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Electrocardiographic findings in twelve cases of left ventricular hypertrophy of the heart

Loyd Raymond Schultz
University of Nebraska Medical Center

THE ELECTROCARDIOGRAPHIC FINDINGS IN TWELVE CASES OF LEFT VENTRICULAR HYPERTROPHY OF THE HEART

Loyd Raymond Schultz

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INTRODUCTION:

The purpose of this paper is to evaluate the criteria used by some of the present day authorities in the field of electrocardiography as to what are the most reliable electrocardiographic evidences of left ventricular hypertrophy.

Since the popularization of the electrocardiograph by Einthoven, the electrocardiographic patterns of left ventricular hypertrophy of the heart have received considerable attention from numerous investigators.

It is generally recognized that inspection, palpation and percussion indicate in an uncertain fashion the degree of total hypertrophy present in a large heart, and that they are especially uncertain indices in determining the preponderant hypertrophy of one ventricle as opposed to the other.

In 1906, Einthoven (1) found that cardiac hypertrophy is usually associated with variations in the form of the electrocardiogram and stated that hypertrophy of one or the other ventricle could be recognized by these changes in the shape of the electric curve. In 1915, Cotton (1) stated that the electrocardiographic signs of ventricular hypertrophy were the most reliable method of estimating either left or right ventricular hypertrophy. He presented a series of 6 cases of hypertrophy and analyzed the abnormal electrocardiographic signs present in the standard limb leads I, II and III.

Since the introduction of the unipolar and precordial leads, there has been renewed interest in the electrocardiographic diagnosis of left ventricular hypertrophy.

METHOD AND MATERIAL USED:

Gross weights and the ventricular thicknes of the hearts in this study were obtained from the University of Nebraska pathology autopsy records. Hypertrophy was considered to be present when the heart weighed 350 grams or more and when the left ventricle was greater in thicknes than 15 millimeters. These are the hearts considered by this department as being hypertrophied.

Twelve autopsied hearts were studied in this analysis. The post mortum weights of these hearts ranged from 350 to 700 grams, the left ventricular thicknes ranged from 15 to 27 millimeters. Thus all should be considered hypertrophied by the criteria used in this paper. All of these patients had had antimortum electrocardiograms and the electrocardiographic diagnoses were noted in each case. Three of the patients included in this study were receiving digitalis at the time the electrocardiograph was taken, three had electrocardiographic evidence of old infarcts, one right bundle branch block and one Wolff Parkinson White Syndrome were included. No controls were used in this study.

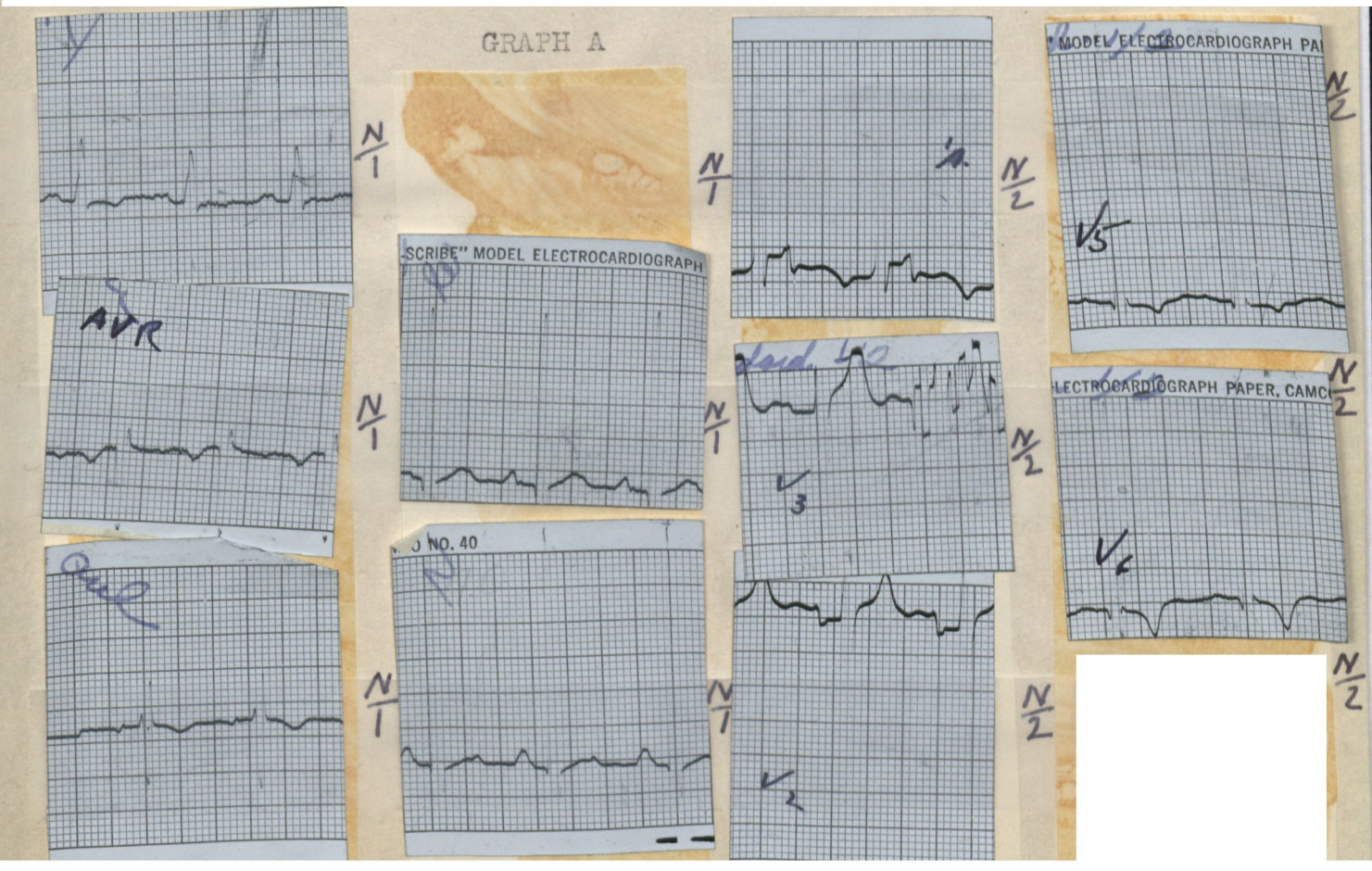
The electrocardiographic abnormalities studied for their significance included: Intrinsicoid deflection in precordial leads V1 and V6; voltage in unipolar limb leads aVL, aVF and precordial lead V5; RST-T pattern in standard leads I and II, precordial lead V5 and unipolar lead aVL if the heart was horizontal or in lead II, III, aVF and V5 if the heart was verti-

