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Cardiac Function in Patients with Angina

Ottar Müller

Abstract

It is a great honour and pleasure for me to be invited to this meeting, commemorating the introduction by your former member, T. Lauder Brunton, of amyl nitrite as a potent remedy for anginal pain, one hundred years ago. For some years, I have been interested in the haemodynamic consequences of coronary heart disease, the most common cause of anginal pain. That this symptom is due to an improper balance between the myocardial demand for energy and the supply available from the coronary circulation has been more or less generally accepted for a long time. This view seems, for instance, to have been held by Allan Burns in his book on cardiology printed in this city in 1809. The exact cause of the pain is still uncertain. It seems, however, to be linked to inadequate delivery of oxygen to the myocardium, either generally or locally. And metabolic studies (Cohen el al. 1965) have indicated changes probably due to hypoxia in patients with coronary heart disease not only during but also in the intervals between attacks of angina pectoris. If reduction of the coronary circulation and abnormal metabolism are common in these patients, it is reasonable to expect a reduction in ventricular function as well. However, one of the main symptoms of impaired left heart function, namely dyspnoea, has not been generally regarded as a feature of this condition (P. Wood 1953).

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CARDIAC FUNCTION IN PATIENTS WITH ANGINA

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It is a great honour and pleasure for me to be invited to this meeting, commemorating the introduction by your former member, T. Lauder Brunton, of amyl nitrite as a potent remedy for anginal pain, one hundred years ago.

For some years, I have been interested in the haemodynamic consequences of coronary heart disease, the most common cause of anginal pain. That this symptom is due to an improper balance between the myocardial demand for energy and the supply available from the coronary circulation has been more or less generally accepted for a long time. This view seems, for instance, to have been held by Allan Burns in his book on cardiology printed in this city in 1809. The exact cause of the pain is still uncertain. It seems, however, to be linked to inadequate delivery of oxygen to the myocardium, either generally or locally. And metabolic studies (Cohen *et al.* 1965) have indicated changes probably due to hypoxia in patients with coronary heart disease not only during but also in the intervals between attacks of angina pectoris. If reduction of the coronary circulation and abnormal metabolism are common in these patients, it is reasonable to expect a reduction in ventricular function as well. However, one of the main symptoms of impaired left heart function, namely dyspnoea, has not been generally regarded as a feature of this condition (P. Wood 1953).

A number of investigations have been published in the last 10 years concerning the cardiac function in patients with coronary heart disease, but in my presentation today, I will concentrate mainly on my own investigations which have been carried out by means of right heart catheterization and include measurements of the pulmonary capillary venous pressures. These observations will also be discussed in the light of two other published studies, of R. Malmborg, 1964 and of Cohen *et al.*, 1965.

My investigations involved patients in whom the diagnosis of coronary heart disease had been established clinically with reasonable certainty, and in whom complicating factors such as valvular disease, hypertension, anaemia, and thyrotoxicosis have been excluded.

The investigations comprised studies both at rest and during light recumbent leg exercise (approximately 140 kgm/min for 5 min).

Figure 1 shows mean values of the main observed and calculated parameters in a resting state, for a control group with the same average age as two groups of patients, one consisting of 18 patients without recognised previous infarction and one of 61 patients with a history of infarction. The only parameter listed which deviated significantly from the controls was pulmonary capillary venous pressure.

REST Mean values

	Controls	A.P.	Infarct
O_2-cons./M^2	130·8	131·8	137·2
C I	3·49	3·52	3·29
Str. Index	49·5	52·8	47·9
PCVP	6·9	8·8	11·4
$BP_{syst.}$		129	122
Heart rate	70·5	68·2	68·7

Figure 1
Mean values for the three groups: Controls, patients with coronary heart disease without previous myocardial infarction and patient with previous myocardial infarction.

