Reliability of Electrocardiographic Diagnosis of Left Ventricular Hypertrophy

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This is an electrocardiographic-pathologic study in which the presence and degree of left ventricular hypertrophy found at necropsy were correlated with electrocardiographic abnormalities suggesting the diagnosis of left ventricular hypertrophy. The nonspecificity of some electrocardiographic findings is emphasized by the occasional erroneous diagnosis of left ventricular hypertrophy.

The diagnosis of ventricular hypertrophy ranks high in importance among the various clinical applications of electrocardiography, because electrocardiographic alterations may exist without other clinical signs of hypertrophy. Such early diagnosis of ventricular hypertrophy may be of great practical import, since the application of many newer medical and surgical methods of therapy often depends on the presence or absence of ventricular hypertrophy.

Although a number of criteria for the electrocardiographic diagnosis of left ventricular hypertrophy have been recommended by various investigators,1-8 a recent survey by questionnaire9 revealed some hesitation on the part of several experts in accepting a strictly defined set of diagnostic criteria.

Many excellent studies of clinical and electrocardiographic correlation of left ventricular hypertrophy have been made. However, relatively little information is available concerning autopsy verification of such studies. In a recent report, Scott et al.10 examined a series of electrocardiograms from patients in whom autopsies showed evidence of left ventricular hypertrophy, in order to assess the reliability of electrocardiographic criteria suggested by various workers. These authors, however, investigated only the positive side of the correlation and made no attempt to explore the possibility of incorrect, false positive electrocardiographic diagnoses. The current study was undertaken to provide additional electrocardiographic-anatomic correlation and to investigate the negative as well as the positive aspects of the problem.

Material and Methods

The study was based upon routine material from the Electrocardiographic department of the Veterans Administration Hospital, San Francisco, and the routine autopsy material from the files of its Department of Pathology, during the period of 1948 to 1955. All electrocardiographic tracings were taken with the Cambridge string-galvanometer electrocardiograph with photographic records. Conventional speed (25 mm. per second) and sensitivity (1 mv. per cm.) were used throughout and all measurements were corrected for minor deviations in string sensitivity. Twelve-lead electrocardiograms were taken routinely, including 3 standard extremity leads, 3 augmented unipolar extremity leads, and 6 precordial unipolar leads. Autopsies were performed with a uniform technic, the gross examination of the heart being carried out according to Saphir’s recommendations. The hearts were weighed after postmortem clots and the great vessels had been removed.

The study was organized as follows: (1) All patients on whom necropsy was performed and who had had an electrocardiogram taken during life within the period of 1948 to 1955 constituted the basic group of 550 cases. (2) The electrocardiographic criteria for left ventricular hypertrophy were collected from the literature and were grouped into 3 classes (table 1). (3) The 550 electrocardiographic tracings were reviewed by 2 observers, who selected records in which criteria for left ventricular hypertrophy were met. These observers had had no knowledge of the necropsy...
findings and disregarded the original interpretations of the electrocardiograms. A single criterion in the classes of increased voltage and prolonged ventricular activation time (table 1) was acceptable as a basis for the electrocardiographic diagnosis of left ventricular hypertrophy. However, abnormalities of the ST-T part of the ventricular complex were not considered specific enough to constitute the sole basis for a diagnosis of left ventricular hypertrophy and were only accepted in combination with 1 or more criteria from the other classes. (4) Of all records in which criteria for left ventricular hypertrophy were met, a smaller group was selected wherein the diagnosis was thought to be uninfluenced by any other factor. This final group of 108 cases was obtained by eliminating all records in which the following conditions existed: (a) those in which an unduly long time had elapsed between the electrocardiographic examination and death; (b) those wherein a new condition may have developed after the last electrocardiographic examination that could have affected the postmortem diagnosis of left ventricular hypertrophy; (c) records showing electrocardiographic evidence of myocardial infarction, recent or old; (d) records with tachycardia (over 110 per minute) or complete heart block; (e) records of patients with obvious electrolyte disturbance; (f) records showing possible drug effect: an arbitrary 2-week limit had been