cal; intra-ventricular conduction time in standard lead II, precordial lead V5 and unipolar lead aVL; Q wave in precordial lead V5 and unipolar leads aVL and aVF; S wave in standard lead I; and position of the heart, whether horizontal or vertical.

The electrocardiograms were analyzed and recorded in chart form. The amplitude of the upright waves was measured from the upper edge of the base line to the peak of the R wave; the inverted waves, from the lower edge.

Graph A is the electrocardiogram of case nine, the largest heart in this series. Characteristic features of left ventricular hypertrophy shown in this electrocardiograph are: (a) high voltage QRS; (b) RS-T segment depression with upward bowing and a T wave that is inverted and asymetric; and (c) delayed intrinsicoid deflection.

GRAPH A


GRAPH A
I
N/1
aVR
N/1
SCRIBE" MODEL ELECTROCARDIOGRAPH
N/1
NO. 40
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N/1
V1
N/2
V3
N/2
V2
N/2
MODEL ELECTROCARDIOGRAPH PA
N/2
V5
ECTROCARDIOGRAPH PAPER, CAMC
N/2
V4
N/2

Table I shows the heart weight, the left ventricular thickness, the anti-mortum electrocardiographic diagnosis and the post mortum microscopic diagnosis in each case used in this study. It will be noted that left ventricular hypertrophy was diagnosed by antimortum electrocardiographs in only three cases out of the twelve. (25%) There was no correlation with this diagnosis and the post mortum weight of the heart.

TABLE I

Case	Heart Weight	Left Ventricle Thickness	EKG Diagnosis	Microscopic Diagnosis
1.	500 grams	20 mm.	(a) old anterior septal infarct (b) LVH (c) LVS (d) 1st degree AV block.	(a) Epidermoid CA of Bronchus Metastatic to the heart.
2.	400 grams	15 mm.	(a) frequent APC	(a) LVH
3.	450 grams	15 mm.	(a) A. fibrulation (b) defective IV conduction	(a) LVH
4.	440 grams	17 mm.	(a) subendocardial infarct or (b) LVS	(a) LVH
5.	350 grams	18 mm.	(a) RBBB (b) possible RVH	(a) LVH (b) RVH
6.	440 grams	21 mm.	(a) LVH (b) LVS	(a) mitral valvulitis (b) old Rheumatic fever
7.	400 grams	16 mm.	(a) Old ant infarct (b) LVS (c) Wolff Parkinson White Syndrome	(a) aneurysm of left ventricle (b) fibrotic ant apical infarct.
8.	400 grams	20 mm.	(a) RAD	(a) LVH

TABLE I
(continued)

Case	Heart Weight	Left Ventricular Thickness	EKG Diagnosis	Microscopic Diagnosis.
9.	700 grams	27 mm.	(a) LVH (b) LVS and/or Digitalis effect	(a) LVH
10.	550 grams	25 mm.	(a) frequent APC (b) frequent VPC (c) auricular hypertrophy	(a) LVH
11.	420 grams	16 mm.	(a) normal	(a) LVH
12.	560 grams	20 mm.	(a) 1st degree block (b) Hyperkalemia	(a) LVH

Comparison of anti-mortum electrocardiographic diagnosis and post mortum microscopic diagnosis in each case studied in this series.

RESULTS

(a) Voltage

The voltage of R in the unipolar leads aVL and aVF and the precordial lead V5 in this study can be seen in Table II. The mean height of the R wave in V5 in the left ventricular hypertrophied hearts was 21 mm. This mean is compared to the mean of R in V5 in normal hearts which is 11.8 mm. (2) This figure was exceeded by 58% of these hearts, the maximum for normal hearts of 25mm. in V5 was exceeded by 25% of the hypertrophied hearts in this study.

TABLE II

Case	aVL	aVF	V5
1.	19mm.	4mm.	21mm.
2.	0mm.	8mm.	13mm.

TABLE II (continued)

Case	aVL	aVF	V5
3.	4mm.	3mm.	22mm.
4.	23mm.	2mm.	8mm.
5.	3mm.	0mm.	10mm.
6.	28mm.	3mm.	37mm.
7.	4mm.	2mm.	4mm.
8.	0mm.	9mm.	11mm.
9.	2mm.	35mm.	82mm.
10.	2mm.	8mm.	26mm.
11.	6mm.	3mm.	18mm.
12.	6mm.	2mm.	3mm.
Percent abnormal	25%	8%	25%

Voltage in the leads analyzed in this study for height of the R wave in millimeters.

Numerous investigators have commented on high voltage QRS complexes as being one of the characteristic features of left ventricular hypertrophy, expecially if the prominent R wave is in the left axillary and precordial leads which face the epicardial surface of the hypertrophied left ventricle. (3, 4, 5, 6). The value of the electrocardiogram in determining high voltage is limited however, because it is difficult to establish any limits of normal amplitude of the QRS. This is because the amplitude is not only a function of the mass of the heart, but also of the nearness of the electrode to the heart. Thus a tall, thin, man may have taller complexes in V5 than the short, thick,

chested man, even though the former may have a normal heart and the latter have considerable left ventricular hypertrophy. (6) Left ventricular hypertrophy often occurs even in normal build individuals, without high voltage appearing in the electrocardiograph. (7, 8) This fact was adaquately demonstrated in this study.

