Electrocardiographic Evidence of Left Atrial Hypertension in Acute Myocardial Infarction

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SUMMARY

The relationship between mean left atrial pressure ($L_{A_m}$) and P-wave abnormalities in lead $V_1$ of standard 12-lead electrocardiograms was evaluated in 30 patients with acute myocardial infarction. In each patient pulmonary artery diastolic pressure or pulmonary wedge pressure was measured, and these values were used as an estimate of $L_{A_m}$. Electrocardiograms recorded at the time of the pressure measurements were used for P-wave analysis. Patients with a history of hypertension were excluded from analysis. There was a significant correlation between the magnitude of the P terminal force in lead $V_1$ (PTF-$V_1$) and the estimated $L_{A_m}$ ($r = -0.78$, $P < 0.001$). Sixteen patients had a normal PTF-$V_1$ on admission; 11 of them had a normal $L_{A_m}$. All 14 patients with an abnormal PTF-$V_1$ (more negative than $-0.03$ mm-sec) on admission had an elevated $L_{A_m}$ ($>12$ mm Hg). Thus, in 25 of the 30 patients, the PTF-$V_1$ correctly identified whether the $L_{A_m}$ was normal or abnormal on admission. On subsequent days, simultaneous measurements of PTF-$V_1$ and estimated $L_{A_m}$ were made in 27 of the patients. Significant changes occurred in both PTF-$V_1$ and $L_{A_m}$; discordant changes were observed on only seven of 56 occasions. Measurement of PTF-$V_1$ appears to be a useful and reliable estimate of left ventricular filling pressure in patients with acute myocardial infarction.

Additional Indexing Words:
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Morrison and his associates, in 1964, investigated the relationship between the P wave of the standard 12-lead electrocardiogram and the presence and severity of valvular heart disease. They found that abnormalities in the terminal half of the P wave in lead $V_1$ correlated well with a pressure or volume overload on the left atrium in patients with valvular heart disease. They also found a significant correlation between left atrial pressure and the P terminal force in lead $V_1$ (PTF-$V_1$, fig. 1). However, they made no observations on patients with acute or transient left atrial involvement. Earlier, Sutnick and Soloff reported transient posterior rotation of the atrial vector during acute left ventricular failure, and other workers observed transient P-wave changes in patients with acute myocardial infarction (MI). More recently, several studies have noted a correlation between P-wave abnormalities and the presence and severity of left ventricular failure in acute MI. Hemodynamic observations were not made in any of these studies, however.

The purpose of the present study was to test the hypothesis that a correlation exists between the magnitude of PTF-$V_1$ and left atrial pressure in patients with acute myocardial infarction.

Methods

The group consisted of 30 patients selected from a consecutive series of the first 51 patients admitted to our Myocardial Infarction Research Unit (MIRU). The reasons for excluding 21 patients were as follows: electrocardiographic evidence of atrial infarction (three patients); serial observation revealed no evidence for an acute MI (four patients); pulmonary artery pressure was not available (six patients); atrial fibrillation (two patients); and a history of previous hypertension (six patients). Each of the 30 patients in the study had a clinical history, electrocardiographic changes, and a serum glutamic oxaloacetic transaminase rise and fall consistent with acute MI. None of the
patients had valvular heart disease, congenital heart disease, chronic pulmonary disease, or chronic congestive heart failure.

The ages of the patients studied ranged from 39 to 83 years. There were 21 males and nine females. At the time of the study, 12 patients had no sign of left ventricular dysfunction, 15 patients had a third heart sound gallop and basal pulmonary rales, two patients were in pulmonary edema, and one patient had cardiogenic shock. Two of the 30 patients (7%) died during hospitalization. Six patients had a history of systemic hypertension prior to hospitalization. Even though none of these had electrocardiographic evidence of left ventricular hypertrophy, they were excluded from analysis because P-wave abnormalities can occur in patients with hypertension in the absence of ECG evidence of left ventricular hypertrophy.9, 10

Right heart catheterization was performed soon after the patient was admitted to the MIRU, using either a no. 6 French Courmand catheter (14 patients) or a no. 5 French flow-directed catheter11 (16 patients). Left atrial mean pressure (LAm) was estimated from the pulmonary artery diastolic pressure in patients in whom the Courmand catheter was used, or from the pulmonary wedge pressure, obtained by inflating the balloon on the flow-directed catheter. In the absence of increased pulmonary vascular resistance, the pulmonary artery diastolic pressure is an accurate reflection of the LAm.12

Pressures were measured using a Statham SP37 strain-gauge transducer, and recorded on a direct-writing recorder. Mean pressures were obtained by electronic integration. Zero reference level for pressure recording was 5 cm below the sternal angle.