**Table 1. Electrocardiographic Criteria for Left Ventricular Hypertrophy**

<table>
<thead>
<tr>
<th>Author</th>
<th>Voltage</th>
<th>Ventricular activation time</th>
<th>ST-T</th>
<th>Others</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gubner</td>
<td>$R_1+S_5$ over 25 mm.</td>
<td></td>
<td>$ST_T$ depr. $T_1$ lowered</td>
<td></td>
</tr>
<tr>
<td>Katz</td>
<td>$aV_R$ neg. over 14 mm. $aV_L : R$ over 12 mm. $aV_F : R$ over 19 mm.*</td>
<td></td>
<td>$S-T_1$ depr. $T_1$ low $T_{V5}$ low, inv.</td>
<td>Left axis deviation with $S_2$</td>
</tr>
<tr>
<td>Schackt</td>
<td>$R_{aVL}$ over 13 mm. $R_{aVF}$ over 20 mm.*</td>
<td></td>
<td>&quot;Strain&quot; $ST$, $T$ in $aV_L$ or $aV_F$*</td>
<td>Long QT in $aV_L$ or $aV_F$*</td>
</tr>
<tr>
<td>Goldberger</td>
<td>$R_{aVL}$ over 11 mm.</td>
<td></td>
<td>[T : R \text{ in } aV_L \text{ less than } 10%]</td>
<td></td>
</tr>
<tr>
<td>Noth</td>
<td>$R_{V1}$ 1 mm. or less $S_{V1}$ 24 mm. or more $R_{V5}$ 33 mm. or more $R_{V6}$ 26 mm. or more</td>
<td>over 0.04 sec. for $R$ waves, over 0.05 sec. for $qR$ waves</td>
<td>$T$ inverted in $V_5$ or $V_6$</td>
<td>Transitional zone to the left</td>
</tr>
<tr>
<td>Wilson</td>
<td>$R_{V1}$ 11 mm. $R_{V5}$ 19 mm.* $R_{V6}$ 26 mm. $S_{V1}$ $R_{V5}$ or $S_{V5}$ + $R_{V6}$ 35 mm.</td>
<td>0.05 sec. or more in $V_5$ or $V_6$ QRS 10 to 11 sec.</td>
<td>$T$ upr. in $aV_R$ $S-T$ depressed in $aV_L$, $aV_F^<em>$ $V_5$ or $V_6$; $T$ low or inv. $aV_L$, $aV_F^</em> V_5$, $V_6$; $R : T$ ratio 10 to 1 or more, $V_5$ or $V_6$</td>
<td>$R/S$ in $V_5$ to $R/S$ in $V_1$ over 100</td>
</tr>
<tr>
<td>Sokolow</td>
<td>$R_{aVL}$ over 11 mm. $R_{aVF}$ over 19 mm.* $R_{V5}$ over 26 mm. $R_{V6}$ over 26 mm. $S_{V1} + R_{V5}$, $S_{V5}$ over 35 mm.</td>
<td>0.05 sec. or more in $V_5$ or $V_6$</td>
<td>$R/S$ in $V_5$ to $R/S$ in $V_1$ over 100</td>
<td></td>
</tr>
</tbody>
</table>

*In the presence of vertical position.
set between the last dose of a cardiac drug (digitalis, quinidine, or procaine amide) and an acceptable electrocardiogram. (5) The autopsy protocols of these 108 patients were carefully studied and the following data recorded: the total heart weight; thickness of each ventricular wall; the examiner’s notation regarding chamber enlargement; gross or microscopic evidence of infarction or scarring; evidence of other myocardial or pericardial disease; the cause of death and anatomic diagnoses; the patient’s weight and height; any other factor that may have had some bearing on the problem.

In considering the anatomic criteria for the presence or absence of left ventricular hypertrophy, it was decided to rely exclusively upon the total cardiac weight. The tables of Zeek were used to calculate the upper limits of normal heart weight in relation to total body length. It was decided that hearts falling into normal and abnormal weight groups should not be separated sharply, but divided by a “borderline” zone that was arbitrarily established to include all hearts falling within plus and minus 25 Gm. of the upper limit of normal for a given body length. Thus, normal hearts were those weighing less than 25 Gm. below Zeek’s upper limit of normal. Abnormally heavy hearts were those heavier than 25 Gm. above that limit.

