The Terminal P Wave Vector in Lead V_1 of the Electrocardiogram in Healthy Young Airmen

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SUMMARY: The terminal P wave vector in lead V_1 of the electrocardiograph (V_1 PTV) was abnormal in 93 (3.2%) of 3119 healthy recruits into the Royal Air Force of mean age 19 (SD 4) years. The inference that an abnormal V_1 PTV indicates left atrial enlargement should be made with caution in otherwise healthy young men.

Introduction

The P wave in lead V_1 of the electrocardiogram is normally upright but occasionally shows a small terminal negative deflection, known as the V_1 P terminal vector (V_1 PTV), which has been shown to represent left atrial depolarisation alone¹. In 1964 Morris et al² gave a quantitative assessment of V_1 PTV as the product of the duration of the wave in seconds and its amplitude in millimetres and propounded that when this product was equal to or more negative than -0.04 millimetre seconds (ie. in routine electrocardiographs one small square wide and one small square deep) it was abnormal. They found this abnormality correlated with raised left atrial pressure and with mitral valve area in over 90% of cases of mitral stenosis. Since then an abnormal V_1 PTV has been found to correlate with raised ventricular end-diastolic pressure³, raised pulmonary capillary wedge pressure⁴ and enlarged echocardiographic left atrial dimension⁵. It has been found to be abnormal in acute pulmonary oedema⁶, acute myocardial infarction⁷, hypertension⁸, and ischemic heart disease⁹, and has become generally accepted as indicating left atrial pressure or volume overload¹⁰. Recently, and as yet inexplicably, it has also been found to be abnormal in cor pulmonale¹¹. While Morris found it never to be abnormal in 100 normal patients, Forfang¹² found 7% of apparently normal middle-aged men had an abnormal V_1 PTV. This paper examines the prevalence of the abnormal vector in healthy young airmen.

Patients and Methods

I examined the resting electrocardiograms (ECGs) of 3119 recruits to the Royal Air Force, of mean age 19 (SD 4) years, accepted as normal after routine medical examination which included chest X ray and clinical examination as well as ECG. The ECG's were recorded on Siemens Mingograf equipment (frequency response 0.05-500 herz) at a paper speed of 25 millimetres per second, and calibrated at one

millivolt to 10 millimetres deflection. For ease of expression the amplitude of the terminal P wave deflection was measured in microvolts rather than millimetres, and since the deflection was always negative, the negative sign was omitted. Thus a V_1 PTV greater than 3 microvolt seconds was regarded as abnormal. The actual measurements were taken horizontally and vertically from the junction of the P wave with the subsequent isoelectric line as described by Morris² (Fig. 1).

Results

V_1 PTV was greater than 3 microvolt seconds in 93 (3.2%) of 3119 cases, and the mean abnormal value was 4.6 (SD 1.4) microvolt seconds.

Discussion

It is generally accepted¹² that the resultant of left atrial depolarisation forces is at right angles to lead V_1 and that hypertrophy or dilatation causes a posterior rotation giving rise to the terminal

negative deflection. Josephson¹³ et al have suggested however that an interatrial conduction defect is the cause, though others have failed to find the inferred increased time of inscription of the vector¹⁴. The resistivity of intracardiac blood is also known to influence the inscription of the surface ECG¹⁵, and acute changes in hematocrit have been shown to alter the morphology of the P wave¹⁶. This latter may be relevant to the finding of an abnormal V₁PTV in cor pulmonale¹¹ and other disorders apparently confined to the right heart¹⁷. Thus while an abnormal V₁PTV is of value in the sequential assessment following myocardial infarction¹⁴, and is often indicative of left atrial enlargement, its presence in 3% of healthy young men, and in other unassociated conditions demands that such inferences be made with caution.

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