

P wave analysis in ischaemic heart disease

An echocardiographic, haemodynamic, and angiographic assessment¹

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Fifty-two men with stable angina pectoris, without associated valvular heart disease or congestive failure, were studied to assess the electrocardiographic P wave abnormalities and their relation to left atrial size, pressure, and left ventricular function. Their mean age was 52 years. Duration of P waves in leads II, III, and aVF and the P terminal force in V1 were examined. Echocardiograms were obtained to assess left atrial size and left atrial-aortic ratio. These measurements were also made in 33 normal subjects. Mean pulmonary artery wedge pressure at rest and exercise, left ventricular asynergy, ejection fraction, and severity of coronary artery disease were determined in all 52 patients.

P terminal force more negative than -0.02 mm s was noted in 69 per cent of coronary artery disease patients but in none of the normal subjects. P terminal force correlated with exercise pulmonary artery mean wedge pressure but not with left atrial size. P wave duration in lead II did not correlate with pulmonary artery mean wedge pressure or left atrial size. P terminal force correlated with left ventricular contraction abnormality but not with ejection fraction or number of vessels diseased. It is suggested that chronic intermittent increases in left atrial pressure in association with angina pectoris result in left atrial hypertrophy which is responsible for the increased magnitude of P terminal force in V1 in patients with ischaemic heart disease.

Morphological changes in the P wave of the electrocardiogram have been seen in valvular heart disease (Morris *et al.*, 1964; Gooch *et al.*, 1966; Kasser and Kennedy, 1969; Rubler *et al.*, 1976), hypertension (Ross, 1963; Tarazi *et al.*, 1966), heart failure (Wood and Selzer, 1939; Sutnick and Soloff, 1962; Romhilt and Scott, 1972), acute myocardial infarction (Master, 1933; Grossman and Delman, 1969; Heikkilä and Luomanmäki, 1970; Chandraratna and Hodges, 1973), and ischaemic heart disease (Bethell and Nixon, 1972; Rios *et al.*, 1974). P wave abnormalities have been correlated with atrial hypertrophy caused by pressure overload and atrial enlargement resulting from volume overload. Abnormal P waves may also result from atrial conduction defects.

In 1964 Morris and his colleagues noted that the P terminal force in lead V1 correlated with left

atrial pressure or volume overload in patients with valvular heart disease. They proposed that a P terminal force with an area of negative value exceeding -0.03 mm s was usually diagnostic of left atrial involvement (Morris *et al.*, 1964). Other commonly used electrocardiographic criteria for left atrial enlargement consist of a P wave duration of 0.12 s or more in the limb leads (Massie and Walsh, 1960).

Previous studies in stable angina pectoris have revealed P wave abnormalities in 18 to 51 per cent of patients. The abnormal P waves suggested the presence of left atrial enlargement but left atrial size was not determined and the patients had not been studied by coronary arteriography (Bethell and Nixon, 1972; Rios *et al.*, 1974).

This study was undertaken to evaluate the P wave abnormalities in patients with stable angina pectoris who have had haemodynamic and angiographic studies as well as a determination of left atrial size by echocardiography.

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Methods

A total of 52 patients with stable angina pectoris who had entered the Palo Alto Veterans Administration Hospital between 1972 and 1975 were studied. All were men and their mean age was 52 years (range 34 to 66). All were considered suitable for saphenous vein bypass graft surgery on the basis of clinical studies and the presence of significant obstructive coronary arterial disease. Significant coronary lesions were judged to be present when arteriography showed a 50 per cent or more reduction in diameter of the lumen.

Patients with valvular heart disease, congestive heart failure, and frequent cardiac arrhythmias were excluded. Of the 52 patients, 24 (46%) had electrocardiographic evidence of a prior myocardial infarction and 9 (17%) had hypertension (systolic pressure exceeding 150 mm and diastolic pressure exceeding 90 mm). Two patients (4%) had electrocardiographic evidence of left ventricular hypertrophy.