Standard 12-lead electrocardiograms were recorded at a paper speed of 25 mm/sec and a sensitivity of 1 mv/cm. P-wave measurements were made using the methods of Morris and associates,3 with special emphasis on the duration of the P wave in lead II, the configuration of the P wave, and the P terminal force in lead V1 (fig. 1). Electrocardiographic tracings and pressure measurements used for correlation analysis were usually made within 2 hours of each other. Measurements of the PTF-V1 and estimated LAm were made on admission and daily thereafter for 3 days. In all patients, follow-up electrocardiograms were recorded at intervals of 3-4 days after their transfer from MIRU to other floors. In our series, we considered a normal value for PTF-V1 to be −0.03 mm-sec or more positive.1 A normal estimated LAm was considered to be 12 mm Hg or less.

**Results**

Bifid P waves ("P mitrale") were observed in only two patients. Four patients had an abnormally
broad (>0.11 sec) P wave in lead II. For the whole group of 30 patients there was no correlation between the width of the P wave and the estimated LA\textsubscript{m} (r = +0.04, P > 0.5).

The relationship between the estimated LA\textsubscript{m} and the magnitude of the PTF-V\textsubscript{1} on admission is shown in figure 2. There is a moderately strong correlation between the two variables (r = -0.78, P < 0.001). The PTF-V\textsubscript{1} was most negative in the patients with the highest estimated LA\textsubscript{m}. Sixteen patients had a normal PTF-V\textsubscript{1} on admission; 11 of these had an estimated LA\textsubscript{m} of 12 mm Hg or less. All 14 patients with a PTF-V\textsubscript{1} more negative than -0.03 mm-sec on admission had an estimated LA\textsubscript{m} greater than 12 mm Hg. Thus, in 25 of the 30 patients (83%), the PTF-V\textsubscript{1} correctly identified whether the LA\textsubscript{m} was normal or abnormal on admission. Furthermore, three of the five patients who had a normal PTF-V\textsubscript{1} and an elevated LA\textsubscript{m} on admission developed an abnormal PTF-V\textsubscript{1} on the second hospital day.

Representative P-wave patterns from two patients are shown in figure 3. Electrocardiograms from a patient admitted twice to the MIRU are reproduced in figure 4. On the first admission he had a subendocardial infarction. Both PTF-V\textsubscript{1} and estimated LA\textsubscript{m} were abnormal on admission, and both returned to normal prior to discharge. Five weeks later he was admitted with a diagnosis of transmural anterior myocardial infarction. Again, both the PTF-V\textsubscript{1} and estimated LA\textsubscript{m} were abnormal, and both returned to normal prior to discharge.

The changes of PTF-V\textsubscript{1} and estimated LA\textsubscript{m} from one day to the next are shown in figure 5. Fifty-six pairs of measurements were available in the 27 patients in whom repeat measurements were made. In many patients significant changes in estimated LA\textsubscript{m} occurred without any change in PTF-V\textsubscript{1}.

![Graph of Relationship between P Terminal Force in Lead V1 and Estimated Mean Left Atrial Pressure](image)

**Figure 2**

Relationship between P terminal force in lead V\textsubscript{1} and estimated mean left atrial pressure on admission. The vertical dotted line represents the upper limit of normal for the estimated mean left atrial pressure. The horizontal dotted line indicates the upper limit of normal for the P terminal force in lead V\textsubscript{1}; any value above this line is abnormal. The regression equation for the data is: estimated LA\textsubscript{m} = 10.4 - 128 × PTF-V\textsubscript{1}. (Standard error of estimate = 5 mm Hg.)