Although lead V5 may normally have an R wave 33 mm. tall (6), Sokolow and Lyon's criteria for R wave voltage in left ventricular hypertrophy was used in this paper. These investigators state that when the R wave in the precordial lead V5 exceeds 25 mm., left ventricular hypertrophy can be suspected. (2).

Of equal value to V5 was R in aVL. In normal horizontal hearts, 99 per cent of the subjects may be expected to have an R wave in aVL of less than 11.1 millimeters. (2). This voltage was exceeded in 25% of these cases of left ventricular hypertrophy. The diagnostic value of aVF was considerably less, since in only one (8%) case was the R wave greater than 20mm., which is the upper limit of normal for this lead. (2)

Einthoven (10) believed that left ventricular hypertrophy was present when the tallest QRS summit was present in lead I and the deepest deflection was present in lead III, and that usually the deflections were of great amplitude. Lewis (11) also commented on the high voltage of R1 and S3, but also noticed the decrease in amplitude of S1 and R3 which was usually present in left ventricular hypertrophy. He felt that Q1, S3 and R1 are left ventricular effects, and that therefore these

electro-cardiographic effects are preponderent in left ventricular hypertrophy.

By using tall QRS complexes as criteria for hypertrophy, Herman (10) showed that the electrocardiogram always showed evidence of left ventricular hypertrophy if the left ventricle weighed more than 300 grams post mortum, and that if the left ventricle weighed less than 250 grams, there was very little relation between the form of the electrocardiogram and the weight of the heart.

According to Falzoi (12), high voltage of the R wave associated with low voltage of the T wave, especially in lead I, V4, V5, and V6, may be an early sign of left ventricular hypertrophy.

Just why the hypertrophied heart should produce a greater amplitude on the electrocardiogram than the normal heart is not known. Even though the infant has a much smaller heart than the adult, the amplitude of the electrocardiographic complexes are almost the same. (10). The manifest potential difference in the electrocardiograph varies with the conductivity of the tissues around the heart and with the plane of the electrode. Einthoven (10) felt that variations in amplitude of the electrical deflections occur with variations in the contractibility of the heart muscle. A more recent proposed explanation for the high voltage curves of left ventricular hypertrophy is that the arrangement of the ventricular conducting system is disturbed in large hearts. (4). Wilson (4) states that the cause for the voltage increase may be due to the combined effects of increase

in muscle mass, the thickness of the septum, free walls and total epicardial area and increase size of the chambers. The high voltage may also be due to the diminished density of the junctions between the purkinje tissue and the subendocardial muscle and an increase in the length of the paths transversed by the excitation wave on it's way to some of these junctions.

There is some evidence that large hearts may not normally produce high voltage. King and his associates (13) found that the R wave in leads corresponding to lead III were low voltage (less than 3 to 4 mm.) in the Beluga whale. This heart, after death, weighed 2722 grams, the left ventricular thickness was 20 millimeters. He admits, however, that the electrodes were some distance from the heart and in moderate proximity to each other, which may have accounted for the low voltage.

(b) POSITION OF THE HEART:

The electrocardiographic position of the heart was determined in each case. (Table III)

TABLE III

Case	Electrocardiographic position	S wave in Lead I
1.	horizontal	present
2.	vertical	absent
3.	horizontal	absent
4.	horizontal	present
5.	horizontal	present
6.	horizontal	absent
7.	horizontal	absent

TABLE III (continued)

Case	Electrocardiographic position	S wave in Lead I
8.	vertical	absent
9.	vertical	present
10.	vertical	present
11.	horizontal	absent
12.	horizontal	absent

Electrocardiographic position and absence or presence of S wave in Lead I.

Hearts were considered, in this study, to be horizontal when lead aVL showed a qR or QR pattern, vertical when aVL showed QS or rS pattern. None of the hearts studied could be classified as being either semi-horizontal or semi-vertical from their electrocardiographic patterns. All hearts that had a horizontal electrocardiographic position showed left axis deviation. As shown in Table III, 66% of the patients had horizontal hearts.

In a study of 147 patients with known left ventricular hypertrophy by Sokolow (2), 57% had horizontal or semi-horizontal hearts. Kaplin (14) found 80% of 148 cases of left ventricular hypertrophy showed left axis deviation. He believes that the absence of the horizontal position may be due to the neutralizing effect of either a concomitant right ventricular hypertrophy or a change in heart position. Other investigators have also noted the frequent association of horizontal hearts with left ventricular hypertrophy. (15). Marked left axis deviation may

also occur in brown atrophy of the heart, (16), pregnancy (17), and obesity (18). About one forth of the normal people will show left axis shift. (14). Occasionally the electrocardiographic position of the heart may change within a short period of time, often with no reason for this change in position being found.(19).

(c) RST-T PATTERN:

RS-T segment and T wave abnormalities are frequently encountered in left ventricular hypertrophy. The frequency of these abnormalities in this study can be seen in Table IV, where 75% of the cases had some abnormality of the characteristic pattern of left ventricular hypertrophy present. (Graph A). This pattern is an RS-T segment which is depressed and bowed upward and a T wave that is inverted and asymetric.

TABLE IV(a)

Case	RST Segment in Leads: I	II	III	aVL	aVF	V5
1.	depressed convex	normal	- - ---	Depressed convex	- - - -	depressed convex
2.	- - - -	normal	depressed	- - - -	normal	normal
3.*	depressed convex	normal	- - - - -	normal	- - - -	normal
4.*	depressed convex	normal	- - - - -	depressed convex	- - - -	depressed
5.	normal	normal	- - - - -	normal	- - - -	normal
6.	depressed convex	normal	- - - - -	depressed convex	- - - -	normal
7.	depressed	normal	- - - - -	normal	- - - -	normal
8.	- - - -	normal	normal	- - - -	normal	normal
9.*	- - - -	depressed convex	normal	- - - -	normal	depressed convex

TABLE IV (a) (continued)

Case	I	II	III	aVL	aVF	V5
10.	- - - -	normal	normal	- - - -	normal	normal
11.	normal	depressed convex	- - - -	normal	- - - -	normal
12.*	depressed convex	normal	- - - -	depressed convex	- - - -	normal

TABLE IV (b)
T waves; inverted and asymetric or not, in leads:

Case	I	II	III	aVL	aVF	V5
1.	yes	no	- - -	yes	- - -	yes
2.	- - -	no	no	- - -	no	no
3.*	no	no	- - -	no	- - -	no
4.*	yes	no	- - -	yes	- - -	no
5.	no	no	- - -	no	- - -	no
6.	yes	no	- - -	yes	- - -	yes
7.	no	no	- - -	no	- - -	no
8.	- - -	no	no	- - -	no	no
9.*	- - -	yes	no	- - -	no	yes
10.	- - -	no	no	- - -	no	no
11.	no	no	- - -	no	- - -	no
12.*	no	no	- - -	no	- - -	no

* Patient was on digitalis at the time that the electrocardiogram was taken.