All patients were studied by cardiac catheterisation, coronary arteriography, and left ventriculography. Cardiac outputs by the Fick method were obtained and pulmonary artery wedge pressures were recorded at rest and during moderate supine cycle-ergometry exercise. Arteriography was performed using the Judkins technique. Left ventricular contraction abnormalities were identified and ejection fractions were calculated by determination of the major and minor axis of the left ventricular chamber at end systole and end diastole. Echocardiograms were recorded using a strip chart recorder (36 patients) or by photographs taken from a Polaroid camera (16 patients). Left atrial diameter was determined at end systole and aortic diameter was measured during end diastole. A left atrial-aortic ratio equal to or greater than 1.2 or a left atrial dimension equal to or greater than 4.2 cm were considered to indicate left atrial enlargement (Brown *et al.*, 1974). Echocardiograms and electrocardiograms were recorded on the same day.

All patients had resting standard 12-lead electrocardiograms recorded at standard paper speed. The electrocardiogram was recorded within 24 hours of the haemodynamic study. None of the patients had chest pain while the electrocardiogram was recorded. P wave abnormalities were not changed in serial electrocardiograph tracings. P wave amplitude and duration were determined by a single observer using a hand lens and callipers. The P terminal force in mm s was calculated from the terminal negative deflection of the P wave in lead V1 using the method of Morris *et al.* (1964) as shown in Fig. 1.

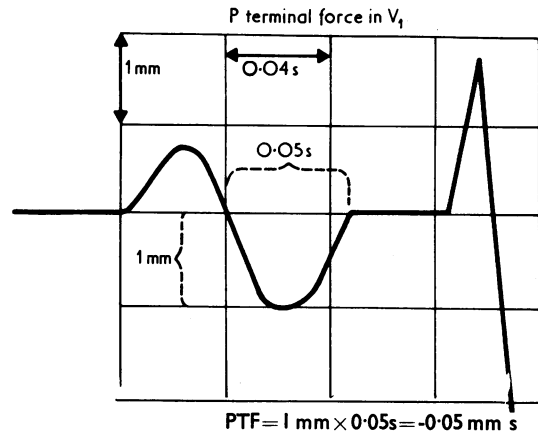


Fig. 1 Calculation of P terminal force (PTF) from terminal deflection of P wave in V1 is shown. Duration in seconds is multiplied by the maximum negative amplitude in millimetres to obtain P terminal force in mm s.

Control data were obtained from 33 normal subjects with a mean age of 37 years (21 to 59). Electrocardiograms and echocardiograms were evaluated by the same methods used in the study group. Haemodynamic and angiographic studies were not performed in the control group.

The data obtained by the above studies were analysed to determine:

- (1) The prevalence and nature of P wave abnormalities in patients with coronary disease compared with normal controls.
- (2) The correlation of P wave abnormalities with:
 - (a) left atrial diameter determined by echocardiography;
 - (b) pulmonary artery wedge pressure at rest and during exercise;
 - (c) left ventricular contraction pattern and ejection fraction;
 - (d) number of coronary arteries with significant obstructive disease.

Table 1 P wave characteristics in patients with coronary artery disease and normal subjects

P wave characteristics	Coronary artery disease (n=52)		Normal subjects (n=33)	
	No.	%	No.	%
(1) P wave duration limb leads				
(a) < 0.11 s	23	44	29	88
(b) ≥ 0.11 but < 0.12 s	11	21	4	12
(c) ≥ 0.12 s	18	35	0	0
(2) PTF-V1 mm s				
(a) Equal to or less negative than -0.02	16	31	33	100
(b) More negative than -0.02	36	69	0	0

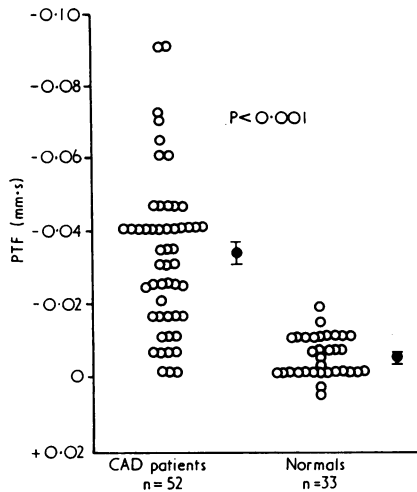


Fig. 2 Range of P terminal force in patients with ischaemic heart disease and in normal subjects. The mean value and ± 1 standard error of the mean are shown. PTF = P terminal force.

