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# Relationship between P Wave Form and Left Atrial Pressure and Volume in Mitral Valve Disease

BENGT FURBERG

*From the Departments of Clinical Physiology and Internal Medicine, University Hospital, Uppsala, Sweden*

## ABSTRACT

**The P wave of the ECG, recorded with both conventional and special techniques, was compared with left atrial (LA) pressure and volume in 22 patients with mitral valve disease. The patients were also compared with 2 groups of normal subjects as to the P wave, recorded with the special technique. Changes in the P wave, such as increased duration, increased bipeak interval or P terminal force were significantly more common in the patients than in normal subjects. Among the patients, no significant correlations were found between degreee of LA pressure elevation, LA enlargement and P wave changes. It is concluded that for the diagnosis of increased LA load it is more useful to study the terminal vector of the P wave than signs of increased atrial asynchrony. Recordings with high amplification and paper speed as in the present special technique are of advantage for such measurements. However, the special leads by themselves were not ideal for study of an increased terminal vector directed to the left and backwards.**

## INTRODUCTION

A broad, notched P wave was first described by Lewis (11) in 1914 in patients with mitral stenosis and later termed 'P mitrale' by Winternitz (20) in 1935. Since then a number of methods for analysing the P wave have been devised to detect left atrial (LA) involvement in mitral valve disease. In addition to a prolonged duration of the P wave, a bipeak interval of more than 4 cs, a ratio of P duration to PR segment greater than 1.60, and a mean P axis of +30° or more to the left have been described as indicative of LA enlargement or hypertrophy. The recognition of the posteriorly and leftward rotated terminal vector (15) in LA enlargement has led to additional expressions such as the duration and amplitude of the negative P deflection in lead $V_1$ and the 'P terminal force', according to Morris et al. (13). A drawback with many such studies is that they have been made on recordings with low (i.e. conventional) amplification and paper speed, thus making precise measurements of the P wave difficult.

The aim of the present investigation was to compare the P wave form, recorded both with the conventional and a special technique, with LA pressure and LA volume in patients with mitral valve disease. The aim was also to compare the patients with a group of normal persons as to the P wave, recorded with the special technique.

## MATERIAL AND METHODS

Two control groups were investigated with the special recording technique. Each group consisted of 52 persons, those of the first group being 40 years old or less (range 19–40, mean age 27), those of the other being more than 40 years old (range 42–65, mean age 51), all without any known cardiovascular disease. The patient group consisted of 22 patients (10 men and 12 women between 27 and 65, mean age 49) with mitral stenosis and/or mitral insufficiency. Five of the patients also had aortic valve disease. All the patients had sinus rhythm. Of the patients, 17 had an LA or pulmonary capillary venous (PCV) pressure of at least 16 mmHg at rest, while an abnormal pressure elevation (to at least 30 mmHg) occurred during exercise in the other 5 patients. The patients were selected from the group of mitral cases at our hospital; all patients with only slight symptoms (not catheterized), as well as those who had atrial fibrillation or atrial flutter, were eliminated from the study.

Right heart catheterization with pressure and flow measurements at rest was performed on all patients in the supine position, and during exercise in all but 4 patients. In 6 patients a transseptal catheterization with measurement of the left atrial pressure was also performed. As reference point for the pressure recordings, the thoracic midpoint at the level of the fourth rib–sternal insertion was used.

Classification of the LA volume was made by an experienced roentgenologist from anteroposterior and lateral chest roentgenograms taken with the patients in the standing position. LA enlargement was expressed on a scale of 0 to 3 (no–mild–moderate–pronounced LA enlargement).