Figure 2 (on page 33) demonstrates individual pulmonary capillary venous pressure readings in the two groups of patients related to heart size assessed by x-ray. None of the patients without

previous myocardial infarcation exceeded the normal upper limit of 14 mm Hg, although the group *average* was elevated. In the group of patients with a history of infarction, pulmonary capillary venous pressure varied with heart size, and one patient was in pulmonary oedema during the investigation. On the other hand, with decreasing heart size, pulmonary capillary venous pressure approached normal levels. Thus, investigated in this way, only a small proportion of patients with coronary heart disease will, *as individuals*, display distinctly abnormal cardiac functions and these will mainly be found among those with enlarged hearts.

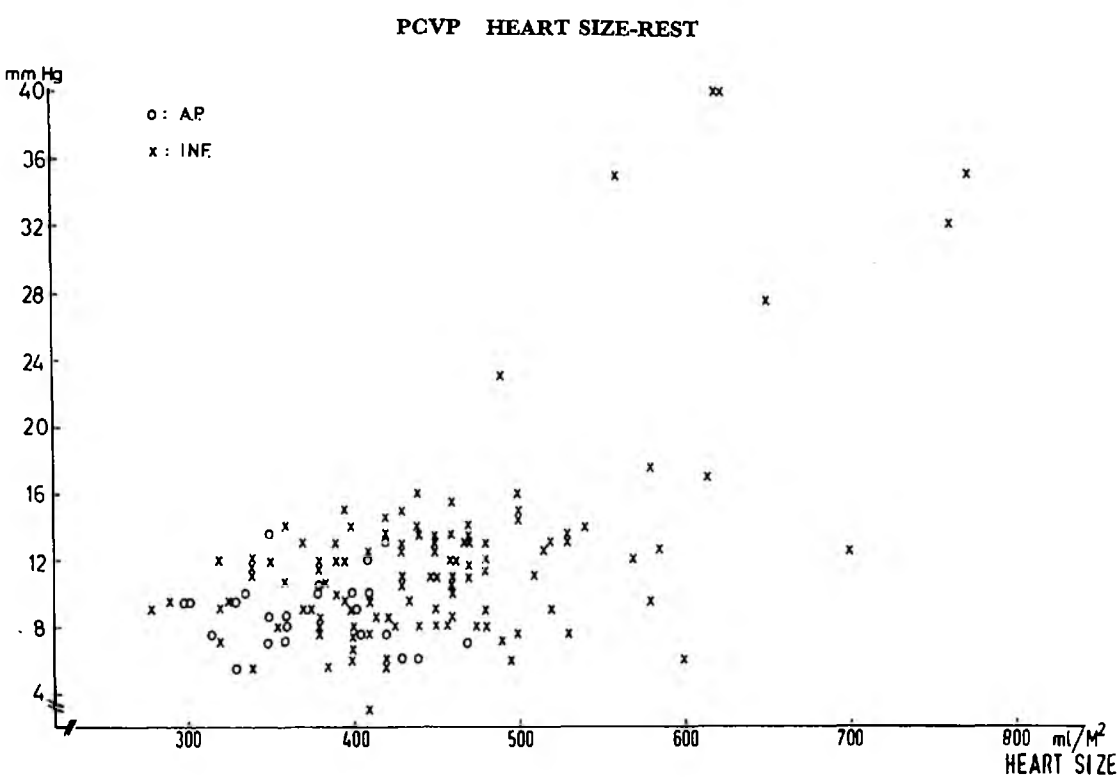
Figure 3 illustrates the mean values obtained during the exercise test. The higher oxygen uptake in the infarct group reflects reduced efficiency and *not* a heavier load. Increase in cardiac output is here expressed as relative to increase in oxygen uptake. Marked deviations from the controls were demonstrated, not only for pulmonary capillary venous pressure in both groups of patients but also for increase in cardiac output and stroke index in the group of patients with previous infarction. This was at least partly due to the patients with increased heart size. During the exercise test mean pulmonary capillary venous

Figure 2
Pulmonary capillary-venous pressure at rest related to heart size (ml/M²). Patients with (Inf.) and without (A.P.) previous myocardial infarction.