Abnormally heavy hearts were considered as showing left ventricular hypertrophy. These were divided into 3 groups showing degrees of cardiac hypertrophy: lesser degree of hypertrophy, weighing less than 450 Gm.; moderate hypertrophy, between 450 and 550 Gm.; severe hypertrophy, those above 550 Gm. Some of the abnormal hearts showed hypertrophy of the right as well as the left ventricle. It was considered that a heart in which the ratio of left-to-right ventricular wall thickness was 3 to 1 or less showed an appreciable degree of right ventricular hypertrophy.

Initially, the clinical records had not been examined and only the principal clinical diagnosis was known at the time of the review of the electrocardiograms. However, in cases wherein left ventricular hypertrophy was thought to be absent at necropsy, a careful review of the clinical records was made in order to determine the presence or absence of any clinical evidence of cardiac disease or any possible cause for left ventricular hypertrophy.

RESULTS

The electrocardiographic diagnosis of left ventricular hypertrophy was made in 108 cases; it was confirmed pathologically in 75 cases, was considered questionable in 16 cases, and was not confirmed in 17 cases. In 24 of the 108 cases the hearts weighed less than the predicted upper limit of normal for body length, but 7 of those fell in the borderline group.

Before these 17 cases were accepted as false-positive cases, a careful review of clinical records was made. In none of them did the past history or final illness suggest a reason for left ventricular hypertrophy. Table 2 summarizes the pertinent clinical information and the essential electrocardiographic findings in the 17 false-positive cases. It is seen that none of the patients died of cardiovascular disease. It was furthermore noted that in no case was cardiac disease or hypertension present during the period of observation, nor was there a history thereof. Pathologic examination revealed the presence of myocardial scarring in 4 patients and myocardial metastasis in 1. The majority of these patients died of non-cardiac malignant tumors and showed emaciation.

A review of the electrocardiographic findings in this group showed that high voltage was present in all but 1 case. In addition, 6 patients showed lesser degree of ST-T abnormalities and 1 showed inversion of T waves in leads V4 to V6. It is noteworthy that of the 7 patients whose electrocardiograms showed ST-T abnormalities, in 4 fibrosis of the myocardium was demonstrated. Six patients showed delayed ventricular activation time in leads V5 and V6.

Thus, 17 patients who had no clinical evidence of cardiac disease and had no more than microscopic or focal myocardial changes at autopsy showed electrocardiographic evidence of left ventricular hypertrophy.

Table 3 summarizes electrocardiographic findings arranged by the 3 groups of criteria in the false-positive cases, in the borderline group, and in cases with pathologically confirmed left ventricular hypertrophy grouped by heart weights. It is seen that high voltage was present in all but 7 cases. It is noteworthy, however, that the false-positive group shows an appreciable number of cases in which high voltage was observed both in extremity and in precordial leads. The incidence of delayed
Table 2.—Summary of Findings in the Seventeen Cases of the False-Positive Group