Results

Eighteen of 52 patients (35%) had a P wave duration in the limb leads equal to or greater than 0.12 s. None of the normal subjects had a P wave duration of greater than 0.11 s (Table 1).

Of the 52 patients, 36 (69%) had a P terminal force more negative than -0.02 mm s but none of the normal subjects exceeded that value. The difference was significant (-0.0331 ± 0.0029 versus -0.0045 ± 0.0008) with a P value of < 0.001 (Fig. 2).

P wave duration in extremity leads and P terminal force correlated poorly with left atrial aortic ratio or left atrial diameter (Fig. 3). Of all 52 patients, 24 (46%) had evidence of increased left atrial diameter; 16 of 36 patients (44%) with abnormal P terminal force values had evidence of increased left atrial diameter. None of the normal subjects had abnormal left atrial size.

P wave duration in the limb leads did not correlate with pulmonary artery mean wedge pressure at rest or during exercise.

P terminal force showed a significant correlation with pulmonary artery wedge pressure during exercise with a P value of < 0.001 and an r value of 0.54 (Fig. 4). The correlation with resting pulmonary artery wedge pressure was not significant.

Twenty-two patients who had a rise of pulmonary artery wedge pressure of greater than 10 mmHg during exercise had a more negative P terminal force (0.0475 ± 0.0043) than 30 patients who had a lesser rise in pulmonary artery wedge

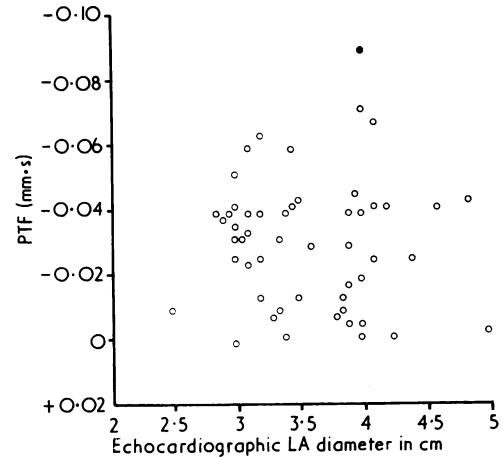


Fig. 3 Relation between P terminal force (PTF) and echocardiographic left atrial diameter in 52 patients with ischaemic heart disease. There was no significant correlation by linear regression between these variables. A poor correlation was also noted when P terminal force (PTF) was compared with echocardiographic left atrial aortic ratio (LA/Ao) ratio.

pressure (-0.0219 ± 0.0026). The difference was significant with a P value < 0.001 . The data are summarised in Fig. 5.

Patients with angiocardigraphic evidence of abnormalities in left ventricular contraction had P terminal force values that were more abnormal (-0.0376 ± 0.0035) than patients with normal left ventricular contraction (-0.0249 ± 0.0048). The difference was significant with a P value of < 0.05 (Fig. 6).

No correlation was noted between P terminal force values and the number of significantly ob-

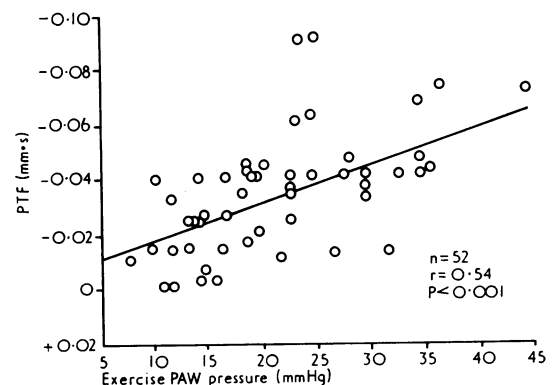


Fig. 4 Linear regression of P terminal force (PTF) versus pulmonary artery wedge (PAW) pressure during exercise in patients with ischaemic heart disease.

