A NEW SIGN OF LEFT VENTRICULAR FAILURE

BY

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The prominent P wave associated with mitral stenosis is well recognized; it is usually widened and is often notched, bifid, or flat-topped; it is generally thought to be a result of left auricular hypertrophy. There is also some evidence that a tall spiked P wave may be produced by right auricular hypertrophy, for such a wave is rarely found except in cases of tricuspid stenosis, congenital pulmonary stenosis, and chronic cor pulmonale. It is the purpose of this communication to draw attention to a third type of P wave which we have found to be commonly associated with hypertensive heart disease, especially when there is left ventricular failure. This P wave is widened and of low voltage; it is also usually bifid or flat-topped. Typical examples of these three types of P wave are shown in Figs. 1–3.



Fig. 1.—Prominent bifid and widened P wave in a case of mitral stenosis.

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The normal P wave may be notched, but its duration does not exceed 0·1 sec. Its voltage in the favourable lead rarely exceeds 0·25 mv. and averages a

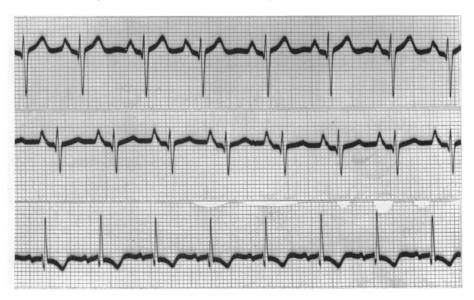


Fig. 2.—Tall spiked P wave in a case of chronic cor pulmonale (proved at autopsy).



Fig. 3.—Wide bifid P wave of the low voltage in a case of hypertensive heart disease with left ventricular failure.

little under 0·15 mv. The P wave to which we are drawing attention commonly measures 0·12 sec. in duration and rarely more than 0·1 mv. in amplitude.

In a series of 1000 consecutive routine electrocardiograms this P wave was

found in 25 instances, or in 2.5 per cent. Four of these were cases of aortic valvular disease, and the remainder were cases of hypertensive heart disease. In the same series there were 25 instances of abnormal P waves associated with mitral stenosis, and five instances of tall spiked P waves with chronic cor pulmonale or with congenital pulmonary stenosis. It is therefore evident that this P wave is as common as the P wave of mitral stenosis, and about five times as common as the tall spiked P wave associated with chronic cor pulmonale or with congenital pulmonary stenosis.

To determine the significance of this P wave, we studied 70 patients with hypertensive heart disease and 25 with uncomplicated aortic valvular disease in various stages of development. As a control group we used 25 patients with ischæmic heart disease without cardiac enlargement. They were examined clinically, radiologically, and electrocardiographically, and no one was included where the diagnosis was in doubt. We divided the patients with hypertensive heart disease and those with aortic valvular disease into five groups which were determined as follows:

Group A .. Those with no symptoms.

Group B .. Those with slight effort dyspnæa.

Group C .. Those with moderate or severe effort dyspnæa.

Group D ... Those with left ventricular failure.

Group E ... Those with right ventricular failure.

The diagnosis of left ventricular failure was made upon the presence of several of the following criteria: orthopnœa, paroxysmal cardiac dyspnœa, pulmonary œdema, persistent bilateral basal pulmonary rales, prolongation of the arm to tongue circulation time, radiological evidence of pulmonary congestion, pulsus alternans and gallop rhythm without bundle branch block. The diagnosis of right ventricular failure was made upon the finding of engorgement of the veins of the neck with enlargement and tenderness of the liver. The presence of dropsy was not considered essential for the diagnosis of right ventricular failure, nor was the latter diagnosis necessarily made because there was dropsy. Of course, all patients with failure of the right ventricle also had failure of the left ventricle.

The electrocardiograms were then analysed with regard to the P waves. These were classified as normal if they lasted for less than 0·1 sec., as doubtful if from 0·1 to 0·11 sec., and as widened if they exceeded 0·11 sec. We considered that the width of the P wave was the essential feature. Widened P waves occurred most frequently in lead II, but were often found in more than one lead. In some cases the bifid nature of P was so marked as to warrant the term "double P wave," for it consisted of two separate deflections, the one immediately following the other and each measuring 0·06 sec. in duration.

The results of this study are presented in Table I. In the hypertensive cases the incidence of a widened P wave increased from zero in the symptomless group to 61 per cent. in the group with left and right ventricular failure. It is important to note that there was no real difference in the incidence of a widened P wave in the group with left ventricular failure alone and the group with left and

right ventricular failure. Similar, but less convincing, results were obtained in the cases of aortic valvular disease. In the control group of 25 cases of ischæmic heart disease a widened P wave did not occur.

	CLASS	P WAVE			NO. OF CASES
		>0.11"	0.1—0.11"	<0.1"	
Hypertensive heart disease	A B C D E	1 (4%) 2 (15%) 7 (50%) 8 (61%)	- 4 (30%) 2 (14%) 1 (8%)	6 (100 %) 23 (96 %) 7 (55 %) 5 (36 %) 4 (31 %)	6 24 13 14 13
Aortic valvular disease	A B C D E			1 5 3 4	0 3 7 8 5
Ischæmic heart disease		_	2	23	25

TABLE I.—RELATIONSHIP OF WIDTH OF P AND DEGREE OF CARDIAC FAILURE

It is evident that the type of P wave which we have described is commonly associated with left ventricular failure.

DISCUSSION

The cause of the abnormal P waves in mitral stenosis has been discussed by several authors. Although von Boros and Trendelenburg believe that an intraauricular conduction defect is responsible, the majority think that the explanation lies in hypertrophy of the left auricle. Some attempts have been made to diagnose from the lead in which the notched P wave occurs (Winternitz) or from the shape of the notching (Holzmann) which auricle is affected, but there is insufficient evidence for these views. In a recent study, Berliner and Master tried to correlate electrocardiographic and post-mortem findings in cases of mitral stenosis. They found hypertrophy of the left auricle in cases with bifid and widened P waves, and hypertrophy of the right auricle in addition in cases in which the P wave was increased in height. Their tallest P waves occurred in cases of combined mitral and tricuspid stenosis. They suggested that a tall P wave might result from a summation effect from hypertrophy of both auricles. Because we are familiar with the tall spiked P wave associated with pulmonary stenosis and chronic cor pulmonale, and because we have noticed that, though tall, the P wave is not widened in these conditions, we suggest that an increase in voltage of the P wave may indicate hypertrophy of the right auricle, whereas widening and notching of the P wave may indicate hypertrophy of the left auricle.

If this view should prove correct, then there is a ready explanation for the wide bifid P wave associated with left ventricular failure, for in this condition an

increased stress falls upon the left auricle. It is tempting to suggest that this type of P wave is yet another sign, and an early one, of left ventricular failure, to be ranked with gallop rhythm.

This is but a preliminary report and is published in the hope that it may stimulate investigation in this field. With the aid of an œsophageal lead, a semi-direct right auricular lead, and careful clinical and post-mortem investigation it should not be difficult to solve the problem. We already have evidence that chest leads taken from the right border of the sternum and paired with the right arm electrode may yield a tall spiked narrow P wave in cases with hypertrophy of the right auricle.

SUMMARY

- 1. A widened P wave of low voltage, usually bifid or flat-topped, has been found in association with left ventricular failure in cases of hypertensive heart disease and of aortic incompetence.
 - 2. It is suggested that this P wave results from left auricular stress.

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