<table>
<thead>
<tr>
<th>Age</th>
<th>Autopsy diagnosis</th>
<th>Weight (Gm.)</th>
<th>Heart criteria for electrocardiographic diagnosis of left ventricular hypertrophy</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Incr. voltage</td>
<td>VAT</td>
</tr>
<tr>
<td>26</td>
<td>Chorionic epithelioma</td>
<td>+ 170</td>
<td>SV₁ + RV₅,₆, aVF</td>
<td>V₅</td>
</tr>
<tr>
<td>75</td>
<td>Carcinoma of left lung</td>
<td>+ 240</td>
<td>SV₁ + RV₅,₆</td>
<td></td>
</tr>
<tr>
<td>57</td>
<td>Carcinoma of prostate</td>
<td>+ 250</td>
<td>SV₁ + RV₅,₆</td>
<td>T flat V₅ &amp; V₆</td>
</tr>
<tr>
<td>80</td>
<td>Carcinoma of bladder</td>
<td>+ 265</td>
<td>SV₁ + RV₅,₆</td>
<td>S-T depr. T inv. V₄, V₅, V₆</td>
</tr>
<tr>
<td>83</td>
<td>Carcinoma of parotid gland</td>
<td>+ 260</td>
<td>R₁ + S₃, Rᵥavl</td>
<td>T flat V₅,₆</td>
</tr>
<tr>
<td>47</td>
<td>Glioblastoma</td>
<td>+ 275</td>
<td>R₁ + S₃, Rᵥavl</td>
<td>V₅</td>
</tr>
<tr>
<td>61</td>
<td>Carcinoma of lung</td>
<td>+ 305</td>
<td>SV₁ + RV₅,₆</td>
<td>V₅, V₆</td>
</tr>
<tr>
<td>60</td>
<td>Carcinoma of prostate</td>
<td>0 290</td>
<td>SV₁ + RV₅,₆</td>
<td>T flat V₅,₆</td>
</tr>
<tr>
<td>74</td>
<td>Adenocarcinoma of gallbladder</td>
<td>+ 300</td>
<td>R₁ + S₃, SV₁ + R₅,₆</td>
<td>T flat V₅,₆</td>
</tr>
<tr>
<td>25</td>
<td>Sarcoma of thigh</td>
<td>+ 325</td>
<td>Rᵥavl, SV₁ + R₅,₆</td>
<td>V₅</td>
</tr>
<tr>
<td>51</td>
<td>Endothelioma</td>
<td>0 320</td>
<td></td>
<td>V₅,₆</td>
</tr>
<tr>
<td>60</td>
<td>Cirrhosis of liver</td>
<td>0 320</td>
<td>Rᵥavl, SV₁ + R₅,₆</td>
<td></td>
</tr>
<tr>
<td>35</td>
<td>Astrocytoma</td>
<td>+ 340</td>
<td>SV₁ + RV₅,₆, Rᵥavl</td>
<td></td>
</tr>
<tr>
<td>63</td>
<td>Lymphosarcoma</td>
<td>+ 330</td>
<td>SV₁ + RV₅,₆</td>
<td></td>
</tr>
<tr>
<td>34</td>
<td>Melanoma</td>
<td>+ 320</td>
<td>SV₁ + RV₅,₆</td>
<td>V₅</td>
</tr>
<tr>
<td>38</td>
<td>Subarachnoid hemorrhage</td>
<td>0 320</td>
<td>SV₁ + RV₅,₆</td>
<td></td>
</tr>
<tr>
<td>60</td>
<td>Carcinoma of pancreas</td>
<td>+ 300</td>
<td>SV₁ + RV₅,₆, Rᵥavl</td>
<td></td>
</tr>
</tbody>
</table>

VAT, prolonged ventricular activation time; ST-T, abnormalities of the ST-T portion of the electrocardiogram; V, vertical; SV, semivertical; I, intermediate; H, horizontal.
ventricular activation time in left precordial leads does not show a significantly different incidence between the negative and the positive groups. Abnormalities of the ST-T part of the ventricular complexes occur in positive and negative cases alike. Lesser degrees of such changes (ST depression and flattened T waves) show no major difference in incidence between the various groups. However, inverted T waves appear to occur almost exclusively in the positive cases. It is noteworthy, however, considering the fact that T-wave abnormalities are thought to be late effects of left ventricular hypertrophy, that not all of the heaviest hearts show T-wave changes and that 6 of the 40 cases in the heaviest group, which is 15 per cent, show essentially normal ST-T positions of the electrocardiogram.

Table 4 was constructed in order to examine a possible relationship between left ventricular hypertrophy and the position of the mean electric axis in the frontal plane. It shows the incidence of tracings showing the electric positions according to the conventional classification of Wilson in the negative group, the borderline group, the 3 positive groups, and in cases where the ratio of left-to-right ventricular-wall thickness suggested combined ventricular hypertrophy. There appears to be only a slight preponderance of a more horizontal rotation in heavier hearts than in lighter and in false-positive cases. The distribution of cases in the last column suggests that combined ventricular hypertrophy may have some effect of rotating the mean axis into a more vertical position, but this cannot be demonstrated in a significant manner in this small series.