In the characteristic pattern of left ventricular hypertrophy, both the RS-T and the T wave are directed downward, in contrast to the usual appearance of acute coronary disease where the RS-T and T are in opposite directions. Thirty-three percent of these patients showed all the characteristics of this

complex to be present. These ST segment and T wave abnormalities, in the leads reflecting the surface of the left ventricle, are the least specific of any for the evidence of left ventricular hypertrophy, according to Dimond. (6). Others however, believe that the ST segment depression with the upward bowing is one of the most common signs of left ventricular hypertrophy. (2). These changes are shown in the standard leads and the left precordial leads because the position of the heart is usually horizontal. Sokolow (2) believes that the precordial leads usually are the first to show RS-T abnormalities in left ventricular hypertrophy. Goulder and his associates (7), made a study of 65 patients with left ventricular hypertrophy, diagnosed on the basis of X ray changes characteristic of this condition. These were compared with 30 normal individuals with horizontal hearts. Their data demonstrated that aVL differientiated the hypertrophied group from the normal individuals more frequently than either V5 or V6 alone. They noted that in aVL, R was greater than 11 mm., T less than .6mm. and the T/R ratio less than 10 percent in 85% of the cases with hypertrophy. Wilson (4) has reported similar findings of changes in aVL, when precordial lead changes are borderline. There was no case in this study, however, where there were changes in lead aVL and none found in Lead I or in pre-cordial lead V5. This fact may have it's explanation in the fact that none of the hearts studied could be considered as having a borderline hypertrophy.

Sokolow (2), found that 136 cases out of 147 cases of left

ventricular hypertrophy showed abnormal RST-T findings. Digitalis may produce depressed ST intervals, but these depressions have certain characteristics which differentiate them from left ventricular hypertrophy. These changes are discussed in some detail in the discussion section of this paper. When both the digitalis effect and ventricular hypertrophy are present in the same electrocardiographic tracing, the two conditions may modify each other in variable ways.

Those investigators who believe that left ventricular strain may occur believe that strain of the left ventricle is almost always accompanied by hypertrophy, but that occasionally one may see an isolated pattern of strain, that is normal QRS complexes, but depressed ST segment and T waves in the left precordial leads. The electrocardiograph of left ventricular hypertrophy without strain according to Kaplan (18), shows deep S waves over the right pre-cordium, tall R waves over the left pre-cordium and normal ST segment and T waves. These T and ST abnormalities are reversible when true strain exists.

Low T waves in the left pre-cordial leads are an early sign of left ventricular hypertrophy, according to some investigators, (2, 12), while inverted T waves in the leads with the tallest R waves are later signs. (4).

King (13), recorded the electrocardiograph of a whale whose heart weight post mortum was 2722 grams and noted inverted T waves but the RS-T segment was slightly elevated.

Rapid changes of the RS-T segment and T waves in the left ventricular leads when serial tracings are made may occur in

left ventricular hypertrophy, which are suggestive of anterior wall infarction. These have been noted when the heart is subjected to some extrinsic stress, such as thyrotoxicosis. (3).

The ST-T deviations in lead I may be evidence of changes in retreat of activation of the heart muscle mass, partly due to widespread changes in the conduction system in the left ventricule, and partly a relative ischemia of the muscle as it outgrows it's blood supply. (14). Another more generally used explanation for this finding is the increase in the intracardiac pressure against the endocardium which occurs in those conditions associated with ventricular hypertrophy. Thus, in the normal individual, there is a gradient in the pressure from endocardium to epicardium but with the increased intracardiac pressure, secondary to the basic disease, this gradient is lost and the entire thickness of the muscle is subjected to an exaggerated pressure. In the normal heart, the epicardial surface is able to repolarize more rapidly than the endocardial surface, while in the patient with increased endocardial pressure, both endocardial and epicardial areas are delayed in their repolarization with the endocardial region recovering first. (6).

T wave alterations are extremely non-specific. Marshall (24), noted that T waves could be altered by endocrine factors (thyroid); electrolyte changes (K and calcium); fever; avitaminosis (bere bere); nervous factors (vagal, sympathetic); and drugs (digitalis, epinepherine, amyl nitrate, atropine, and quinidine); also be eating, by drinking cold water, hypoglycemia, by neurocirculatory asthenia and by emotions. France (25) has

noted flattening or T wave inversion with normal respirations.

In the vertical heart, the electrocardiographic pattern of left ventricular hypertrophy is not the same as the pattern in the horizontal heart, In the vertical heart, leads II, III, and aVF reveal the RST-T abnormalities which originate in the left ventricle. However, precordial leads V5 and V6 show the same type of abnormalities in left ventricular hypertrophy, whether the heart is horizontal or vertical. Sometimes, however, the only abnormality in V5 and V6 is only slight depression of the RS-T segment, with a transitional zone displaced to the left. Diagnosis of left ventricular hypertrophy on this abnormal electrocardiographic finding only, should be made with great caution, however. In a study of 650 athletes, Klemola (22) noted frequent abnormal T waves in lead I and II. In almost all of these cases, no other signs of heart disease could be found.