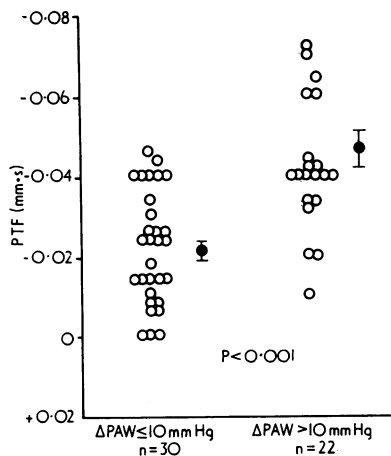


Fig. 5 Values of P terminal force (PTF) in two groups of patients with ischaemic heart disease. The group on the left had a rise in pulmonary arterial wedge pressure with exercise equal to or less than 10 mmHg and the group on the right raised pulmonary artery wedge pressure with exercise by more than 10 mmHg. The closed circles indicate the means and the brackets ± 1 standard error of the mean.

structed coronary arteries or with left ventricular ejection fraction (Table 2).

There was no correlation between left atrial size and pulmonary artery wedge pressure at rest or during exercise.

Discussion

P wave abnormalities in ischaemic heart disease have been studied in acute myocardial infarction (Master, 1933; Grossman and Delman, 1969; Heikkilä and Luomanmäki, 1970; Chandraratna and Hodges, 1973) but few studies have been done in patients with stable angina pectoris. Bethell and Nixon (1972) noted that in 152 patients with angina the incidence of a P terminal force more negative than -0.02 s was 18 per cent compared with an incidence of 0.5 per cent in 206 normal subjects. Rios *et al.* (1974) noted P wave abnormalities compatible with left atrial abnormalities in 51 per cent of 84 patients with ischaemic heart disease.

In the present study the higher incidence of P terminal force abnormalities (69%) is probably related to the presence of more severe disease in the study population. The difference cannot be explained by the incidence of previous myocardial infarction since patients with previous infarction had the same incidence of P terminal force abnormalities (71.4%) as patients without earlier infarc-

Table 2 Coronary artery disease and left ventricular ejection fraction in patients with normal or abnormal P terminal force

	P terminal force equal to or less neg than -0.02 mm s		P terminal force more neg than -0.02 mm s
No. of vessels with obstruction	1.88 \pm 0.20 (n=16)	NS	2.11 \pm 0.12 (n=36)
Ejection fraction (%)	76.5 \pm 3.7 (n=16)	NS	68 \pm 4.0 (n=36)

tion (70.8%). However, the incidence of abnormal P terminal force in this study would be significantly lower (46% or 24/52) if Morris's criteria were used for abnormal P terminal force.

Morris and his colleagues (1964) evaluated P wave abnormalities in valvular heart disease and suggested that a P terminal force equal to or less negative than -0.03 mm s could be considered normal. Bethell and Nixon, however, suggested that a P terminal force of -0.02 mm s should be the limit of normality. The present study supports this latter view.