Table 5 shows the incidence of single electrocardiographic criteria compared with combined criteria. It is noted that electrocardiograms showing more than 1 criteria appear to be more reliable in the diagnosis of left ventricular hypertrophy. A combination of high voltage or increased ventricular activation time with inversion of T wave appears to be the most specific combination for the diagnosis.

Discussion

The interpretation of the findings of this study has been approached with a great deal of caution because of the difficulty in the pathologic definition of what constitutes left ventricular hypertrophy. The most satisfactory method of determining the presence of left ventricular hypertrophy is the direct determination of left ventricular weight after the separation of the musculature of the 2 ventricles, as suggested by Lewis and others. The time-consuming preparation of such a specimen, however, does not permit routine use of this method for necropsies outside a selected and limited study. Investigation based on routine autopsy material can take into account either the measurement of the thickness of left ventricular wall or the total heart weight. Measurement of left ventricular wall is the less reliable of the 2 indices because of the variability of thickness in various parts of the heart, making comparison difficult, and because of unreliability of such a measurement in the presence of cardiac dilation. On the other hand, left ventricular musculature accounts for at least 75 per cent of the total cardiac weight, so that its hypertrophy will bring the total cardiac weight promptly out of the normal range, whereas right ventricular hypertrophy would affect total weight only in severe cases. In this study, in particular, the unlikely possibility of isolated right ventricular hypertrophy being responsible for increased total heart weight is almost entirely eliminated by the selection of the material, for only cases with electrocardiographic evidence of left ventricular hypertrophy were included. It was considered, therefore, that the most suitable index for left ventricular hypertrophy for this study was the increase in total cardiac weight, with the use of predicted weight values related to body length. The elimination of the borderline cases served as a better way to separate the normal and abnormal. On the other hand, the diagnosis of right ventricular hypertrophy had to depend on the much less reliable measurement of its wall thickness. An
arbitrary ratio of thickness of the 2 ventricular walls of 3 to 1 or less, was accepted as evidence that right ventricular hypertrophy is present in addition to left ventricular hypertrophy.

The decision to use total cardiac weight as a pathologic index of normality and to eliminate borderline cases was fully rewarded by the finding that in none of the 17 cases considered normal by such criteria was there clinical evidence of any factor that might have led to left ventricular hypertrophy. Thus, with no clinical evidence of left ventricular overload and no pathologic evidence of hypertrophy, this group of 17 cases could be unequivocally accepted as showing false-positive electrocardiographic findings for an acceptable diagnosis of left ventricular hypertrophy.

This group constitutes 16 per cent of cases in which the generally accepted electrocardiographic criteria for the diagnosis of left ventricular hypertrophy have been applied. The failure of such criteria in these 17 cases constitutes the most important finding of this study. The principal question appears to be what is the reason for an apparently normal heart producing an abnormal electrocardiographic tracing?

In answering this question, it should be pointed out that electrocardiographic criteria for the diagnosis of left ventricular hypertrophy are empirical, but have some theoretical basis. In the first place, the increased muscle mass creates a higher electric potential in the process of depolarization, thus accounting for the increased voltage. Furthermore, a longer time is required to depolarize the thickened muscle, which may be shown in the electrocardiogram by the delay in the onset of the intrinsic deflection and the prolongation of the QRS complexes, particularly the R wave in left-sided precordial leads. Finally, the repolarization process is altered either by the increased muscle mass or by the increased tension of the overloaded muscle resulting in ST-T changes in the electrocardiogram.

