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

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SUMMARY In routine reporting of electrocardiograms, a frequent problem is presented by the presence of repolarisation abnormalities (ST depression and/or T wave inversion) in the lateral leads without the accepted QRS voltage criterion of left ventricular hypertrophy.

To help resolve this problem, the electrocardiograms of 41 patients with severe aortic stenosis who had no evidence of coronary disease were compared with the electrocardiograms of 20 patients with lateral myocardial infarction who had no clinical evidence of left ventricular hypertrophy. Nine of the patients with aortic stenosis were found to show repolarisation abnormalities in the lateral leads without the standard voltage criterion of left ventricular hypertrophy. The repolarisation pattern of aortic stenosis could frequently be distinguished from that of coronary disease by the presence of one or more of the following five features: depression of the J point, asymmetry of the T wave with rapid return to the baseline, terminal positivity of the T wave ("over-shoot"), T inversion in V6 greater than 3 mm, and T inversion greater in V6 than in V4.

One of the most common problems of an electrocardiogram reporting service is knowing how to interpret repolarisation abnormalities (ST depression and/or T wave inversion) confined to the lateral leads.[1] If the QRS voltage is abnormally great, the tracing is likely to be reported as indicating left ventricular hypertrophy; otherwise it will probably be reported as suggestive of ischaemia.

We set out to answer two questions. First, may repolarisation abnormalities in the lateral leads without abnormal RS voltage be a result of left ventricular hypertrophy? Secondly, can such abnormalities be differentiated from those seen in coronary arterial disease?

Subjects and methods

In essence, our method was to take a group of patients with severe aortic stenosis without significant coronary arterial disease and compare their electrocardiograms with those of a group of patients with lateral myocardial infarction without clinical evidence of left ventricular hypertrophy.

LEFT VENTRICULAR HYPERTROPHY GROUP

This group consisted of 41 adult patients with aortic stenosis on whom haemodynamic studies (including in 39 cases coronary arteriography) were performed as part of an assessment for aortic valve replacement.

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ST depression or T inversion in the lateral leads and excluded. None of these patients gave a history of previous myocardial infarction and none was receiving digoxin or any similar drug. Their ages ranged from 26 to 69 years, with a mean of 54 years. Twenty-one were men and 20 were women. The aortic valve gradients ranged from 45 to 170 mmHg, with a mean of 90 mmHg. Two patients, one woman aged 26 and one man aged 35, were not submitted to coronary arteriography. Of the 39 patients in whom coronary arteriography was performed, the arteriogram was entirely normal in 26; and in the remaining 13 there was less than 50% narrowing of a single coronary artery branch. The aetiology of most of the cases was presumed to be calcific degeneration of a congenitally deformed valve.

All 41 patients had several electrocardiograms recorded at intervals of months or years before operation, each of which had been standardised at 1 mV=10 mm. In 11 of the patients two or more of their electrocardiograms showed significantly different repolarisation patterns. Thus, these 41 patients yielded 58 electrocardiograms for analysis, with patterns ranging from normality, through minor ST depression and T wave flattening, to profound T wave inversion.

LATERAL MYOCARDIAL INFARCTION GROUP

This group consisted of 20 patients (17 men, three women) with proven myocardial infarction showing

Other causes of repolarisation abnormality were frequently in other leads also. Patients with clinical or radiological evidence of left ventricular hypertrophy, hypertension, or valvular disease were excluded. None was on digoxin or any similar drug. Their ages ranged from 27 to 65 years, with a mean of 53 years. Each of these patients had several electrocardiograms recorded after one or more attacks of myocardial infarction. From the many electrocardiograms available, we assembled a selection showing a spectrum of repolarisation abnormalities corresponding to that in the left ventricular hypertrophy group. We excluded all tracings showing ST elevation on the grounds that these could not cause any confusion with the pattern of left ventricular hypertrophy. We were thus left with 37 electrocardiograms with patterns ranging from the slightest degree of ST depression to deep T wave inversion.