The duration of the P wave in extremity leads is less useful than the P terminal force as a sign of left atrial pressure overload. P wave duration did not correlate with the rise in pulmonary artery

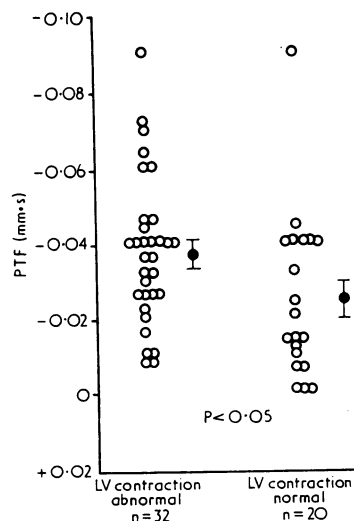


Fig. 6 Values of P terminal force (PTF) in two groups of patients with ischaemic heart disease. The group on the left had left ventricular (LV) contraction abnormalities on angiography and the group on the right had normal left ventricular contractions. The closed circles indicate the means and the brackets ± 1 standard error of the mean.

wedge pressure during exercise whereas the P terminal force did.

By echocardiographic criteria an increase in left atrial dimension was noted in 44 per cent of patients. Left atrial size, however, correlated poorly with the duration of the P wave in the ECG limb leads and the P terminal force. The P terminal force correlated significantly with pulmonary artery wedge pressure during exercise. Patients who had a rise in pulmonary artery wedge pressure of >10 mmHg had more abnormal P terminal force values than patients who had a lesser rise in pulmonary artery mean wedge pressure. Abnormal P terminal force values also correlated with abnormalities in left ventricular function. These observations suggest that abnormal P terminal force values in angina pectoris are the result of pressure overload and not of an increase in left atrial size alone. It is now well known that spontaneous or induced angina is frequently associated with a rise in left ventricular end diastolic pressure (Kasparian and Wiener, 1968). In patients with chronic angina repeated elevations of diastolic pressure could result in left atrial hypertrophy which may not be accompanied by an increase in left atrial volume. Anatomical studies correlating left atrial wall thickness and left atrial volume with P wave abnormalities in ischaemic heart disease are needed to investigate this hypothesis. The data relating P wave abnormalities to left atrial anatomy available at present are not sufficient to answer this question (Bachmann, 1941; Pirani and Langendorf, 1949; Mazzoleni *et al.*, 1964; Kasser and Kennedy, 1969; Romhilt *et al.*, 1972).

Of the 52 patients, 4 had a P terminal force more negative than -0.03 mm s with a normal pulmonary artery mean wedge pressure during exercise and normal left atrial dimensions. Factors other than pressure overload or atrial dilatation are probably responsible for the P wave abnormalities in those patients. Saunders *et al.* (1967) have noted that P wave abnormalities may be present in patients with normal left atrial dimensions. Atrial conduction abnormalities involving Bachmann's bundle may result in P wave abnormalities (Bachmann, 1941). It is, therefore, likely that some P wave abnormalities in ischaemic heart disease may be the result of atrial conduction defects resulting from atrial infarction or ischaemic injury as well as infiltrative or inflammatory disease.

P wave abnormalities may be present in patients with systemic hypertension (Ross, 1963; Tarazi *et al.*, 1966). In the present study 9 patients had hypertension and abnormal P terminal force values were present in 7 of these patients. The incidence was only slightly greater than that observed in

patients without hypertension; hence hypertension could not explain the high incidence of P terminal force values observed in the present study.

In conclusion, this study has shown that patients with stable angina pectoris frequently have abnormal P waves in lead V1 consisting of a P terminal force more negative than -0.02 mm s. This abnormality can be correlated with a rise in left ventricular filling pressure during exercise and abnormal left ventricular function, but not with left atrial diameter. An abnormal P terminal force in lead V1 is more commonly observed than an increased P wave duration. It is suggested that the P wave abnormalities in most patients with angina pectoris are the result of intermittent rises of left atrial pressure occurring during episodes of left ventricular ischaemia. Left atrial hypertrophy rather than left atrial dilatation appears to be the more likely anatomical cause of the P wave abnormalities; this possibility requires investigation by anatomical studies.