The finding, that in an appreciable number of cases abnormally high voltage is recorded in the electrocardiogram in the absence of increased left ventricular mass could be explained in 2 ways: either the normal heart can, under some circumstances, generate stronger depolarization forces, or some electrocardiographic leads may exaggerate and magnify a normal depolarization force. The first possibility is unlikely. On the other
hand, exaggeration of the electric potential by various indirect leads is a well-known phenomenon. Conventional extremity leads reflect fairly accurately a projection of the spatial mean vector, representing the main electric forces, onto the frontal plane. Precordial leads reflect the electric forces projected onto the transverse plane. Under ideal circumstances, the magnitude of the spatial mean vector could be accurately ascertained from its projection onto 2 planes. However, this relationship is marred in the human body by the variability of the conductivity of the transmitting media and by the unequal distances of the electrodes from the heart. It is generally agreed that extremity leads reflect electric forces more accurately than do precordial leads. The nearness of the precordial electrode to the heart distorts the electrocardiogram by magnifying the voltage of complexes. In children, adolescents, and young adults unusually high voltage is common in precordial leads. This is thought to be due to the thin chest and close proximity of the electrodes to the heart. Findings of this study demonstrate that a similar distortion may occur in older individuals. Most of the false-positive cases occurred in patients with considerable body emaciation caused by malignant tumors. This offers a reasonable explanation in some, though not all, cases. Other possibilities causing undue magnification of precordial QRS voltage could include vagaries of the anatomic position of the heart in the thorax, or changes in conductivity of the media between the heart and the electrode.

Prolonged ventricular activation time, measured in left-sided precordial leads, was the next most common criterion of left ventricular hypertrophy found in the false-positive group, having been abnormal in 6 of the 17 cases. It is noteworthy that it appears to be an unreliable diagnostic sign as evidenced by the fact that in almost half of the cases with the heaviest hearts, the ventricular activation time was normal. Inasmuch as there is no reason to doubt the validity of the relationship between the thickness of the muscle mass and the duration of the ventricular activation time in the direct electrogram, one has to conclude that the conventional precordial electrocardiogram may exaggerate or minimize its duration. Whether this is caused by the distance between the electrode and the heart or by a more tangential spread of the depolarization process is unknown.

Abnormalities of the ST-T portion of the electrocardiogram associated with left ventricular hypertrophy are the subject of controversy. The point in question is whether the alteration of the repolarization process that rotates the main ST and T forces away from the main QRS forces is caused by the increase in the muscular mass, thus being another direct effect of left ventricular hypertrophy, or is caused by changes developing secondarily to the hypertrophy or to the disease process, such as increased tension, relative ischemia, etc. This second viewpoint has been responsible for the use of the term "ventricular strain" to connote ST-T changes as opposed to "ventricular hypertrophy," which is applied to increased QRS voltage and increased ventricular activation time. This study
cannot shed light on this controversy. It does demonstrate, however, the importance of adding the ST-T abnormalities to the criteria of left ventricular hypertrophy, which otherwise, if it were to be based on increased voltage and delayed activation time, could never be diagnosed with certainty. To be sure, S-T segment depression, flattening and inversion of T waves are electrocardiographic abnormalities that can be caused by a great variety of pathologic and physiologic processes. Yet such abnormalities, when combined with increased voltage and prolonged activation time, add to the specificity of diagnostic criteria of left ventricular hypertrophy. The more advanced change, the inversion of T waves in leads with strongly positive QRS complexes, appears the most valuable addition to the diagnostic criteria, for only once was such change found in the false-positive group, and that was in a case in which focal fibrosis of the myocardium was present, which was the obvious cause of this electrocardiographic abnormality. Lesser ST-T abnormalities, such as S-T segment depression and flattening of T waves were found occasionally in the false-positive cases. Some such cases, though not all, showed the presence of focal disease of the myocardium. It is also possible that such electrocardiographic abnormalities might have been caused by the tendency of the T-wave vector to rotate more vertically and anteriorly in elderly individuals, wherein flat T waves appear in left precordial leads.

Abnormalities of the repolarization process appear to be such an integral part of the electrocardiographic pattern of left ventricular hypertrophy that it was thought to be preferable to use the all-inclusive term “hypertrophy” rather than the dual concept of “hypertrophy” and “strain.” The concept of strain appears to have some merit in acute processes wherein S-T and T changes appear or disappear under circumstances in which the time is too short for the development or the regression of ventricular hypertrophy. However, until more is known about the exact nature of “strain,” it may be assumed that it represents the same physiopathologic process that stimulates the growth of heart muscle.

