

Electrocardiogram of pure left ventricular hypertrophy and its differentiation from lateral ischaemia

Sir,

I was delighted to read Beach, Kenmure, and Short's paper¹ emphasising features of the final ventricular configuration (repolarisation) that distinguishes left ventricular hypertrophy from myocardial ischaemia. Credit, however, should have been given to the pioneers who first described them.

Pardee² first pointed out the configuration of the T waves which are characteristic of myocardial infarction. These T waves were subsequently called Pardee waves or coronary T waves. I was taught these findings during my junior year by Herrick who was still giving an occasional lecture at that time. Pardee quoted Herrick's famous case,³ the first clinical example of a coronary T wave. At Herrick's suggestion, Smith⁴⁻⁶ ligated coronary arteries in dogs and was able to demonstrate typical coronary T waves.

Barnes and Whitten⁷ were the first to describe in detail the T wave configuration in predominant ventricular hypertrophy, which they called strain. They acknowledged the influence of Willius, their colleague at the Mayo Clinic, and also Wilson's concept of the ventricular gradient. Wilson objected to the word strain because it is not an electrical term. Barnes re-emphasised these findings and the differentiation from those of ischaemia in his monograph on electrocardiographic patterns.⁸ The T wave findings in left ventricular hypertrophy appeared in Ashman and Hull's elementary⁹ and in Sodi-Pollares and Calder's advanced textbook on electrocardiography.¹⁰ Emphasis was placed upon the depression of the ST segment even when it was as little as 0.5 mm, the asymmetric T wave, and the low and flat T wave. Goldberger¹¹ noted the appearance of a roller-coaster effect with downward T waves terminating above the baseline.

The differentiation between the configuration of repolarisation caused by myocardial ischaemia from that of left ventricular hypertrophy is clearly described in the books by Katz¹² and by Sodi-Pollares and Calder.¹⁰

This distinction of course is not absolute because of the occasional simultaneous presence of both lesions and because of the superimposed effects of drugs

(digitalis) or electrolyte abnormalities; there may also be a problem if the electrocardiogram is taken soon after the ingestion of a high carbohydrate diet, after an injection of insulin, or immediately after physical activity.

Beach *et al.*¹ correctly state that QS complexes in the right precordial leads are not diagnostic of an infarction. They may occur, as they state, in isolated left ventricular hypertrophy and may also occur in infiltrative disorders of the myocardium (amyloidosis, tumours) frequently when the leaves of the diaphragm are depressed (emphysema), in right atrial enlargement (tricuspid regurgitation), and, rarely, in normal individuals when there is pronounced clockwise rotation of the electrical axis. If the electrodes are erroneously placed too high on the thoracic wall, which is a not uncommon error in women with large breasts, QS complexes may be seen.

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References

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This letter was shown to Drs Beach, Kenmure, and Short who reply as follows:

Sir,

We are grateful to Dr Soloff for reminding us of observations on the repolarisation pattern in left ventricular hypertrophy made by earlier workers in this field. We have reviewed most of the publications to which Dr Soloff refers. It is true that ST depression, T wave asymmetry, and a "roller-coaster" pattern are described. Nevertheless, the descriptions are con-

fused and conflicting, and we found no evidence of any attempt to quantify the degree of asymmetry in left ventricular hypertrophy to enable this to be used to discriminate between hypertrophy and ischaemia.

It is therefore not surprising that the idea that there might be definable differences between the pattern of repolarisation in left ventricular hypertrophy and that in coronary disease was never established. We do not know of a single textbook on electrocardiography, published in the past 20 years, which draws attention to these differences; indeed most state or imply that the patterns are indistinguishable.

Early workers were limited by the fact that they were not able to exclude the coexistence of coronary narrowing along with left ventricular hypertrophy except at necropsy. It is now possible to do this during life; so we have a secure basis for comparing the electrocardiography of "pure" left ventricular hypertrophy with that of coronary heart disease.

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