EXERCISE Mean values

	Controls	A.P.	Infarct
O_2-cons./M²	350·5	319·6	367·2
$\frac{dCO}{dO_2\text{-cons.}}$	7·7	6·9	6·2
Str. Index	59·8	58·9	52·1
PCVP	10·1	18·9	23·2
$BP_{syst.}$		139	144
Heart rate	91·3	87·0	88·8

Figure 3
Mean values for the three groups during light exercise.



pressure showed a more marked difference between patients and controls than at rest.

Figure 4 demonstrates individual pulmonary capillary venous pressure in the two groups of patients related to heart size. Only a small proportion of the *patients'* pulmonary capillary venous pressure valves remained close to the normal limit of 15 mm Hg. A wide scatter was found and cases with greatly increased pressure were distributed regardless of heart size in both groups. With enlarged hearts, however, near-normal observations were lacking. It seems therefore that under the conditions of our exercise test, impaired left ventricular function may be revealed in varying degree by increased left ventricular diastolic, left atrial and pulmonary venous pressures in most patients in whom the diagnosis of coronary heart disease can be established by ordinary criteria. The exercise load used produced anginal pain in only a minority of the patients.

Figure 4
Pulmonary capillary-venous pressure during exercise related to heart size.

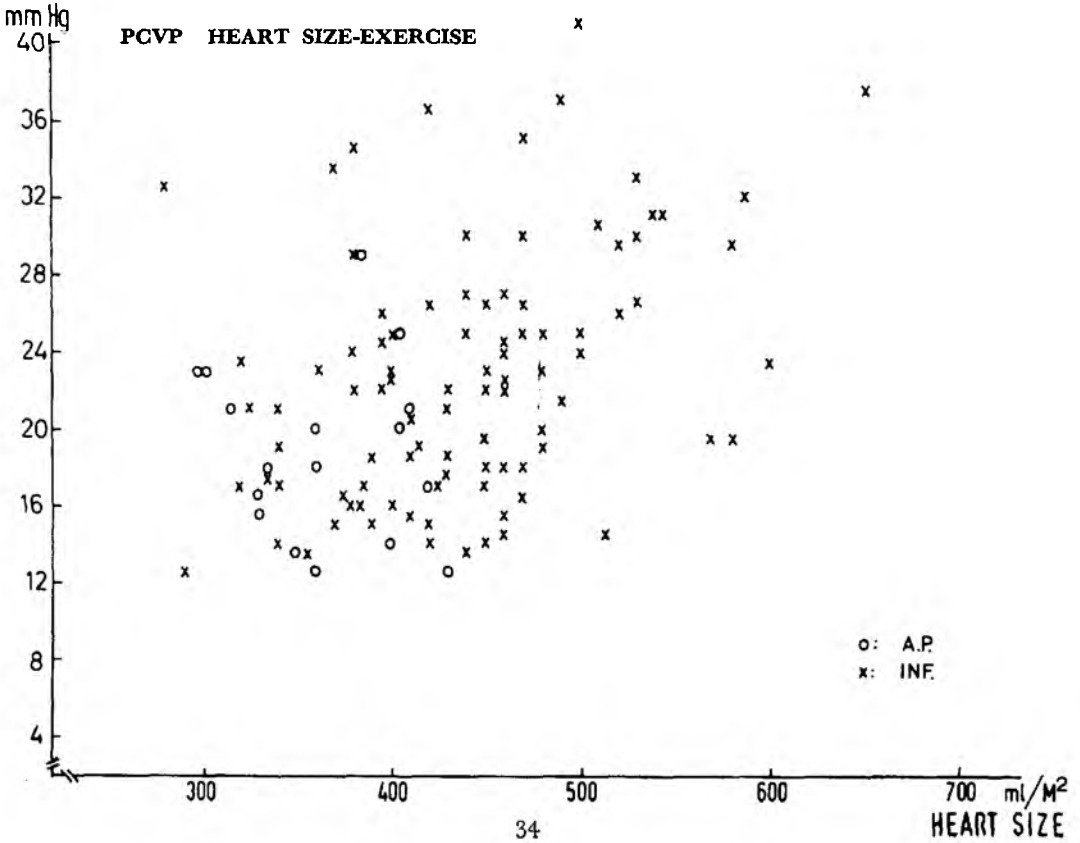
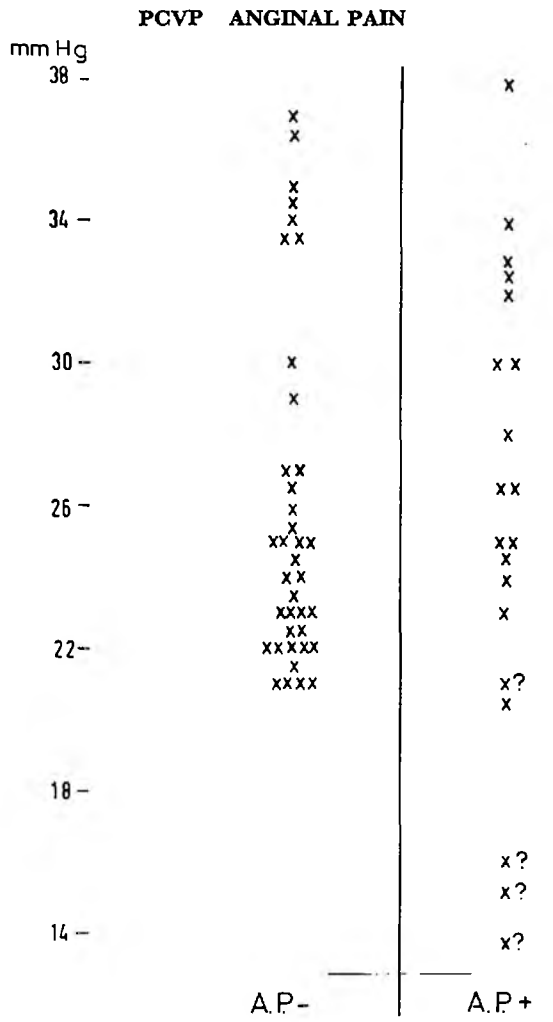