The findings of this study suggest strongly that presently available criteria for the electrocardiographic diagnosis of left ventricular hypertrophy are reasonably satisfactory. Yet, on the one hand, normal hearts can undergo depolarization and repolarization in such a manner that conventional electrocardiographic tracing shows a seemingly abnormal picture, and, on the other hand, grossly hypertrophied hearts can, under some circumstances, present a near-normal electrocardiogram. This is presumably caused by the various distortions of the electric activity of the heart in their projections onto the conventional electrocardiographic leads. Because of these distortions, the magnitude, the direction, and the duration of the electric forces cannot be estimated accurately, so that no absolute criteria separating normal tracings from those of left ventricular hypertrophy can exist. Rather, one should consider electrocardiographic criteria for the diagnosis of left ventricular hypertrophy as expressing a probability that the left ventricle
is hypertrophied. Such probability appears to be higher in the presence of an average body build, and lower in the presence of emaciation, thin chest wall, or some other factor capable of distorting the transmission of the electric cardiac potential.

The greatest possibility of error in the diagnosis of left ventricular hypertrophy lies in the diagnosis of early hypertrophy. The characteristic picture of fully developed left ventricular hypertrophy consisting of leftwards (horizontal) rotation of the mean QRS axis, the high voltage, the delayed ventricular activation time, the prolonged QRS duration, and the characteristic S-T segment deviation and T-wave inversion are very unlikely to lead to a diagnostic error. However, it is important to note that severe hypertrophy of the left ventricle may exist while fulfilling only one of these criteria, so that the electrocardiographic diagnosis of "early" and "late" or "mild" and "severe" hypertrophy may be subject to a considerable error.

Throughout the study, an attempt has been made to visualize alterations of the electrocardiogram as a variation of the sequence of activation and the magnitude of the spatial electric forces, rather than as changes in the contour of the various waves. One is justified in asking the question, whether direct vectorcardiography would be superior in the diagnosis of left ventricular hypertrophy to conventional electrocardiography. At the present time, no uniform standards exist and the information at hand is yet inconclusive. However, it is doubtful that this method will succeed in the area in which conventional electrocardiography is most disappointing: the separation of early left ventricular hypertrophy from the normal heart.

**Summary**

In a series of 550 unselected electrocardiograms taken on patients in whom necropsy findings were later available, 108 tracings showed the pattern of left ventricular hypertrophy according to currently accepted electrocardiographic criteria. The analysis of the necropsy findings based on heart weights revealed that left ventricular hypertrophy was believed to be present in 75 cases, absent in 17 cases, and questionable in 16 cases.

A careful analysis of the 17 cases with normal cardiac weights was made and it was found that in none of the cases was there known cause for cardiac hypertrophy, nor was there significant cardiac disease present. The majority of patients in this group died of malignant disease and showed considerable emaciation.

The value of the 3 principal classes of electrocardiographic criteria was examined not only in the light of confirmed and false-positive cases, but also according to their incidence in cases with mild, moderate, and severe left ventricular hypertrophy determined by cardiac weight. The prolonged ventricular activation time was found to be the least reliable sign, having been present in many false-positive cases and absent in some cases with severe hypertrophy. Increased voltage of precordial leads, the most sensitive of the criteria, was present in most cases. It was also, however, most frequently responsible for a false-positive diagnosis of left ventricular hypertrophy. The depression of S-T segments, flattening and inversion of T waves in leads showing the highest electro-positive deflections when added to the other 2 groups of criteria materially increased the specificity of the diagnosis. However, the relationship between the extent of the electrocardiographic abnormalities and the severity of hypertrophy is only fair.

It is believed that these inaccuracies in the electrocardiographic diagnosis are inherent in the method and demonstrate the fact that conventional electrocardiography registers in a rather crude way the electric forces of cardiac action, being influenced in addition by such extraneous factors as body build, vagaries of anatomic positions of the heart, the degree of insulating effect of outside structures, and probably other, as yet unknown, factors.