Results

ELECTROCARDIOGRAPHIC PATTERNS IN AORTIC STENOSIS

In 26 of the patients with aortic stenosis, each of the available electrocardiograms showed the accepted voltage criterion of left ventricular hypertrophy[2 3]; that is S in V1 plus R in V5 or V6 (whichever was the greater) exceeding 35 mm (3·5 mV). In all but two of these patients, the electrocardiogram also showed repolarisation abnormalities (Table). Nine patients had one or more electrocardiograms showing repolarisation abnormalities in the lateral leads without the voltage criterion of left ventricular hypertrophy; seven had T inversion and two had T wave flattening (Fig. 1 and 2). In three patients, the initial electrocardiogram showed a pattern of complete bundle-branch block (two right, one left), and three patients had electrocardiograms which were consistently within normal limits. There was no precise correlation between the electrocardiographic pattern and either the age of the patient or the gradient across the aortic valve.

Table *Electrocardiographic pattern in 41 patients with severe aortic stenosis at the time of cardiac catheterisation*

Voltage criterion of left ventricular hypertrophy[2 3] *with* repolarisation abnormality	24
Voltage criterion of left ventricular hypertrophy *without* repolarisation abnormality	2
Repolarisation abnormality without voltage criterion of left ventricular hypertrophy	9
Bundle-branch block	3
Within normal limits	3

In 10 patients significant electrocardiographic deterioration took place during the period of observation. Two progressed from minor T flattening to deep T inversion: one over a period of three years, the other over a period of three months. Another patient progressed from a normal pattern to complete right bundle-branch block in the course of seven and a half years. By contrast, three patients showed significant improvement in the electrocardiogram while awaiting surgical treatment.

COMPARISON BETWEEN ELECTROCARDIOGRAMS OF AORTIC STENOSIS AND LATERAL MYOCARDIAL INFARCTION

Voltage

The mean R voltage in V6 was greater in the group of patients with aortic stenosis than in the group with infarction, though there was considerable overlap in

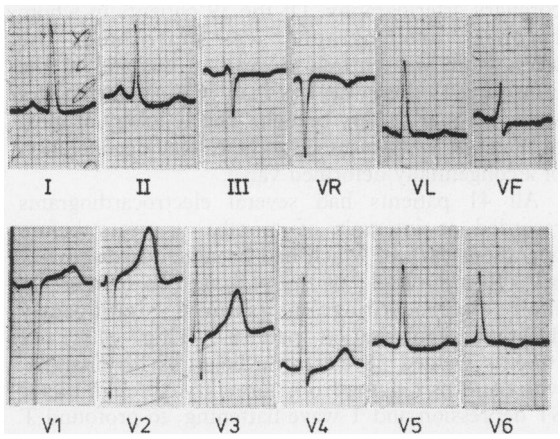

Fig. 1 *Repolarisation abnormality without abnormal RS voltage in a patient with severe aortic stenosis. Obese woman aged 38. Gradient across aortic valve 80 mmHg. Coronary arteriogram normal. This electrocardiogram and those illustrated in Fig. 2 and 7 were all standardised at 10 mm=1 mV.*

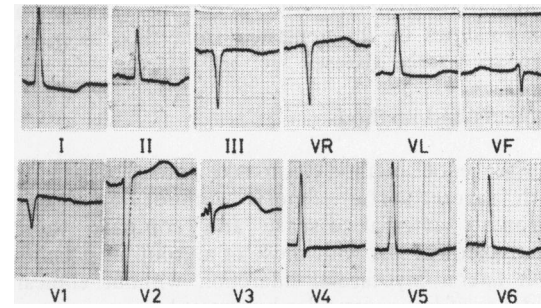

Fig. 2 *Repolarisation abnormality without abnormal RS voltage in a patient with severe aortic stenosis. Obese woman aged 68. Gradient across aortic valve 110 mmHg. Coronary arteriography showed no abnormality, though visualisation of the left coronary artery was technically substandard. There was no clinical or electrocardiographic evidence of myocardial infarction either before this tracing was recorded or during the eight years during which the patient was observed after operation.*

individual cases (Fig. 3). The same was true to a lesser extent of the sum of S in V1 plus R in V5 or V6 (whichever was the greater) (Fig. 4).

