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Left ventricular hypertrophy : a study of the accuracy of electrocardiographic criteria compared with cases proven at autopsy

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LEFT VENTRICULAR HYPERTROPHY

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INTRODUCTION

The electrocardiograph is valuable in three general abnormalities of the heart: namely, arrhythmias, myocardial infarctions, and cardiac hypertrophy. It is within the realm of the latter to which this paper pertains.

For years empiricism has reigned in the electrocardiographic diagnosis of left ventricular hypertrophy. Rigid criteria have been set down for the diagnosis. Recently Scott et al. (1) correlated the EKG criteria of numerous groups--Gubner and Ungerleider, Katz, Schach, Rosenman and Katz, Goldberger, Goulder and Kissane, Roth, Myers and Klein, Wilson and associates, and Sokolow and Lyon--with autopsied cases proven to have left ventricular hypertrophy. In their study, the criteria of Sokolow and Lyon proved to be the most accurate. In recent years spatial vectorcardiography has been introduced, for which Grant has been the main crusader. This method provides a more liberal means for cardiac evaluation and allows for variances of the EKG, as it takes into consideration age, body build, and associated diseases. It is the purpose of this paper to compare these two approaches by correlating the criteria of Sokolow and Lyon, and Angle with cases of proven left ventricular hypertrophy.

CAUSES OF HYPERTROPHY

Persiste t hypertension or valvular insufficiency causes changes in the heart. These changes are a result of increasing diastolic filling and systolic residue. If this assumes significant proportions (that is, beyond the peak of Starling's curve), strain, stretching or dilatation occurs. This results in greater initial length in fibers which gives a persistence of exaggerated contraction. This eventually leads to hypertrophy of the musculature of the heart. Not only hypertension and valvular insufficiency lead to hypertrophy. Impaired nutrition causes injury to cardiac muscle. This in turn causes dilatation and consequent hypertrophy. Thus, decreased blood supply to cardiac muscle or toxic or inflammatory states may produce hypertrophy. In coronary insufficiency, hypertrophy occurs between periods of relative anoxia, according to Conolly and Littmann (2).

It has not been proved, however, that hypertrophy increases the strength of the contraction, although it does increase its effectiveness. It has been shown that in strenuous exercise the cardiac work per unit of time in a human with a normal heart may exceed that required of the heart in a patient with cardiac hypertrophy. Thus, it appears that the normal heart is capable of as great strength

of contraction as is produced ordinarily by the hypertrophied heart. Grant offers the suggestion that perhaps hypertrophy makes possible the maintenance of increased work over a long period of time. Perhaps hypertrophy supplies some metabolic or energy substration to accommodate to the increased work. He concludes that hypertrophy may not be a source of increased work but a consequence (3).

VENTRICULAR EXCITATION

Normally left ventricular excitation extends first down the endocardial surface of the anterior and diaphragmatic walls of the left ventricle toward the base on the superior and posterior endocardial surfaces, usually terminating at the remotest posterobasal region of the free wall. The epicardial excitation tends to follow this same sequence, but more slowly. The terminal QRS vector forces, therefore, as they are the resultant of vectors from the last epicardial regions to be depolarized, tend to be directed posteriorly and somewhat leftward. This is the normal sequence of events and explains the direction of the mean QRS vector in the normal subject (4).

It has long been known that hypertrophy produces changes on the electrocardiogram. These deviations from the normal are a result of changes in the spread of the excitation wave through the ventricular wall.

What accounts for the various deviations seen in left ventricular hypertrophy? Fenichel (5), in 1932, suggested that the deviation of the electrical axis in ventricular preponderance is probably an effect of the greater potential differences generated by the wall of the more hypertrophied ventricle. Grant (4) offers a more detailed explanation by stating that hypertrophy delays the progression of the excitation across the myocardium. This results in an extension of the period of epicardial excitation and more clearly separates in time the vectors generated from the last region of the left ventricle from those generated from the more intermediate regions. Thus, the terminal vectors are more exclusively contributed from the superior and posterior regions of the left ventricle in the hypertrophied than in the normal heart, and hence have a more superior and posterior direction than normally. Also because of the increase in epicardial surface the magnitude of these vectors is greater. Another factor leading to leftward deviation of the mean QRS vector is the bowing superiorly and posteriorly of the free wall of the left ventricle seen in some cases.

Wilson, MacLeod, and Barker (6) add that because of the fact that the wave of excitation travels much faster along the endocardial surface than it does across the wall of the left ventricle, hypertrophy causes a greater

difference in rate of movement and this causes the wave of excitation advancing across the myocardial wall to become more and more oblique to the surface rather than perpendicular as in the normal subject. This causes the direction of the mean QRS vectors to be more leftward and posterior with left ventricular hypertrophy than in the normal heart.

