Examination of the P wave in lead V_1 of the electrocardiogram: its value in routine electrocardiography

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SUMMARY. While reporting on the electrocardiograms recorded in this Department during the past four-and-a-half years, interest was aroused by the occasional appearance of a terminal negative component in the P wave in lead V_1. I tried to find out what significance, if any, it had. In the ensuing study a relationship between such a negative component—expressed as a terminal negative force (-ve Ptf V_1 for short)—and the presence of ischaemic heart disease emerged. Such a negative Ptf V_1 is not an infallible marker of ischaemic heart disease nor does it necessarily persist in the repeat electrocardiogram of any given patient. It is, however, suggested that attention be routinely paid to the morphology of the P wave in lead V_1 and that when a negative terminal component is present in this wave follow-up study of this feature in serial electrocardiograms may help in the assessment of prognosis in any given patient.

Introduction

The first half of the P wave represents depolarisation of the right atrium, the terminal half depolarisation of the left atrium. The morphology of the P wave is best shown in chest lead V_1. When the left atrium is overloaded it rotates to the left and posteriorly in the horizontal plane causing a well marked negative component of the P wave in this lead. This terminal negative component can be expressed as a force and its size can be quantified by multiplying its depth in millimetres by its duration in seconds. Figure 1 shows a Ptf V_1 of −0.04 mm/sec.

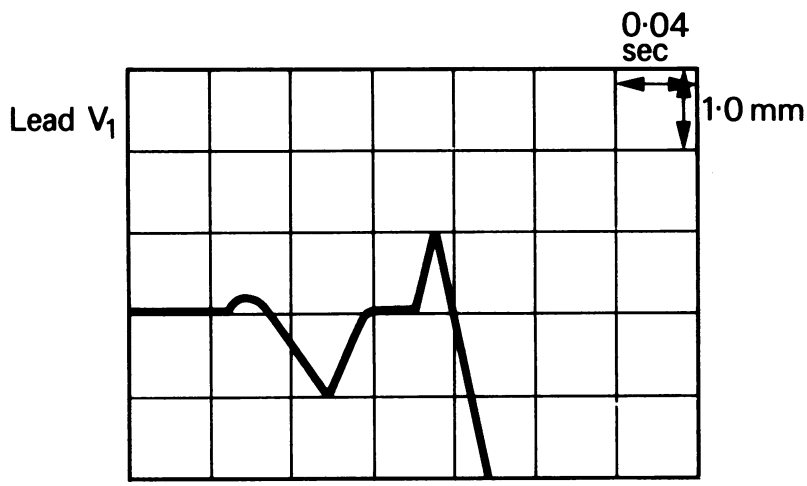

Figure 1

The terminal force of the P wave in lead V_1 (Ptf V_1) is calculated by multiplying its amplitude in mm by its duration in seconds.
In this example, Ptf measures −0.04mm/sec.

Aim

To determine the significance, if any, of the appearance of a terminal negative component in the P wave in lead V_1.

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Method

All the electrocardiograms (ECGs) recorded in this Department between 1 July 1970 and 31 December 1974 were studied. Each record consisted of the usual 12 leads and in the latter half of the period leads V_3R and V_4R were added. There were 335 recordings from 264 patients, 125 men (aged 21–82) and 139 women (aged 12–87). Only tracings in normal sinus rhythm and of good quality were considered. The ECGs of 20 patients (seven men and 13 women) had to be discarded either through technical faults in the tracing or because they were not in normal sinus rhythm at the time of electrocardiography.

In the tracings of the 244 patients remaining three groups could be identified:

Group A. In this group Ptf V_1 was equal to, or more negative than, —0.04 mm/sec.

Group B. In this group Ptf V_1, although still negative, was less negative than —0.04 mm/sec.

Group C. In this group the P wave was either wholly positive or was iso-electric. (For accurate measurement a good hand-lens is essential.)