Presently available electrocardiographic criteria for the diagnosis of left ventricular hypertrophy appear to be moderately satis-
factory. They have to be applied, however, with the understanding of their limitations: it is necessary to accept them as an expression of probability rather than a diagnosis of left ventricular hypertrophy. The disappointing inaccuracy of electrocardiography in the field of the diagnosis of early left ventricular hypertrophy is emphasized.

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SUMMARIO IN INTERLINGUA

In un serie de 550 non-seligite electrocardiogrammas, obtenite ab patientes pro qui reportos necroptic deveniva subseuentemente disponibile, 108 exhibiva le configuration de hypertrophia sinistro-ventricular secundo le currentemente acceptate criterios electrocardiographic. Le analyse del reportos necroptic revelavava pesos cardiac indicante le presentia de hypertrophia sinistro-ventricular in 75 casos e su absentia in 17 casos. In 16 casos le reporto necroptic eseva indecise.

Un analyse meticulose del 17 casos con normay pesos cardiac eseva effectuate. Esseva constata que il existeva in nulle de iste casos un causa cognoscite de hypertrophia cardiac, e nulle eseva caracterisate per le presentia de significative lesiones cardiac. Le majoritate del patientes in iste gruppo moriva ab morbos maligne e exhibiva grados considerabile de emaciaition.

Le valor del 3 classes principal de criterios electrocardiographic eseva examine non solmente in le lumine de casos confirmate e de casos false-positive sed etiam con referentia a lor incidentia de casos de leve, moderate, e sever hypertrophia sinistro-ventricular, judicate super le base del pesos cardiac. Esseva trovate que prolongation del tempore de activation ventricular es le minus fidel del signos de hypertrophia sinistro-ventricular. Illo eseva presente in numerose casos false-positive e absent in plures con hypertrophia sever. Augmento de voltage in de-

rivationes precordial eseva le plus sensibile del criterios. Su presentia eseva notate in le majoritate del casos, sed illo eseva etiam responsabile pro un plus grande portion del diagnoses false-positive que non importa qual altere criterio de hypertrophia sinistro-ventricular. Le depression del segmento S-T e le applanation e inversion del unda T in derivationes exhibite le plus alte deflexiones electro-positive—addite al criterios del 2 altere gruppos—aumentava le specificitate del diagnose a grados significative. Tamen, le relation inter le extension del anormalitates electrocardiographic e le grado de severitate del hypertrophia es solmente "satis bon."

Es formulate le opinion que iste inexactitudes del diagnose electrocardiographic inhere in le methodo mesme e servi a demonstrar le facto que le electrocardiographia conventional registra le fortia del action cardiac in un maniera paucu raffinata, proque illo es influentiate etiam per varie factores extrane, como per exemplo le conformation del corpore del patiente, le erratic variationes in le position anatomic del corde, le grado del effecto isolator de structuras externe, etc., incluse, il es probabile, un numero de factores que es ancora incognoseite.

Le currentemente disponibile criterios electrocardiographic pro le diagnose de hypertrophia sinistro-ventricular pare esser "moderateamente satisfactori." Tamen, illos debe esser applicate in plen recognition de lor limitationes. Il es necessari acceptar los como expression de probabilitate plus tosto que como diagnostic pro hypertrophia sinistro-ventricular. Es subliniate le disappunctante inexactitude del electrocardiographia in le campo del diagnose de hypertrophia sinistro-ventricular in stadios precoce.

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The authors report the clinical and postmortem findings in the case of a 73-year-old man who had suffered a coagulation defect since childhood diagnosed hemophilia on the basis of a prolonged coagulation time and a thromboplastin-generation test indicative of deficient antihemophilic globulin. He had experienced angina pectoris for 3 years before his death, which occurred during an episode of gastrointestinal bleeding. At postmortem examination, atheroma of the coronary arteries and aorta was found. It is suggested that such a case is evidence against Duguid's theory of the thrombotic origin of atheroma.

Kurland
Reliability of Electrocardiographic Diagnosis of Left Ventricular Hypertrophy
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