Levine (23) made a study of 150 consecutive autopsied cases and states that left ventricular hypertrophy, when present, would probably have been diagnosed in 7 of the cases which were missed if additional tracings had been taken one or two interspaces higher or had been recorded farther to the left in the posterior axillary or scapular line. For this reason, Johnston (19) believes that the usual sights for placement of the exploring electrodes are not sufficient if the routine tracing shows any pecularity in the ventricular complex such as broad transition zone, displacement to the left or right, or failure to obtain tracings of the left or right ventricular

type with the routine multiple precordial leads and that additional tracings at higher or lower levels or farther to the right or left should be taken to help explain any of these abnormalities.

(d) INTRINSICOID DEFLECTION:

In the present series of 12 cases of left ventricular hypertrophy, five (42 %) had intrinsicoid deflections of .06 seconds or longer in V5. There was no correlation between the weight or the left ventricular thickness of the heart and the length of the ventricular activation time. The results of the intrinsicoid deflection measurments in both V1 and V5 can be seen in table V. In only one heart (case 5), was the ventricular activation time in V1 greater than .03 seconds, which is considered upper limit of normal in this lead.

TABLE V

Case	Lead V1	Lead V5
1.	.00	.06
2.	.02	.04
3.	.03	.06
4.	.00	.04
5.	.10	.04
6.	.02	.06
7.	.02	.12
8.	.00	.04
9.	.02	.06
10.	.00	.04
11.	.02	.04
12.	.02	.04

Intrinsicoid deflection in seconds in Lead V1 and V5.

Many investigators have emphasized the importance of the time of the appearance of the intrinsic deflection in the recognition of ventricular hypertrophy and bundle branch block. (2, 4). An increase in the mass of the myocardium, as is present in left ventricular hypertrophy, would be expected to lengthen the time of the onset of the intrinsic deflection, which is the time of the onset of the QRS to the peak of the R wave or the beginning of the abrupt downstroke of the QRS. This represents the time interval required for the passage of the impulse through the ventricle to the epicardium under the exploring electrode. (3, 4). However, the delay in the intrinsicoid deflection may not be a true index of the endocardial-epicardial thickness of the muscle . It has been found that there is considerable degree of tangential spread of the wave of depolarization present, rather than a simple radial endo-epicardial route as was previously thought. This would tend to lessen the value of the intrinsic deflection in measuring the degree of hypertrophy. (6, 19). The natural delay in direct writing instruments also decreases the diagnostic value of the intrinsic deflection. (6).

The intrinsic deflection in leads V5 and V6 was found to be less than .06 seconds in three different series of normal people comprising over 280 cases. (2, 9). Kossman (9), states that the time of the intrinsic deflection in the normal individual averages .02 second in lead V1 and .04 second in V5. Other investigators state that the upper limit of normal in V1 is .03 second and in V5 is .05 second. (2). Sokolow and Lyon (2),

found in a study of 147 cases of known left ventricular hypertrophy, that 58% had ventricular activation times in V5 of .05 to .08 seconds. In this same study they also noted that 40% of these cases had normal intrinsicoid deflections, even though other findings of left ventricular hypertrophy such as depressed RST segment and inverted T waves were present. On the other hand,however, in some cases with roentgenologic evidence of left ventricular hypertrophy, a delayed intrinsic deflection in V6 was the only abnormal electrocardiographic sign present.

Ungerleider (24) found that 13% of a series of 147 patients who had ventricular activation times of .06 seconds, had no roentgenologic evidence of cardiac hypertrophy. Cardiac hypertrophy may be present without roentgenologic evidence of such, according to Sokolow (2).

(e) INTRAVENTRICULAR CONDUCTION:

The total QRS duration and it's relation to the intrinsicoid deflection was studied in this series of 12 hypertrophied hearts. Since two of the cases included in this study had associated disease processes which could account for the prolonged QRS (Case 5 with right bundle branch block and Case 7 with Wolff Parkinson White Syndrome), these cases were not included in the analysis of the QRS duration. In the remaining ten cases, 30% had QRS durations of .11 seconds or longer, but none had left ventricular activation times exceeding .08 second, which is characteristic for bundle branch block as will be pointed out later.

TABLE IV

Case	Lead II	Lead V5	Lead aVL
1.	.10	.11	.12
2.	.08	.09	.08
3.	.11	.11	.09
4.	.09	.09	.09
5.*	- -	- -	- -
6.	.12	.11	.11
7.**	- -	- -	- -
8.	.10	.09	.09
9.	.09	.08	.09
10.	.08	.10	.08
11.	.08	.07	.08
12.	.08	.10	.06

QRS duration in seconds in leads II, V5, and aVL.
* Not included in this analysis because of associated right bundle branch block.
** Not included in this analysis because of associated Wolff Parkinson White Syndrome.

Wilson and his associates (4) have noted that in left ventricular hypertrophy, the total length of the QRS may exceed .10 seconds, often being .12 seconds without the electrocardiographic pattern of left bundle branch block being present. In a study of 147 cases of left ventricular hypertrophy, 12% of these patients had QRS durations of .11 to .12 seconds. None had evidence of left bundle branch block.(2).

Some investigators believe that, as the heart hypertrophies, the capillary blood supply becomes insufficient and not commensurate with the increase of myocardial mass. This, they state is the cause for the prolonged conduction times seen in

right and left ventricular hypertrophy. (25, 26).

Since the QRS complex is considered to be the duration of spread of the excitation wave through the ventricles, persons with larger than normal hearts, or with enlarged hearts from hypertension, the time intervals may be above the generally recognized upper limits of normal for adults of .10 seconds, yet these people may not have myocardial disease, per se, to produce atrioventricular or bundle branch block. (27). White (27) states that as the heart increases in size, the normal QRS interval increases. He found in electrocardiograms run on animals with large hearts, that the QRS interval normally ranged between .12 to .18 seconds for elephants and .19 to .22 seconds for the whale.