Group A

There were nine patients (five men and four women) in this group. Four of the men had electrocardiographic (ECG) evidence of ischaemic heart disease (three with the recognised hallmarks of myocardial infarction). The remaining man satisfied the accepted ECG criteria of left ventricular hypertrophy. Of the women two had ECG evidence of ischaemic heart disease without infarction, one had left bundle branch block and the fourth (on treatment for diastolic hypertension and diabetes mellitus) had no ECG evidence of heart disease.

Group B

There were 34 patients (18 men and 16 women) in this group: in each case Ptf V_1, although negative, was less negative, than —0.04 mm/sec. Sixteen of these patients (eight men and eight women) showed ECG evidence of ischaemic heart disease. This group is not, however, considered in detail as the cut-off point for inclusion in the Ptf V_1: negative category had for the purposes of this study been fixed at: 0.04 mm/sec.

Group C

From the 201 patients whose Ptf V_1 was either positive or iso-electric a random sample of 25 men and 25 women was drawn with the aid of a table of random sampling numbers. The age range of the men was 30–81 and of the women 36–84 years. Of the 25 men two showed ECG evidence of ischaemic heart disease both with the hallmarks of myocardial infarction, and two showed sinus bradycardia (ventricular rate under 60 per minute). The remaining 21 had no ECG evidence of heart disease—of these, two presented with diastolic hypertension at the date of electrocardiography. Among the 25 women there were four examples of electrocardiographic ischaemic heart disease (one with evidence of infarction), one showed ECG evidence of left ventricular hypertrophy and one had " minor non-specific changes ". The remaining 19 had no ECG evidence of heart disease —of these, three had diastolic hypertension at the date of electrocarciography.

Summarising the figures in Groups A and C: of nine patients in whom Ptf V_1 was equal to or more negative than —0.04 mm/sec., six showed ECG evidence of ischaemic heart disease. In a random sample of 50 patients drawn from the same population in whom Ptf V_1 was either positive or iso-electric there was ECG evidence of ischaemic heart disease in six. Although the numbers in the first group are admittedly small, the difference is statistically significant.

In order to discover whether these findings are applicable to cardiological practice I was given access to the records of the Electrocardiography Department, through the

courtesy of Dr (now Professor) D. G. Julian. From the large number of recordings (about 25,000) made in 1971, I drew a statistically random sample of 100 men (aged 26–84) and 100 women (aged 12–87).

For the purposes of this study the ECGs of four patients had to be rejected on technical grounds. Of the remaining 196, 31 (18 men and 13 women) had tracings in which the terminal force of the P wave in lead V_1 measured —0.04 mm/sec. or more. Subsequent examination of the ECG reports of these 31 patients showed that in 18 (11 men—four with infarction and seven women—five with infarction) the presence of ischaemic heart disease was recorded. The following diagnoses had been allotted to the remaining 13 patients (six men and seven women)—P. pulmonale—one;? digitalis effect—one; left bundle branch block—one; sinus tachycardia—one; cerebrovascular accident? myocardial infarct—one; first degree heart block—two; normal sinus rhythm —six.

From the ECG recordings of the remaining 165 patients in whom Ptf V_1 was either positive or iso-electric a statistically random sample of 25 men (aged 38–83) and 25 women (aged 17–85) was drawn. In this sample the ECG reports were as follows:

A. *Men.*—Ischaemic heart disease—three (including two with myocardial infarction), sinus bradycardia—three, non-specific T wave changes—two, first degree heart block, left bundle branch block and partial right bundle branch block—one each, normal ECG—14.

B. *Women.*—Ischaemic heart disease—two (no examples of myocardial infarction), sinus tachycardia—two, sinus bradycardia—one, non-specific T wave changes—one, ? hypokalaemia—one, normal ECG—18.