T wave inversion

T inversion in V6 was seen in 31 of the 38 patients with aortic stenosis (excluding those with bundle-branch block) and in 17 of the 20 patients with lateral infarction. Deep inversion was commoner in aortic stenosis than in infarction. Twenty-one of the patients with aortic stenosis had inversion to a depth of 3 mm or more, compared with only four of the patients with infarction. Five of the patients with aortic stenosis had inversion to a depth of 5 mm or more, compared with only one of the patients with infarction. In aortic stenosis, T inversion was greater in V6 than in V4 in 28 of the 31 cases. In infarction, on the other hand, T inversion was greater in V6 than in V4 in only eight of the 17 patients.

There was also a difference between the two groups in the form of the inverted T wave. In aortic stenosis, the T wave was usually less symmetrical than in infarction. The asymmetry of the T wave could be expressed by the ratio between the angle of descent and the angle of ascent (see Fig. 5). In aortic stenosis, the ratio ranged from 1·3 to 3·0, with a mean of 2·0, whereas in infarction the ratio ranged from 1·0 to 1·7, with a mean of 1·3 (Fig. 6). A ratio of over 1·75 was seen in 19 of the 31 cases of aortic stenosis with T inversion of 1 mm or more, but in none of the 13 cases of infarction. Conversely, a ratio of less than 1·25 was seen in seven of the 13 cases of infarction, but in none of the cases of aortic stenosis.

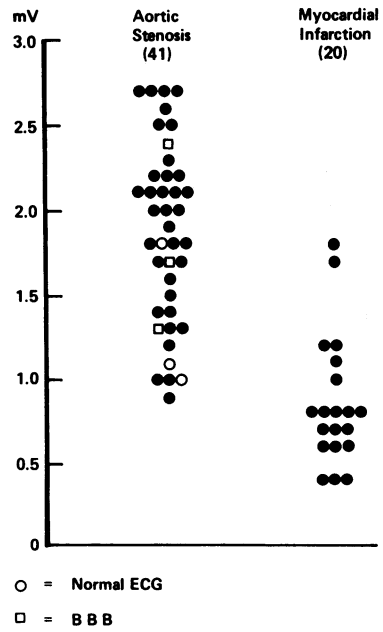

Fig. 3 *Comparison between R wave voltage in V6 in patients with severe aortic stenosis and those with infarction.*

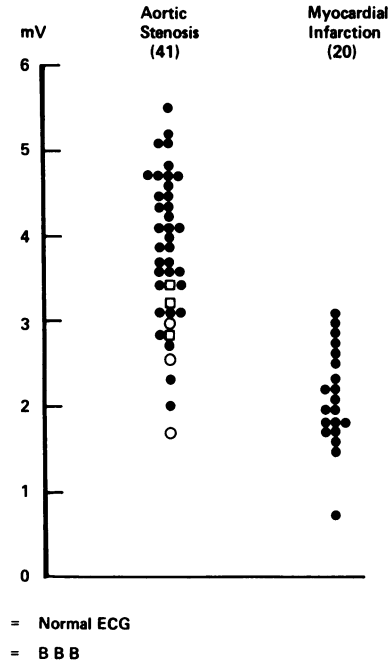

Fig. 4 *Comparison between the sum of S wave voltage in V1 and R wave voltage in V5 or 6 (whichever was the greater) in patients with severe aortic stenosis and those with infarction.*

Terminal positivity of T wave

In a considerable proportion of cases of aortic stenosis, the terminal portion of the T wave was positive when most of the T wave was inverted or flat. When the T wave was deeply invered, this terminal positivity had the appearance of an "overshoot" (Fig. 5). Terminal positivity of the T wave was seen in 22

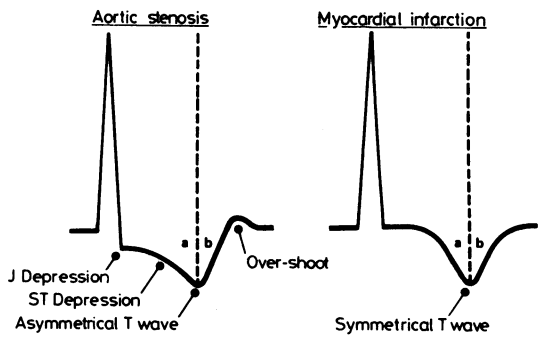

Fig. 5 *Diagram showing characteristic features of inverted T wave in aortic stenosis compared with those in infarction.*

out of the 52 electrocardiograms of patients with aortic stenosis (excluding those with a normal electrocardiogram or bundle-branch block) (Fig. 7) but in only three out of the 37 electrocardiograms of patients with infarction.