Electrocardiographic differences between left bundle branch block and left ventricular hypertrophy, both of which may have prolonged QRS intervals are: (a) in left bundle branch block, the ventricular activation time almost always exceeds .08 seconds, whereas it rarely reaches this figure in left ventricular hypertrophy; (b) in left ventricular hypertrophy with a QRS duration of over .12 seconds and a delayed intrinsic deflection, the peak of the R in the ventricular complex is usually found to be tall and sharp, while in left bundle branch block, the peak of the R in leads V5 or V6 is bread topped, notched, or "M" shaped, reflecting the delay in the spread of the impulse through the left ventricle. (2).

(f) Q WAVES:

Q waves were very commonly seen in this series of cases

with left ventricular hypertrophy, being found in 83% of the cases in either the unipolar leads aVF or aVL, or the precordial lead V5. In only one case did the Q wave exceed 3.00 millimeter in depth, and in three cases the maximum Q/R ratio of 25% was exceeded. The width and depth of the Q waves in the leads studied are showen in Table VII.

TABLE VII
Q wave

	Width in seconds			Depth		
Case	aVL	aVF	V5	aVL	aVF	V5
1.	.04	0.0	.02	2.0 (12%)	.0	1.0*
2.	.00	.03	.02	0.0	.0	0.5*
3.	.04	.00	.00	1.0(25%)	.0	0.0
4.	.00	.00	.00	.0	.0	.0
5.	.01	.00	.00	.25*	.0	.0
6.	.03	.00	.04	1.00*	.0	.0
7.	.03	.00	.04	1.00(25%)	.0	.0
8.	.00	.01	.00	.00	1.0*	.0
9.	.00	.03	.03	.00	2.0*	1.0*
10.	.00	.00	.00	.00	.0	.0
11.	.02	.00	.00	1.00*	.0	.0
12.	.02	.00	.00	1.5(25%)	.0	.0

Q wave width in seconds and depth in millimeters, in the leads analyzed in this series. Percentage listed refers to the percent of the height of that particular Q wave to the succeeding R wave.
* less than 10% of the succeeding R wave.

Criteria for abnormality of Q waves varies widely. (3, 25, 28). According to Sokolow and Lyon, (2) Q waves may normally be found in the left precordial leads and in any unipolar lead taken from a point on the body toward which the left ventricular

potentials are directed. Thus in horizontal hearts, Q waves may normally be seen in aVL as well as in leads V4 through V6.

Q waves represent negative potentials transmitted from the endocardial surface of the left side of the septum through the intervening structures to the axilla. This wave is recorded durring the brief period that normally elapses before the impulse reaches the subendocardial layer of the lateral wall.

About 33% of normal people will show Q waves in the left precordial leads and in lead aVL. (2). These Q waves are rarely greater than 3 millimeters in depth or greater than 25% of the preceeding R wave. (2, 3). Myers (3) studied 1,875 patients with multiple precordial leads and autopsy and found no case without pathological demonstrated infarct encountered in which the Q wave in the left ventricular lead V6, exceeded 25% of the subsequent R wave. Dimond (6) believes that laberal myecardial infarct should be considered as being present if the Q wave in lead aVL is .04 seconds wide, if it represents 50% of the preceeding QRS complex and when it is followed by a convex elevated RS-T segment with late inversion of the T wave.

Occasionally, however, in normal people Q deflections of greater than 30% of the entire QRS complex and a width greater than .04 seconds may occur being due to a vertical heart with marked clockwise rotation and displacement of it's apex backward. This may or may not be associated with a downward T wave. (28)

A number of investigators all found that in the presence of large Q3, left ventricular hypertrophy was present in a high percentage of cases. (21, 25). In France's (21) series

of 12 autopsies on patients with deep Q3 waves, left ventricular hypertrophy was present in 10, while infarct was present in only five. Unverlieder (24) believes that the deep Q3 in left ventricular hypertrophy is present because a thick left ventricle will accentuate the degree of left axis deviation and will cause a positive potential in the left arm electrode. Wilson (4) on the other hand, has stated that the deep and wide Q wave is possibly due to the delay in activation of the subendocardial muscle. The exaggerated Q waves which are often seen in V5 in left ventricular hypertrophy may also be due to : (a) the greater voltage developed durring activation of the hypertrophied septum or (b) improved transmission to the axilla because of the closer approach of the enlarged left ventricle to the thoracic cage. (3).

France (21) noted that conspicuous Q3 wave could occur in patients with an elevated diaphram, with lateral displacement of the apex, in pregnant women in the last trimester, and occasionally in cases where the cardiac axis was changed durring respiration.

(g) S WAVE CHANGES:

Table III records the absence or presence of an S wave in lead I in this series of cases. In this series, there were no S waves in this lead in 75% of the cases.

Absent S wave in lead I has been reported as being the most frequent single electrocardiographic abnormality that occurs in left ventricular hypertrophy. (24) In a series of 171 cases of advanced hypertension and left axis deviation, there were

no S waves in lead I in 66% of the cases, which was about two times as frequent as any other electrocardiographic abnormality shown. (24). Absent S waves in this lead is not very specific however, since it occures in about 18 percent of normal individuals with left axis deviation.

DISCUSSION:

The heart weight that should be considered hypertrophied is extremely difficult to determine when routine autopsy weights are used. The Armed Forces Institute of pathology - 1954 (29) stated that the average weight for the normal adult heart of men between 20 and 30 years of age is 275 to 325 grams. The left ventricularthickness average is 8-10 millimeters. Since most of the patients used in this study were in a considerably older age group, it was felt that these weights were too low to be considered normal in this study.