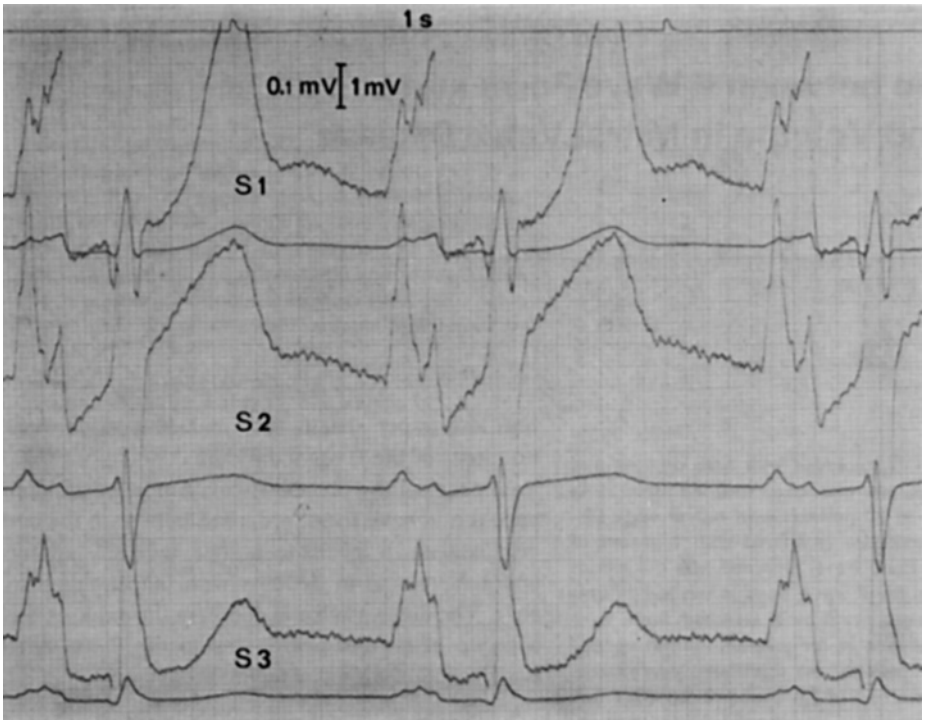


*Fig. 1.* Example of recording with the special technique in a patient with mitral valve disease. Paper speed 100 mm/s. 1 mV=10 mm in channels 2, 4 and 6, 0.1 mV=10 mm in channels 1, 3 and 5.

A conventional 12-lead ECG (amplification 1 mV=10 mm, paper speed 50 mm/s) was recorded, using a direct-writing ECG machine (Mingograph 61, Siemens-Elema Ltd., Sweden). In addition, a special recording technique (14) was used, three bipolar chest leads being recorded. The common reference electrode was placed at the midline of the sternal angle and three different electrodes were placed at the highest attainable point of the armpit in the left mid-axillary line (S 1), at the caudal end of the sternal body (S 2) and at a point over the vertebral column at the transthoracic level of the sternal angle (S 3). The three bipolar chest leads were recorded with both conventional amplification (1 mV=10 mm) and with tenfold amplification, using a 3-channel differential preamplifier. The recording was made with a Mingograph 81, with a paper speed of 100 mm/s.

The duration of the P wave in lead II, the maximum bipeak interval in bifid P waves in any lead (19), and the P terminal force (13) in $V_1$ were measured in the conventional recordings. In the special recordings the duration of the P wave was measured in lead S 2 and the bipeak interval in bifid P waves in the lead where it was maximal.

## RESULTS

The average duration of the P wave in lead S 2 in the younger control group was 10.7 cs (S.D. 0.93 cs, range 9–12 cs). In the older control group the average duration was 11.4 cs (S.D. 1.00 cs, range 10–14 cs), the difference being significant ($p<0.001$). From these results, a value of 12 cs may be accepted as the upper normal limit for P duration in lead S 2 in persons below 40 and 13 cs in persons above 40. Fig. 1 shows an example of a recording with the special technique in a patient with mitral valve disease. Table I shows P wave variables in patients with different degree of LA enlargement. The mean values and ranges demonstrate that there is no significant difference as to P duration or bipeak interval, either in the special or the conventional recordings, between mild, moderate and pronounced LA enlargement in our cases.