J depression

Depression of the ST junction (J point) of 1 mm or more (Fig. 5) was seen in 23 out of the 52 electrocardiograms of patients with aortic stenosis (Fig. 7) but in only four out of the 37 electrocardiograms of patients with infarction.

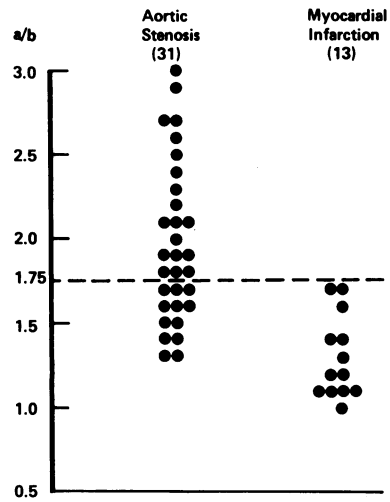

Fig. 6 *Comparison between the asymmetry ratio of the inverted T wave in patients with aortic stenosis and those with infarction. The asymmetry ratio is the ratio between the angle of descent and the angle of ascent of the T wave (see Fig. 5). The measurements were limited to patients whose electrocardiograms showed T inversion of 1 mm or more. The ratios are the mean of two readings in each case.*

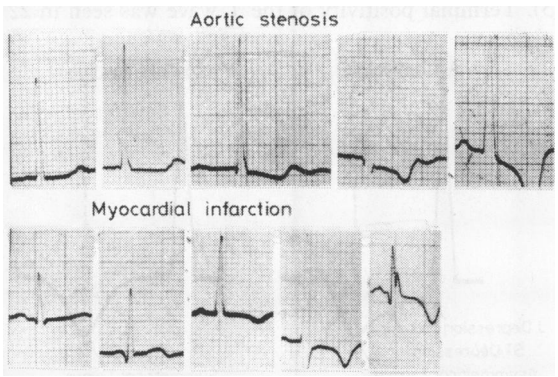

Fig. 7 *The spectrum of distinctive repolarisation abnormalities in V6 seen in cases of aortic stenosis and myocardial infarction.*

If one considers only electrocardiograms showing flat T segments or inversion of less than 1 mm, the finding of J depression or terminal positivity of the T wave or both was strongly associated with aortic stenosis. These patterns were seen in 17 out of 21 cases of aortic stenosis but in only four out of 24 cases of infarction.

Q wave

None of the patients with aortic stenosis showed abnormal Q waves, as defined in the revised Minnesota Code,[4] in V6; though seven showed QS waves in leads V1 to V3. In the infarction group, two patients showed abnormal Q waves in leads V4 and V6, and seven in V4 but not in V6. Fourteen patients with infarction showed abnormal Q waves in anteroseptal or inferior leads. Absence of a q wave in V6, without widening of the QRS complex, was noted in 14 electrocardiograms (in 11 patients) with aortic stenosis and in nine electrocardiograms (in five patients) with infarction.

Axis

Apart from the three patients with complete bundle-branch block, none of the patients with aortic stenosis showed an abnormal mean frontal axis. Four of the patients with myocardial infarction showed an axis within the range -30 to $-60°$.

Discussion

T wave inversion or flattening in the lateral leads of the electrocardiogram without abnormal R wave voltage is one of the commonest abnormalities encountered in electrocardiogram reporting.[1] Such a pattern is often regarded as suggesting lateral myocardial infarction or ischaemia, but we have long suspected that left ventricular hypertrophy might present in this way. The present study was undertaken in an attempt to establish this point and to discover whether the repolarisation abnormality in left ventricular hypertrophy could be distinguished from that in ischaemia and infarction.