Many authors have commented on the inaccuracy of the cardiac weights and measurements for cardiac hypertrophy as it was used in this series of cases. Stoffer, (30) for example, stated that estimates of hypertrophy based on measurements of the thickness of the ventricular walls are at the best only rough approximations and are seriously in error in cases where presence of dilitation, scarring, or contraction presists after death. Fulton (31) has noted the inaccuracy of traditional methods of recording the weight of the heart due to the large and varying proportion of the weight that is accounted for by structures other than the myocardium. This may include up to 100 grams of non-myocardial tissue. He believes that the thickness of the ventricular wall is not satisfactory because is may

be greatly modified by dilitation of the cavity. He described a technique of dividing the heart into the free right ventricular wall, the free left ventricularwall and the septum and gives the range of normal for these weights in 202 normal hearts. His total upper normal ventricular weight was 250 grams. Another method of removing the heart has been devised by Myers. (3) He severs the great vessels at the point of passage through the pericardium. Normal left and right ventricle segment weights by this method are 181 grams for males and 146 grams for females. This is about 59% of the total heart weight, which would mean that the total heart weight normal for males is approximately 308 grams and for females approximately 250 grams. Another reason that any heart weight is a deceptive measure of heart hypertrophy or size is because it also weighs tissue in the heart other than muscle, such as tumor tissue, fibrosis and necrosis. (32).

Only about one-quarter of the cases with hypertrophy of both ventricles is likely to show a characteristic electrocardiogram. (33). In this series normal curves occuredin 8% of the cases and the signs of left ventricular hypertrophy occured in the remaining 92%. Although the one heart which was considered normal did have one of the signs suggestive of left ventricular hypertrophy present, (absent S wave in lead I), this sign was not considered to be specific enough for a diagnosis of left ventricular hypertrophy to be made. One other case, (number 10) had only a single abnormal sign present, this was high voltage in V5. (26mm) It is questionable whether or not this could be considered an abnormal electrocardiogram on this basis.

A comparison of the abnormalities found in this study with those found by other investigators is shown in Table VIII.

TABLE VIII

Criteria	this study	Pagnoni Goodwin	Sokolow Lyon	Heine Sackett Serber	Unger-leider Guber	Kap lin
RaVL greater than 11 mm.	25%		22%	19%		
RV5 greater than 26mm.	25%		20%	39%		
ID of V5 greater than .05 seconds.	42%	16%	35%	11%		
ST-T abnormal	75%	43%	93%	42%	30%	
Horizontal heart	67%		57%			80%
Vertical heart	33%					
QRS longer than .10 seconds.	30%*					
S in Lead I absent	58%				64%	

* Two cases in this series were excluded when the QRS duration was analysed because of associated conditions: RBBB and Wolff Parkinson White Syndrome in case 5 and 7 respectively.

Comparison of Incidence of Electrocardiographic Criteria of LVH used in this study and in those studies by other investigators.

RST segment changes were seen most commonly in lead I. The greater incidence of RST-T segment abnormalities in this series than in all the other series noted in table VIII except that of Sokolow and Lyon could be due to : (a) the type of hearts studied in this series or (b) the inclusion of 4 cases that were on digitalis at the time the electrocardiograph was taken. The so-called digitalis S-T-T contour consists of a depressed, downwardly sloping, straight lined S-T segment which continues directly into an inverted T wave without any indication

of the junction between the S-T and T waves. The digitalis pattern does not usually develop with therapeutic doses of digitalis, but appears only when the toxic dose of digitalis is approached. (34). Since the patients in this series who were on digitalis had all taken less than 1.2 grams of digitalis when the electrocardiographs were taken, it was felt that RST-T segment abnormalities could be included in the analysis of the effects of left ventricular hypertrophy.

The RST depression and upward bowing was more commonly encountered than inversion and asymetry of the T wave in this series. In one case, (no. 3), RST depression and bowing was the only abnormality encountered in the RST-T segment. Since this patient was on digitalis at the time that the electrocardiogram was taken, the significance of this finding is questionable.

The relatively high incidence of delayed onset of the intrinsicoid deflection in this study may be due to: (a) The high incidence of the cases used with advanced left ventricular hypertrophy, (b) the inclusion of cases with electrocardiograms whose total QRS duration was above .10 seconds, thus including in the study possible intraventricular conduction defects. Sokolow and Lyon (2) considered .12 seconds as being the upper limit of QRS duration which indicated left ventricular hypertrophy and ruled out intraventricular conduction defects. In left ventricular hypertrophy, this wide QRS is associated with an absence of slurring of the QRS complex and a ventricular activation time of less than .08 seconds.

If this criteria is used, all of these cases could be considered as possible left ventricularhypertrophy.

There are certain exceptions to the maximum normal values for QRS voltage used in this study. For instance, Shack (35), reported maximum normal value for RaVL as 12 mm. Kossman and Johnston (9) reported amplitudes of RV5 up to 33.0 mm in normal people.

Another complicating factor is the fact that the maximum normal voltage of RV5 for adults is influenced by age. (36) Thus, the amplitude of RV5 under the age of 40 can be as high as 28.4 mm., from the age of 50 to 60 years it can normally be only as high as 17.0 mm. Heine (36) believes that since this maximum normal amplitude of RV5 decreases with age, the criteria of high voltage in the precordial leads should be interpreted more conservatively in younger patients and more liberally in older patients. This also invalidates the criteria used in this study for RV5 in children.

Another variable in QRS voltage is the electrical conductivity of the tissues and the distance of the electrodes from the heart. Diminished voltage is seen in emphysema and obesity and higher voltage is seen in thin chested individuals. The influence of these factors was not considered in this series.

Proper technique in recording the electrocardiogram is essential for accurate data. Factors which enter into this variable are standardization, damping and electrode placement. If the precordial electrode for V5 is placed too far medially, the amplitude of the R will be higher than if placed more laterally.

Intraventricular conduction was delayed in 30% of the patients. Two of the cases were excluded from the analysis of the QRS because of the presence of right bundle branch block in one and Wolff Parkinson White syndrome in the other. Both of these conditions was associated with prolonged QRS complexes. None of the cases analysed had QRS durations greater than .12 seconds, which according to Sokolow, (2) is the upper limit of normal for left ventricularhypertrophy.

Q waves in leads facing the left ventricle are common in left ventricular hypertrophy. However, their diagnostic value is questionable since they are found in these same leads in about 33% of the normal people with left axis deviation. Rarely does the Q wave exceed 3.0 mm. in depth or 25% of the Q/R ratio in simple left ventricular hypertrophy. One case (number 7) had an anterior wall infarct in the healed stage, which was diagnosed microscopically at autopsy. The Q waves in the electrocardiogram of this patient in lead aVL and V5 were probably a result of the fibrotic area in the heart muscle, which was residual of this infarct.