Figure 5 (on page 35) illustrates the pulmonary capillary-venous pressure values recorded during the exercise test in patients with anginal pain as the pain complaint and heart sizes of 500 ml/M² or below. The great majority of patients who developed typical anginal pain had markedly elevated pulmonary capillary venous pressure, while most of the patients with atypical sensations in the chest had only slightly or moderately elevated pressures. On the other hand, a number of patients developed markedly increased pulmonary capillary venous pressure but denied any discomfort. A further group, *not marked in the figure*, had neither unpleasant sensations nor markedly increased pulmonary capillary venous pressure. Observations indicating impaired left ventricular function in patients with coronary heart disease, especially during anginal pain and when exposed to stress have been obtained by several groups of investigators. Abnormal increases in pulmonary capillary venous pressure on exertion and particularly when this precipitates anginal pain have been reported by Malmborg

(1964). Ross and co-workers (1962) observed increased left ventricular end-diastolic pressure during spontaneous angina, while Benchimol and Dimond (1963) recorded, tracings indicative of altered left heart function by apex cardiography.

Figure 5
Pulmonary capillary-venous pressures during exercise in patients who developed anginal pain during the exercise test (AP+) and who did not (AP—). Heart sizes $\leqq$ 500 ml/M². See also text.



In an interesting publication, Cohen *et al.* in 1965 reported the observation of abnormally reduced increases in left ventricular ejection rate and stroke volumes in patients with coronary heart disease during exercise and especially so in the presence of anginal pain. All these investigations confirm that signs of impaired left ventricular function may be observed in different haemodynamic parameters, and that this impairment is a common occurrence in patients with coronary heart disease during exercise and most markedly during attacks of anginal pain. There are, however, marked discrepancies between some of the observations reported. Malmborg (1964), for example, in his study of 36 patients obtained a mean pulmonary capillary venous pressure during exercise which is very close to my figure, but with a markedly higher work load (mean oxygen) uptake *ca* 480 ml/M², compared to *ca* 345 ml/M²), with higher heart rates (113 compared to 90), higher systolic blood pressures (182 compared to 142) and a higher proportion of patients with anginal pain (3/4 versus 1/3). Cohen and coworkers (1965) found a consistent rise in left ventricular end-diastolic pressure neither during exercise tests precipitating anginal pain nor during anginal pain provoked by other stimuli leading to higher heart rates than observed by me, but with moderate peripheral blood pressure levels. The reason for this conflict in observation is obscure. It seems, however, to be of some importance to know whether high left ventricular diastolic and high left atrial pressures are a common occurrence in patients with coronary heart disease.

Gorlin's group (Cohen *et al.* 1965) in one of their papers suggested that the observed increase in pulmonary capillary venous pressure depended on high systolic (peripheral) blood pressures, leading to increased work and high myocardial oxygen demand.

Malmborg's observations may support this assumption. Mean systolic blood pressure in his patients during anginal attacks was as high as 190 mm Hg. compared with 162 in patients without anginal pain and with more moderate increase in pulmonary capillary venous pressure during the exercise test. Systolic blood pressure levels seem, however, to explain fully neither the contrast between Malmborg and my own patients with regard to pulmonary capillary venous pressure, nor the great discrepancy between the observed left ventricular end-diastolic pressures of Cohen *et al.* and the pulmonary capillary venous pressure observed by me during exercise.

The other main determinant of diastolic ventricular and atrial pressure at a given ventricular

function and systolic blood pressure is stroke volume, and this parameter suggested by animal-experiments seems indirectly to influence myocardial oxygen consumption.

Stroke volume seems to vary remarkably from one investigation to the other.

Figure 6 illustrates mean stroke volume, peripheral systolic blood pressure, heart rate and pulmonary capillary venous pressure in my patients with markedly elevated pulmonary capillary venous pressure, arranged according to whether or not they developed anginal pain during the exercise test. There was a higher average peripheral blood pressure in the group of patients with angina during the test. On the other hand, patients without angina had a greater average stroke volume. The differences were, however, very slight – and systolic blood pressure in both groups moderate as exercise-pressures.

$PCVP_{ex.}$ 24 mm Hg. or above

	A.P.+	A.P.—
PCVP	27·3	28·2
H.R.	88	86
$BP_{syst.}$	146	136
Str. Ind.	46·2	51·7

Figure 6
Mean values for patients with marked increases in pulmonary capillary-venous pressures during exercise. The two groups comprise patients who did (AP+) and did not (AP—) develop anginal pain during the test.

In Malmborg's series, patients with comparable pulmonary capillary venous pressure had an average stroke index of under 40 ml.; while in Cohen's study the group of patients who developed anginal pain during the exercise test had an average stroke index as low as 32 ml. In the group without anginal pain the figure was 38 ml., compared to 49 ml. in the control group.

These variations in stroke index from group to group of patients seem to me to offer a probable explanation of the differences in observed pulmonary capillary venous pressure and left ventricular diastolic pressure. Nevertheless, I agree with the view that systolic blood pressure may be of some importance as well.

Conclusion

Signs of impaired left ventricular function are obtainable from coronary heart disease patients under stress, and especially during attacks of anginal pain. The manner in which this left ventricular impairment manifests itself seems, however, to vary with several haemodynamic parameters of which systolic ventricular pressure and – especially – stroke volume may be the most important.

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