et al.* (1962) and Parker *et al.* (1965) noted a rise in left ventricular end-diastolic pressure during angina pectoris; but Cohen *et al.* (1965) found only occasional increases.

Müller and Rørvik (1958) observed pathological increases in pulmonary arterial pressure and pulmonary wedge pressure in 2 patients with spontaneous angina pectoris, and in the same patients they also observed a higher oxygen saturation in mixed venous blood during the attacks. They were inclined to believe that the cardiac output was increased during angina pectoris in these patients who, however, were both anaemic. The recordings of cardiac output in our patient are more in accordance with the results of recordings during exertional angina pectoris.

The reproducible results from the three attacks of spontaneous angina pectoris in our patient may reasonably be interpreted as the haemodynamic consequences of "pure" spontaneous angina pectoris, as there was no reason to believe that the recordings were influenced by myocardial insufficiency from causes other than the reversible anginous attacks.

Summary

Spontaneous angina pectoris has been studied with recordings of heart rate, blood pressure, and serial determinations of cardiac output.

The patient was a man of 76 with a history of exertional and spontaneous angina pectoris. There were no signs of previous myocardial infarction, and no signs of heart failure. The patient had normal blood pressure and had no anaemia. The heart was normal on chest *x*-ray.

Three attacks of spontaneous angina pectoris were observed. The recordings show that these attacks were followed by a conspicuous decrease of cardiac output while there were increases of heart rate and blood pressure. Both the pain and the abnormal haemodynamic changes were rapidly abolished when nitroglycerin was given.

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