Some may consider such an exercise to be merely academic, arguing that in either case the repolarisation abnormality is "ischaemic" in nature. But this is to blur an important distinction. Admittedly, ischaemic fibrosis is frequently found in the left ventricle in cases of severe aortic stenosis in the absence of acute coronary occlusion.[4] Nevertheless, the abnormal electrocardiogram, and particularly the abnormal vectorcardiogram, of aortic stenosis may revert toward normal very quickly after aortic valve replacement,[5] suggesting some more rapidly reversible process than fibrosis. We therefore consider it useful to maintain a distinction between

the repolarisation abnormalities in left ventricular hypertrophy and those in coronary disease.

To obtain a group of patients with left ventricular hypertrophy without significant coronary arterial disease, we chose cases of aortic stenosis which had been investigated with a view to valve surgery and had been shown to have normal or near normal coronary arteriograms. Admittedly, the finding of a normal coronary arteriogram does not exclude the possibility of a previous occlusion; nevertheless none of our patients had a history or previous electrocardiogram suggesting such an event.

The current basic electrocardiographic criterion of left ventricular hypertrophy is a voltage measurement. This is the criterion originally proposed by Sokolow and Lyon[2] and accepted by the latest WHO expert committee on arterial hypertension[3]; namely the sum of the S voltage in V1 and the R voltage in V5 or V6 (whichever is the greater) must exceed 3·5 mV (35 mm). The limitations of this criterion, however, are not always appreciated. There is a wide range of RS voltage in healthy subjects[2 6] and hence considerable overlap between the voltage in left ventricular hypertrophy and that in normal individuals. If the dividing line is set low enough to include all patients with left ventricular hypertrophy it inevitably includes a considerable proportion of those who do not have left ventricular hypertrophy. On the other hand, if the dividing line is set high enough to exclude all normal individuals, it excludes a considerable proportion of patients with left ventricular hypertrophy. Sokolow and Lyon chose the latter course. They recommended a dividing line which excluded all the normal individuals in their series, and in so doing they excluded two-thirds of their patients with left ventricular hypertrophy. Thus, the Sokolow and Lyon criterion is highly specific but relatively insensitive. It would not be surprising, therefore, if some individuals with a naturally low RS voltage in the praecordial leads should develop secondary repolarisation changes in response to left ventricular hypertrophy before the voltage exceeded that of normal individuals with a naturally high voltage in these leads. This is, in fact, what our study has shown. Over one-fifth of our patients with severe aortic stenosis (without significant coronary disease) showed repolarisation abnormalities in the lateral leads without the accepted voltage criterion of left ventricular hypertrophy. In some, admittedly, there were hints of left ventricular hypertrophy in the limb leads or in the depth of the S wave in V2.

This finding may have wide implications in routine electrocardiogram reporting. For if the electrocardiogram in hypertension evolves in a manner similar to that in aortic stenosis, the finding of repolarisation abnormalities in the lateral leads without abnormal RS voltage might frequently indicate left ventricular hypertrophy rather than ischaemia—since hypertension is such a common condition in the general population.

This, of course, raises the question as to whether the repolarisation abnormality of left ventricular hypertrophy can be distinguished from that of coronary disease without reference to voltage criteria. We found five features which were more common in hypertrophy than in ischaemia: (1) depression of the J point, (2) asymmetrical inversion of the T wave with rapid return to the baseline, (3) terminal positivity of the T wave ("over-shoot"), (4) T inversion to a depth of 3 mm or more in V6, and (5) deeper inversion of the T wave in V6 than in V4. The presence of J depression and/or terminal positivity of the T wave was particularly frequent in the electrocardiograms of patients with aortic stenosis without frank T inversion in V6. Conversely, there are patterns that indicate coronary disease rather than left ventricular hypertrophy; for example ST elevation and abnormal Q waves in the anterolateral or inferior leads. Abnormal Q waves in the anteroseptal leads should not be taken as being indicative of myocardial infarction, since an identical pattern may be seen in aortic stenosis.[7 8]

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