SIMILAR STUDIES

In 1930, Wilson and Herrmann (7) correlated the QRS interval with ventricular weight and the thickness of the wall of the left ventricle.

Levine and Phillips (8), in 1951, using EKG criteria of Wilson and his co-workers, correctly diagnosed 25 of 37 cases of left ventricular hypertrophy proven at autopsy.

Noth, Myers, and Klein (9), in 1947, analyzed the findings in the precordial leads in 84 cases of pathologically proved cases of left ventricular hypertrophy. In addition, a control group of 52 cases in which the hearts were normal at autopsy and 50 young male hearts that were normal by clinical and roentgen examination were studied. They report that in 98 per cent of the normals, the time interval from the onset of the QRS to the peak of the R was less than .05 second. In 93.1 per cent of the normals,

the time interval from the onset of the R wave to its peak was less than .04 second. Of the 84 cases studied with left ventricular hypertrophy proved at autopsy, 40.5 per cent showed an abnormal Q-R and/or R duration. They also found that differentiation between the pattern of left ventricular hypertrophy and of normals could not be made from the amplitude of the R waves in V5 and V6. In 1914, Lewis (10) and in 1922, Herrmann and Wilson (11) correlated EKG findings on the standard limb leads with the weight of the left ventricle.

Grant (12) meticulously studied heart position at autopsy and related position to electrocardiographic findings. He showed that QRS axis deviations commonly seen with ventricular hypertrophy are not due to anatomic rotation of the heart. The lie of the left and right ventricles in the body was remarkably similar in all hearts, normal and hypertrophic. In the presence of left ventricular hypertrophy, the electrical axis was often markedly rotated leftward while the anatomic position of the left ventricle in these cases was essentially the same as in the normal heart.

Lipsett and Zinn (13) analyzed the precordial and augmented limb leads of 73 electrocardiographs from patients with autopsy evidence of combined ventricular hypertrophy.

METHODS AND MATERIALS

The autopsy protocols of the Pathology Department of the University of Nebraska College of Medicine and The Bishop Clarkson Memorial Hospital in Omaha were examined, and cases with a left ventricular thickness of 13 mm. or more and a heart weight of more than expected normal for the length of the body (14) were collected. Any case from within the University of Nebraska which had a right ventricular thickness of more than 5 mm. was not included.

Because of different techniques in measurement, any case from Bishop Clarkson Hospital which had a right ventricular thickness of more than 8 mm. was not included. Cases with left bundle branch block, right bundle branch block or those with pathologic evidence of myocardial infarction were not included. The last electrocardiogram taken before death was used unless the last record was technically unsatisfactory.

The amount of left ventricular hypertrophy was graded, using heart weight as basis for classification. Hypertrophy was classified one to four plus according to heart weight above the upper limits of normal employing Zeek's (14) criteria. The electrocardiographs were read and results classified from 0 to 4 plus according to the number of criteria present satisfying the diagnosis of left ventricular hypertrophy. Two sets of diagnostic

criteria were used, namely, Sokolow and Lyon, and Angle.

Sokolow and Lyon have formulated the following criteria for the diagnosis of left ventricular hypertrophy from the unipolar limb leads: (1) RS-T segment depressed more than 0.5 mm. in aVL or aVF; (2) flat, diphasic or inverted T waves, with an R wave of 6 mm. or more in aVL or aVF and item 1; (3) voltage of R wave in aVL greater than 11 mm. or in aVF greater than 20 mm.; (4) upright T wave in aVR. Also used was the following criteria of Sokolow and Lyon for diagnosis of left ventricular hypertrophy in the precordial leads: (1) the RS-T segments are depressed and the T waves low or inverted in V5 or V6; (2) R waves in V5 or V6 exceed 26 mm.; (3) the onset of the intrinsicoid deflection in V5 or V6 exceeds 0.05 seconds; (4) the R to T ratio in V5 or V6 is 10 or greater; (5) the R to S ratio in V1 is greater than 100; (6) the sum of the R wave in V5 or V6 and the S wave in V1 exceeds 35 mm.

Angle uses the following criteria for diagnosis of left ventricular hypertrophy: (1) high voltage of R for age and body build; (2) left or posterior deviation of QRS axis for average normal for age and body build; (3) widened QRS for age; (4) delayed intrinsicoid deflection; (5) qR in lead showing tallest R; (6) ST-T changes in leads with R.

Because of the nature of the criteria of Sokolow

and Lyon, these did not lend themselves well to tabulation and critical evaluation; thus, they were not classified according to hypertrophy nor tabulated. Rigidity of the criteria did not allow critical evaluation in cases with emphysema, obesity, anasarca, etc.