With the present criteria for normality, 13 of the 22 patients with mitral valve disease had an abnormal P wave duration in the special recording. In lead II of the conventional ECG, a P wave duration of more than 11 cs was found in 17 patients, of more than 12 cs in 10 patients. In the conventional ECG, a bipeak interval of more than 4 cs was found in 13 patients, of more than 5 cs in 9 patients. The mean values for P terminal force became more negative

Table I. *Relationship between P wave form, left atrial pressure at rest and degree of left atrial enlargement in patients with mitral valve disease*

0=no, 1=mild, 2=moderate, 3=pronounced left atrial enlargement

| | Left atrial enlargement | | | |
|---|---|---|---|---|
| | 0 | 1 | 2 | 3 |
| *Special recording* | | | | |
| P duration lead S 2 (cs) | | | | |
| *n* | 2 | 5 | 9 | 6 |
| $\bar{x}$ | 12.0 | 13.8 | 14.8 | 13.8 |
| Range | 10–14 | 12–17 | 12–19 | 12–16 |
| Bipeak interval (cs) | | | | |
| *n* | 1 | 3 | 9 | 5 |
| $\bar{x}$ | 6.0 | 7.0 | 7.2 | 6.6 |
| Range | | 4–9 | 3–12 | 4–8 |
| *Conventional recording* | | | | |
| P duration lead II (cs) | | | | |
| *n* | 2 | 5 | 9 | 6 |
| $\bar{x}$ | 11.0 | 12.0 | 13.3 | 13.2 |
| Range | 10–12 | 10–16 | 12–16 | 10–16 |
| Bipeak interval (cs) | | | | |
| *n* | 1 | 2 | 7 | 5 |
| $\bar{x}$ | 7.0 | 7.0 | 6.4 | 5.4 |
| Range | | 5–9 | 4–8 | 4–8 |
| P terminal force, lead $V_1$ ($\mu V \cdot s$) | | | | |
| *n* | 2 | 5 | 9 | 6 |
| $\bar{x}$ | −3.5 | −4.0 | −6.6 | −8.2 |
| Range | −3 to −4 | −2 to −8 | 0 to −14 | −3 to −27 |
| LA (PCV) pressure at rest (mmHg) | | | | |
| *n* | 2 | 5 | 9 | 6 |
| $\bar{x}$ | 19.0 | 21.2 | 25.1 | 20.8 |
| Range | 18–20 | 15–29 | 13–39 | 15–33 |

with increasing atrial enlargement. However, this tendency is weakened if one patient with a P terminal force of −27 $\mu V \cdot s$ is excluded from enlargement group 3. A P terminal force more negative than −3 $\mu V \cdot s$ was found in 14 patients.

With 11 cs, 4 cs and −3 $\mu V \cdot s$ as highest normal values for P duration in lead II, P bipeak interval and P terminal force at least one P wave abnormality was found in 18 of the 22 patients with mitral valve disease. With the same criteria at least one P wave abnormality was found in 41 of 152 normal subjects (1). The difference is statistically significant ($p<0.001$).

The special technique used in the present study was analysed as to its capacity to display the terminal part of the P wave. The study revealed that none of the three special leads was ideal for the study of an increased terminal vector directed to the left and backwards.

Table II shows the P wave variables in relation to the different levels of LA or PCV pressure at rest. No consistent tendency or significant correlation was observed.

## DISCUSSION

The usual explanation for the occurrence of broad and notched P waves is that they represent LA enlargement or hypertrophy, or both. However, deeply notched P waves of greater than normal duration may be attributed to asynchronous activation due to defective inter-atrial conduction without atrial enlargement, as reported by Bachmann (3), Feiring (6) and Decherd et al. (5). Broad and notched P waves are therefore not specific signs of LA enlargement. Thomas & Dejong (19) suggested the term 'left atrial abnormality' (LAA) for P waves with signs of increased atrial asynchrony.

The mechanism of P wave changes in mitral valve disease obviously depends on the way electrical activity is normally spread in the atria. In 1910,

Table II. *Relationship between P wave form and left atrial pressure at rest in patients with mitral valve disease*

| | LA (PCV) pressure at rest, mmHg | | |
|---|---|---|---|
| | 10–19 | 20–29 | 30–39 |
| *Special recording* | | | |
| P duration lead S 2 (cs) | | | |
| *n* | 10 | 8 | 4 |
| $\bar{x}$ | 14.3 | 13.6 | 14.3 |
| Range | 10–19 | 12–18 | 13–17 |
| Bipeak interval (cs) | | | |
| *n* | 8 | 6 | 4 |
| $\bar{x}$ | 7.6 | 6.3 | 6.5 |
| Range | 3–12 | 4–9 | 4–8 |
| *Conventional recording* | | | |
| P duration lead II (cs) | | | |
| *n* | 10 | 8 | 4 |
| $\bar{x}$ | 13.4 | 11.8 | 13.3 |
| Range | 10–16 | 10–14 | 12–16 |
| Bipeak interval (cs) | | | |
| *n* | 7 | 5 | 3 |
| $\bar{x}$ | 6.4 | 5.6 | 6.7 |
| Range | 5–9 | 4–7 | 6–8 |
| P terminal force, lead $V_1$ ($\mu \cdot s$) | | | |
| *n* | 10 | 8 | 4 |
| $\bar{x}$ | −5.8 | −5.4 | −8.5 |
| Range | 0 to −27 | −1 to −12 | −6 to −13 |