In summary, of 31 patients (18 men and 13 women) whose Ptf V_1 measured — 0.04 mm/sec. or more, 18 (11 men and seven women) showed definite ECG evidence of ischaemic heart disease. In a random sample of 25 men and 25 women drawn from the same population whose Ptf V_1 was either positive or iso-electric there was ECG evidence of ischaemic heart disease in five (three men and two women). The difference in the incidence of ischaemic heart disease in the two groups is statistically significant.

Discussion

As long ago as 1933 Master pointed out that P wave changes can occur after acute coronary occlusion. He studied only the standard limb leads and he noted an increase, up to 2.00 mm or more, in the height of the P wave, most evident in lead II. This occurred in 32 of 40 patients suffering from recent coronary occlusion. The increase in height was most marked during the first few days of the illness and there was a return to the normal height later. Master suggested that this finding is due to " acute auricular dilatation ".

The concept of the terminal force of the P wave in lead V_1 and the method of quantifying this force were first suggested by Morris and his colleagues in 1964. They concluded that the P wave measurement most effectively distinguishing between normal subjects and those with left-sided valvular disease is that centred about the terminal portion of the P wave in lead V_1. They also suggested that the value is abnormal if it is more negative than —0.03 mm/sec. Studies on Ptf V_1 after the onset of ischaemic heart disease have been recorded by, among others, Sutnick and Soloff (1962), Heikkilä and Luomanmäki (1970), Heikkilä, Hugenholtz and Tabakin (1973) and, in this country, by Hassell and Hoffbrand (1970) and by Bethell and Nixon (1972, 1974). Kasser and Kennedy (1969) pointed out that changes in the P wave terminal force show a highly significant correlation with left atrial volume, but relate less well to changes in left atrial pressure, while Romhilt *et al.* (1972) define left atrial involvement as terminal negativity in the P wave in lead V_1 of 1.00 mm or more in depth and 0.04 seconds or more in duration. Heikkilä, Hugenholtz and Tabakin (1973) regard alterations in the Ptf V_1 as a useful and simple non-invasive tool for the quantitative assessment of acute changes in left

ventricular pre-load in seriously ill patients while Bethell and Nixon (1972) urge that Ptf V_1 more negative than -0.02 mm/sec. in an apparently normal subject should lead to a suspicion of ischaemic heart disease, and to further investigation.

In my study of two separate outpatient populations a relationship has been found between a negative component in the P wave in chest lead V_1 and the occurrence of ischaemic heart disease. It must, however, be realised that the size and form of the terminal component in this lead are not invariable in any given patient. Of 22 patients in our Department in whom there was initially no negative component in the P wave in lead V_1 five showed some degree of negativity on repeat electrocardiograms. On the other hand, three of ten patients who initially showed some negativity in Ptf V_1 reverted subsequently to a wholly positive P wave in that lead. Such changes are probably due to increase or decrease in left atrial volume: these in turn may well be determined, in cases of damage to the myocardium of the left ventricle, by corresponding changes in left ventricular stroke volume or left ventricular compliance.

It is suggested that a note of the morphology of the P wave in lead V_1 should be included in the study of every electrocardiogram. The additional scrutiny involved is not time-consuming and the occurrence of a negative component in the terminal portion of this wave should alert one to the possibility of left atrial overload with all its complications. Follow-up studies of such 'negative' cases may yield results of prognostic value.

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EVALUATION OF AN AMBULATORY MEDICAL-CARE DELIVERY SYSTEM

The authors designed a medical-care delivery system specifically to relieve the congestion and long waits that resulted from the substitution of personal fees by prepaid plans, Medicare and other third-party payment plans. Patients went first of all not to a doctor, but to a paramedically-staffed health evaluation service (questionnaires and screening tests) that divided them into four groups, the well, the worried well, the asymptomatic sick and the sick. The result was to make it possible for one doctor and a team of paramedical staff to look after 110 new patients and 306 return patients in one working day. The overall costs were also considerably reduced. Nearly all the patients and most of the staff preferred the new system.

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