Evaluation of hypertrophy of the left ventricle based on Angle's criteria was accomplished in the following manner: Varying degrees of emphasis were placed on different criteria. Also of an individual criteria, the amount of abnormality shown (incorporating in the evaluation changes produced by accompanying conditions as body build, non-related diseases, etc.) was evaluated and upon these results, the final evaluation of the EKG, as to the amount of hypertrophy, was based. Most emphasis was placed on voltage and secondly on posterior axis deviation succeeded by the following in decreasing amount of emphasis: (1) ST-T depression, (2) left axis deviation, (3) widening of QRS complex, (4) delayed intrinsicoid deflection, and (5) qR in leads with tallest R waves.

RESULTS

As mentioned, the amount of hypertrophy was not evaluated on basis of Sokolow and Lyon criteria. Evaluation based on Angle's criteria is tabulated in Table I. There is remarkably good correlation between EKG

TABLE I

COMPARISON OF ANGLE'S EVALUATION WITH POST MORTEM EVALUATION

Case Number	Angle	Post mortem
1	I	II
2	I	I
3	II	II
4	II	III
5	I	I
6	I	II
7	II	II
8	II	II
9	I	I
10	I	I
11	III	III
12	I - II	II
13	IIII	II
14	I	II
15	I	III
16	I	III
17	III	II
18	II	II
19	I	III
20	I	III
21	II - III	III
22	I	III

evaluation and post mortem evaluation in the cases from the University Hospital. Discrepancies are noted in the cases taken from the Bishop Clarkson Memorial Hospital (cases 15-22). These may be based on different technique used by prosectors, but may be result of the different type of patients seen at private hospital as compared to the charity hospital. Hearts from the Clarkson Hospital tended to be heavier in general than ones from the University Hospital, which may be a result of the nature and nutritional status of patients seen at Clarkson. This is mere speculation, however, and as stated this may represent only differences in individual prosectors and their techniques. As indicated previously, the measurements for the right ventricular thickness were in general larger in cases from Clarkson, again indicating probable differences in technique at the post mortem examination.

Causes for discrepancies may be explained on basis of incipient variances in body build or unrelated accompanying diseases as emphysema, hiatus hernia, etc.

In all cases was one or more of the criteria of Angle satisfied. In the two cases listed as $\frac{1}{2}$ the changes were minimal with exception of posterior axis deviation, thus qualifying the evaluation.

In only one case of hypertrophy were the EKG findings normal as appraised by criteria of Sokolow and Lyon.

This case showed only posterior axis deviation, which did not show changes in Sokolow's criteria. The case in point is Number 22 which, by post mortem examination, showed a heart weight of 520 grams and was graded as three plus hypertrophy. This was rated ℓ according to Angle's criteria. In reviewing the chart this patient showed, besides hypertension and diabetes, findings of marked pulmonary fibrosis and pulmonary arteriosclerosis. The case was taken from Clarkson's files and showed right ventricular thickness of 5-7 mm. and left ventricular thickness of 20-25 mm.

ST-T changes were found most frequently. In all but one case were there ST-T abnormalities. In one case the ST-T changes were minimal. Voltage changes, however, as well as axis deviation especially posterior axis deviation, were most helpful in evaluation of the degree of hypertrophy, and to these factors can be accredited the satisfactory results in this evaluation. Sokolow's rigid criteria would not allow proper evaluation of the voltage of the R wave.

REMARKS

Only the positivity of the two authors' criteria in cases of proven cases of left ventricular hypertrophy were tested in this paper. False positives, obviously,

could not be tested by method used.

SUMMARY AND CONCLUSIONS

1. The criteria of Sokolow and Lyon and Angle for the diagnosis of left ventricular hypertrophy have been analyzed in 22 cases of left ventricular hypertrophy proven at post mortem examination.

2. The criteria of Sokolow and Lyon did not allow critical evaluation and tabulation of the degree of hypertrophy, whereas according to the criteria of Angle the electrocardiographs were critically analyzed and evaluated as to degree of hypertrophy.

3. All cases based on Angle's criteria showed evidence of hypertrophy on EKG.

4. Evaluation of degree of hypertrophy on basis of Angle's criteria showed remarkably good correlation.

5. Only one case based on criteria of Sokolow and Lyon showed no changes diagnostic of left ventricular hypertrophy.

6. ST-T changes were found most frequently.

7. Voltage changes and axis deviation, especially posterior axis deviation, were found to be most helpful in evaluation of degree of hypertrophy, these being found in most cases of marked hypertrophy.

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