![Electrocardiograms](image)

**Figure 3**

(A) Lead V\textsubscript{1} from patient G.D., who had a diaphragmatic myocardial infarction. The P terminal force in lead V\textsubscript{1} and the pulmonary wedge pressure were normal throughout his stay in the MIRU. (B) Tracings of lead V\textsubscript{1} from a patient with an anteroseptal myocardial infarction. On admission (top) both the pulmonary wedge pressure and the P terminal force in lead V\textsubscript{1} were normal. The wedge pressure and the P terminal force in lead V\textsubscript{1} both returned to normal prior to discharge from the MIRU (bottom).
Clearly discordant changes were rare, however. In only seven of the 56 measurements were the changes of PTF-V1 and estimated \( L_A \) in opposite directions (fig. 5). The PTF-V1 returned to normal in 11 of the 14 patients in whom it was abnormal on admission, usually within 6 days after admission.

**Discussion**

The present study confirms the findings of others\(^5-7\) that patients with acute myocardial infarction often have an abnormal PTF-V1. Furthermore, our results indicate that an abnormal PTF-V1 is usually associated with an elevation of left atrial pressure. The good correlation between the degree of P-wave abnormality in lead V1 and the degree of left atrial hypertension suggests that a cause-and-effect relationship may exist.

Inversion of the terminal portion of the P wave in lead V1 can result from a number of abnormalities: (1) left atrial hypertension or enlargement,\(^13\) (2) atrial infarction,\(^8\) (3) severe pectus excavatum,\(^14\) (4) severe emphysema with low diaphragms,\(^15\) and (5) rarely, in severe chronic right atrial hypertension.\(^15\) In the present study, there were no patients with pectus excavatum, severe emphysema, or severe chronic right atrial hypertension. Since patients with recurrent atrial arrhythmias and elevation or depression of the P-T segment were excluded from the study, it is unlikely that atrial infarction played a significant role in the genesis of the P-wave abnormalities in our patients. Bifid and broad P waves are abnormalities known to be associated with enlargement of the left atrium,\(^16\) but they were rarely seen in this series. This may mean that the left atrium does not dilate very much in left atrial hypertension associated with acute myocardial infarction. In view of these considerations, and the finding of a correlation between PTF-V1 and estimated \( L_A \), it seems reasonable to conclude that an abnormal PTF-V1 is causally related to left atrial hypertension in patients with acute myocardial infarction.

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P-wave abnormalities have been noted in patients with systemic hypertension.6,10 Six of the 51 patients considered for this series had a history of prior systemic hypertension. The values for PTF-V1 and LAa for these six patients were not clearly different from the other 30 patients. However, in view of the uncertainties about P-wave abnormalities in patients with prior systemic hypertension, we feel that estimates of LAa should not be made from PTF-V1 in such patients.

Acute hypoxemia can lead to an increase in pulmonary vascular resistance,17 and it is known that many patients with acute myocardial infarction have arterial hypoxemia.18 Thus, increases in pulmonary vascular resistance could have led to an error in our estimate of LAa in the 14 patients in whom pulmonary artery diastolic pressure was used to estimate LAa. However, the arterial pO2 was 65 mm Hg or lower at the time of study in only two of the 14 patients in whom pulmonary artery diastolic pressure was used. Furthermore, separate analyses gave identical results for the relationship between PTF-V1 and pulmonary artery diastolic pressure (14 patients), and PTF-V1 and pulmonary wedge pressure (16 patients).

Serial observations of the PTF-V1 and estimated LAa revealed that clearly discordant changes were rare. Although changes in estimated LAa were often accompanied by no change in PTF-V1, changes of PTF-V1 toward normal were usually accompanied by a reduction in estimated LAa (fig. 5). Moreover, in the four patients in whom PTF-V1 returned toward normal and estimated LAa increased, the LAa increases were only 3 mm Hg or less. Thus, it appears that changes toward normal of PTF-V1 can be taken as an indication that the LAa is either decreasing or remaining stable. In 11 of 14 patients the abnormal PTF-V1 measured on admission returned to normal before discharge from the hospital. This regression suggests that the abnormal PTF-V1 occurred acutely after the myocardial infarction. Since the maximum abnormality of the PTF-V1 was noted on admission in the majority of our cases, it is likely that the P-wave changes occur within a few hours of the acute elevation of left atrial pressure.

It is of interest that two recent communications support our conclusions. Hugenholtz, Heikkilä, and Nelson19 also found a good correlation (r = -0.82) between the pulmonary wedge pressure and PTF-V1. Romhilt and Scott20 found abnormal values for PTF-V1 in patients admitted to the hospital with acute pulmonary edema. During recovery, the PTF-V1 returned to normal in 72% of their patients.

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