Lewis et al. (12) denied the existence of a special conduction system in the atria, characterizing atrial conduction instead as continuous. In 1916, however, Bachmann (2) described a myocardial bundle connecting the right and left atria and was convinced that the normal sinus impulse was preferentially conducted through it to the left atrium.

Only within the past decade has it been widely accepted that there is an atrial specific conduction system with one interatrial and three internodal pathways. The arguments in favour of this have been extensively reviewed by James & Scherf (9). The significance of focal atrial lesions has been confirmed by Beeson & Teabeaut (4), who found microscopically advanced fibrosis of Bachmann's bundle in cases of mitral stenosis. If the P wave changes indicating increased atrial asynchrony in mitral valve disease are caused by fibrosis in the atrial conduction system, unrelated to degree of LA enlargement and instead dependent on, e.g., degree of rheumatic inflammatory reaction, it is not difficult to explain the absence of correlation between P wave change and degree of LA enlargement or elevation of the LA pressure, as found both in the present study and earlier, by other authors (16, 18).

The present study did not show any significant correlation between mean LA pressure and LA volume. This finding is similar to that of Soloff & Zatuchni (18) and Sauter et al. (17). Factors such as duration of the pressure elevation and degree of fibrosis of the atrial myocardium may be of importance for this lack of correlation.

Goldman (7) gives an upper limit of 11 cs for the P wave duration in lead II. With the recording technique we have used, this limit seems to give too many false-positive results. In a group of 152 middle-aged men without known cardiovascular disease, 31% had increased P wave duration when 11 cs was used as upper normal limit (1). It would seem that 12 cs (or 13 cs for older persons) is to be preferred as an upper normal limit for the P wave duration, even in conventional recordings.

Thomas & Dejong considered a bipeak interval of more than 4 cs as a sign of LA abnormality. With this criterion, 9% of 152 middle-aged men without known cardiovascular disease had signs of LA abnormality (1). An upper normal limit of 5 cs is therefore to be preferred.

Morris et al. (13) consider a P terminal force more negative than $-3\ \mu V \cdot s$ as a sign of LA abnormality. Less than 3% of 152 control subjects (1) fulfilled this criterion. Thus, the occurrence of false-positive results seems to be low enough. With this criterion, an abnormal P terminal force was found in 64% of our patients with mitral valve disease; this was the method with the highest sensitivity.

In patients with acute left heart failure, Heikkilä et al. (8) found that the P terminal force was well correlated to the level of LA mean pressure. Others (10, 13) found a significant correlation between P terminal force and LA volume and/or pressure in cases with mitral valve disease. These findings could be explained by a change of the activation of myocardial cells in the dilated or hypertrophied atrium, causing a change in the terminal part of the P loop, and need not indicate a lesion in the atrial conduction system. While prolonged duration of the P wave, increased bipeak interval and P/PR ratio appear to be indications of increased atrial asynchrony, the 'P terminal force' thus may represent a functional change due to LA dilatation or hypertrophy which may occur without significant prolongation of the P wave.

Signs of increased atrial asynchrony seem to be diagnostically useful only for discriminating patients with LA abnormality from normal persons. Changes of the terminal portion of the P wave in lead $V_1$ may have some usefulness for prediction of the degree of LA enlargement or pressure elevation, although without much precision. To improve the measurement of the terminal part of the P loop, it seems reasonable to use recordings with higher amplification and paper speed than conventional ones. Recordings of lead $V_1$ with high amplification and paper speed may be the best method for clinical routine use.

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Address for reprints:

Bengt Furberg
Department of Clinical Physiology
Central Hospital
S-800 07 Gävle
Sweden