It will be noted from Table IX that all the hearts analyzed in this series had at least one of the abnormal criteria present which was studied in this series. Vertical hearts showed the least number of abnormalities, that is, three of them had only one abnormality, one had three abnormalities present.

None of the hearts showed all of the characteristics of left ventricular hypertrophy which were studied in this paper. There seemed to be no correlation between the size of the heart or the left ventricular thickness and the number of abnormalities exibited in the electrocardiograph.

Table IX

Case	Left ventricle thickness	Weight in grams	Voltage	Position	RST-T segment
1.	20 mm	500	abnormal	horizontal	abnormal
2.	15 mm	400	normal	vertical	abnormal
3.	15 mm	450	normal	horizontal	abnormal
4.	17 mm	440	abnormal	horizontal	abnormal
5.	18 mm	350	normal	horizontal	normal
6.	21 mm	440	abnormal	horizontal	abnormal
7.	16 mm	400	normal	horizontal	abnormal
8.	20 mm	400	normal	vertical	normal
9.	27 mm	700	abnormal	vertical	abnormal
10.	25 mm	550	abnormal	vertical	normal
11.	16 mm	420	normal	horizontal	abnormal
12.	20 mm	560	normal	horizontal	abnormal

* Considered normal when all leads analyzed were with in those limits considered normal in this study, if one or more of the leads showed changes beyond these normal limits, the case was considered abnormal.

SUMMARY OF THE CONDITIONS FOUND IN THIS STUDY, WITH HEART WEIGHTS.

Levine (23) states that, in his experience, left ventricular hypertrophy is invariably found at autopsy when it has been diagnosed electrocardiographically. Since only hearts of known hypertrophic size were used in this series, this statement could not be analyzed in this series.

Intrinsicoid deflection	Intraventricular conduction	Q wave	S wave in lead I	Case
abnormal	abnormal	normal	present	1.
normal	normal	normal	absent	2.
abnormal	abnormal	abnormal	absent	3.
normal	normal	normal	present	4.
abnormal	abnormal	normal	present	5.
abnormal	abnormal	normal	absent	6.
abnormal	abnormal	abnormal	absent	7.
normal	normal	normal	absent	8.
abnormal	normal	normal	present	9.
normal	normal	normal	present	10.
normal	normal	normal	absent	11.
normal	normal	abnormal	absent	12.

SUMMARY AND CONCLUSIONS:

1. The electrocardiographic criteria of left ventricular hypertrophy used by some of the present day authorities were evaluated and compared with the electrocardiograms of 12 hearts which proved to be hypertrophied at autopsy.

2. The hearts used varied in weight from 350 grams to 700 grams, left ventricular thickness varied from 15 mm. to 27 millimeters.

3. The characteristic electrocardiographic changes in the precordial, standard and unipolar leads found in these cases of left ventricular hypertrophy studied, in their order of frequency are:

   (a) Abnormal RST-T segment in 75% of the cases. This consists of a depressed ST-T segment with upward bowing, inverted and asymetric T waves. Depression of the RST with the upward bowing was more commonly encountered than inversion and asymetry of the T wave, being found in 75% and 33% of the cases, respectively.

   (b) Absent S wave in lead I in 58% of the cases.

   (c) Delayed intrinsic deflection in V5 of greater than .05 seconds in 42% of the cases.

   (d) Delayed intraventricular conduction, making the QRS complex greater than .10 seconds but less than .12 seconds found in 30% of the cases.

   (e) Abnormalities in voltage of the QRS complex in which the R wave in V5 exceeds 26 mm. and RaVL exceeds 11 millimeters were encountered in 25% of the cases each.

4. Left ventricular hypertrophy is more often associated with the horizontal electrocardiographic position (67%) than with the vertical (33%).

5. The same changes noted in V5 usually appear in aVL, if the heart is horizontal and in aVF if the heart is vertical.

6. The voltage of R in V5 and in aVL is helpful in the diagnosis of left ventricular hypertrophy. Each of these leads showed higher than normal voltage in 25% of the cases.

7. Delay in the intrinsic deflection in V5 is shown to be an important feature of left ventricular hypertrophy. This was greater than normal (.05 seconds) in 42% of the cases. Delay in intrinsic deflection in V1 of longer than .03 seconds is less important in the diagnosis of left ventricular hypertrophy. It occured in only one case.

8. The value of RST-T abnormalities could not be evaluated because of the inclusion in this series of four patients who were taking digitalis. This, however, was the most frequent abnormality encountered in this series. (75%)

9. Although RST-T abnormalities and left axis deviation are not specific diagnostic criteria, both can be considered as being confirmatory of left ventricular hypertrophy and often become of diagnostic importance in this way.

10. Q waves in leads facing the left ventricle are common in left ventricular hypertrophy but their diagnostic significance is questionable. Rarely does the Q wave exceed 3.0 millimeters in depth or 25% of the succeding R wave in simple left ventricular hypertrophy.

11. Absence of S waves in lead I is common in left ventricular

hypertrophy, but is quite non-specific. It may be valuable as confirmatory evidence if it is associated wit h the more specific diagnostic criteria of left ventricular hypertrophy, however.

12. Vertical hearts show less abnormalities than horizontal hearts in left ventricular hypertrophy if these criteria are used.

13. No definite correlation could be made between the post mortum weight of the heart and the left ventricular thickness and the type or number of electrocardiographic abnormalities encountered in the electrocardiogram.

14. Customary autopsy measurments of heart weight and size are not precise enough to determine specific chamber wall hypertrophy or accurately deliniate the normal from the hyper-trophied heart.

15. Left ventricular hypertrophy is a difficult diagnosis to make from electrocardiographic findings. This is evidenced by the fact that only 25% of these patients were diagnosed as having left ventricular hypertrophy by electrocardiographic findings anti-mortum.

16. The electrocardiographic criteria used today as evidence of left ventricular hypertrophy were found to be variable and inconsistantly present in this series of cases.

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