References

- Bachmann, G. (1941). The significance of splitting of the P wave in the electrocardiogram. *Annals of Internal Medicine*, **14**, 1702-1709.
- Bethell, H. J. N., and Nixon, P. G. F. (1972). P wave of electrocardiogram in early ischaemic heart disease. *British Heart Journal*, **34**, 1170-1175.
- Brown, O. R., Harrison, D. C., and Popp, R. L. (1974). An improved method for echographic detection of left atrial enlargement. *Circulation*, **50**, 58-64.
- Chandraratna, P. A. N., and Hodges, M. (1973). Electrocardiographic evidence of left atrial hypertension in acute myocardial infarction. *Circulation*, **47**, 493-498.
- Gooch, A., Calatayud, J. B., Rogers, J. B., and Gorman, P. A. (1966). Analysis of P wave in severe aortic stenosis. *Diseases of the Chest*, **49**, 459-463.
- Grossman, J. I., and Delman, A. J. (1969). Serial P wave changes in acute myocardial infarction. *American Heart Journal*, **77**, 336-341.
- Heikkilä, J., and Luomanmäki, K. (1970). Value of serial P wave changes in indicating left heart failure in myocardial infarction. *British Heart Journal*, **32**, 510-517.
- Kasparian, H., and Wiener, L. (1968). Left ventricular compliance and volume changes in coronary heart disease. *Circulation*, **38**, 240.
- Kasser, I., and Kennedy, J. W. (1969). The relationship of increased left atrial volume and pressure to abnormal P waves on the electrocardiogram. *Circulation*, **39**, 339-343.
- Massie, E., and Walsh, T. J. (1960). *Clinical Vectorcardiography and Electrocardiography*. Yearbook Medical Publishers, Chicago.
- Master, A. M. (1933). P wave changes in acute coronary artery occlusion. *American Heart Journal*, **8**, 462-470.
- Mazzoleni, A., Wolff, R., Wolff, L., and Reiner, L. (1964). Correlation between component cardiac weights and electrographic patterns in 185 cases. *Circulation*, **30**, 808-829.
- Morris, J. J., Estes, E. H., Whalen, R. E., Thompson, H. K., and McIntosh, H. D. (1964). P wave analysis in valvular heart disease. *Circulation*, **29**, 242-252.
- Pirani, C. L., and Langendorf, R. (1949). Pathology of the cardiac atria. An anatomic-electrocardiographic correlation

- based on a study of 85 human hearts (abstract). *American Heart Journal*, **37**, 663.
- Rios, J. C., Schatz, J., and Meshel, J. C. (1974). P wave analysis in coronary artery disease: an electrocardiographic-angiographic and hemodynamic correlation. *Chest*, **66**, 146-150.
- Romhilt, D. W., Bove, K. E., Conradi, S., and Scott, R. C. (1972). Morphologic significance of left atrial involvement. *American Heart Journal*, **83**, 322-327.
- Romhilt, D. W., and Scott, R. C. (1972). Left atrial involvement in acute pulmonary edema. *American Heart Journal*, **83**, 328-331.
- Ross, G. (1963). Effect of hypertension on the P wave of the electrocardiogram. *British Heart Journal*, **25**, 460-464.
- Rubler, S., Shah, N. N., and Moallem, A. (1976). Comparison of left atrial size and pulmonary capillary pressure with P wave of electrocardiogram. *American Heart Journal*, **92**, 73-78.
- Saunders, J. L., Calatayud, J. B., Schulz, K. J., Maranhao, V., Gooch, A. S., and Goldberg, H. (1967). Evaluation of ECG criteria for P wave abnormalities. *American Heart Journal*, **74**, 757-765.
- Sutnick, A. I., and Soloff, L. A. (1962). Posterior rotation of the atrial vector. An electrocardiographic sign of left ventricular failure. *Circulation*, **26**, 913-916.
- Tarazi, R. C., Miller, A., Frohlich, E. D., and Dustan, H. P. (1966). Electrocardiographic changes reflecting left atrial abnormality in hypertension. *Circulation*, **34**, 818-822.
- Wood, P., and Selzer, A. (1939). A new sign of left ventricular failure. *British Heart Journal*, **1**, 81-85.

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