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Original Communications

THE VENTRICULAR COMPLEX IN LEFT VENTRICULAR HYPERTROPHY AS OBTAINED BY UNIPOLAR PRECORDIAL AND LIMB LEADS

MAURICE SOKOLOW, M.D., AND THOMAS P. LYON, M.D.
SAN FRANCISCO, CALIF.

THE electrocardiographic patterns of left ventricular hypertrophy or left ventricular strain in standard limb lead records have received considerable attention from investigators.[1-10] Less detailed attention has been paid to the pattern of left ventricular hypertrophy when using the unipolar limb and precordial leads. The typical pattern obtained by unipolar techniques has been described previously, [11-15] but the atypical ones and those showing lesser degrees of abnormalities have not been detailed adequately. The practical importance of the electrocardiographic position of the heart and of the time of onset of the intrinsic deflection of the ventricular complexes as an aid in diagnosis has been emphasized already.[11,16] The purpose of the present investigation is to evaluate the criteria, using unipolar limb and precordial leads, for the recognition of the atypical and early patterns of left ventricular hypertrophy; to determine the frequency of the characteristic changes noted by Wilson and his associates[11]; and to study the diagnostic value of the electrocardiographic position of the heart and of the time of onset of the intrinsic deflection (ventricular activation time).

METHODS AND SUBJECTS

Two hundred patients were selected whose electrocardiograms were abnormal and in whom a cardiac disorder capable of producing increased strain on the left

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TABLE I. THE VENTRICULAR DEFLECTIONS IN UNIPOLAR LIMB AND PRECORDIAL LEADS (MEASUREMENTS IN MILLIMETERS)

LEAD	LEFT VENTRICULAR HYPERTROPHY (147 CASES)				NORMAL (151 CASES)				NORMAL—LEFT AXIS DEVIATION (21 CASES)			
	MEAN	±ST. DEV.	MIN.	MAX.	MEAN	±ST. DEV.	MIN.	MAX.	MEAN	±ST. DEV.	MIN.	MAX.
V_1												
Q	0	0	(0)		0	0	(0)		0	0	(0)	
R	1.1	1.3	0	12.0	2.3	1.5	0	7.0	1.8	1.3	0	4.5
S	13.1	5.5	3.0	35.0	8.6	4.3	2.0	25.0	8.0	3.7	3.5	18.0
T	1.5	1.84	−2.5	+5.5	0.15	1.58	−4.0	+4.0	0.4	1.32	2.5	+3.0
ID*	0.013	0.008	0	0.03	0.020	0.007	0	0.03	0.02	0.006	0	0.02
V_2												
Q	0.01	0.05	0	1.0	0	0	0		0	0	0	
R	4.0	3.4	0	18.0	5.9	3.1	0	16.0	5.0	2.9	2.0	12.0
S	15.4	6.5	0	46.0	12.7	5.3	0	29.0	10.3	4.8	4.0	18.0
T	4.86	2.75	−0.5	+13.0	5.52	3.32	−3.0	+18.0	4.08	2.68	−2.5	+9.0
ID	0.01	0.008	0	0.03	0.025	0.006	0	0.04	0.03	0.006	0.01	0.035
V_3												
Q	0.04	0.27	0	2.0	0.01	0.06	0	0.5	0	0	0	
R	9.3	6.4	0	26.0	8.9	4.3	1.5	26.0	8.6	4.0	4.0	17.0
S	10.8	6.7	0	36.0	8.8	5.3	0	25.0	7.5	4.3	3.0	16.0
T	3.53	3.27	−5.0	+11.0	5.38	2.96	−2.0	+16.0	4.67	4.37	−0.1	+13.0
ID	0.03	0.01	0	0.07	0.03	0.007	0.02	0.04	0.03	0.005	0.02	0.04
V_4												
Q	0.15	0.39	0	2.0	0.1	0.4	0	3.0	0.05	0.16	0	0.5
R	16.8	8.2	0.5	42.0	14.2	5.5	4.0	27.0	12.5	5.0	5.0	27.0
S	6.3	5.3	0	30.0	5.2	4.0	0	20.0	4.3	3.2	0	13.0
T	1.63	3.44	−6.0	+11.0	4.8	2.76	0	+17.0	4.2	2.67	−0.1	+13.0
ID	0.041	0.009	0	0.07	0.034	0.007	0.02	0.05	0.04	0.007	0.03	0.045

V_1												
Q	0.4	0.74	0	5.0	0.3	0.6	0	3.0	0.3	0.4	0	1.0
R	18.9	7.5	5.0	48.0	12.1	4.4	4.0	26.0	11.8	5.4	6.0	26.0
S	1.5	2.3	0	13.0	1.5	1.5	0	6.0	1.2	1.1	0	4.0
T	−0.28	2.93	−9.0	+7.0	3.43	1.62	0	+9.0	3.45	1.66	+1.0	+9.0
ID	0.048	0.009	0.03	0.07	0.04	0.01	0	0.05	0.04	0.007	0.025	0.05
V_6												
Q	0.45	0.76	0	5.0	0.4	0.5	0	2.0	0.4	0.4	0	1.0
R	16.4	8.9	6.0	58.0	9.2	3.6	4.0	22.0	8.9	4.1	4.0	19.0
S	0.43	1.06	0	10.5	0.6	1.0	0	7.0	0.2	0.5	0	1.5
T	−0.63	2.53	−9.0	+4.5	2.43	1.11	−0.5	+5.0	2.4	1.18	+0.5	+6.0
ID	0.049	0.009	0.03	0.08	0.04	0.01	0.02	0.05	0.04	0.01	0.03	0.05
aV_L												
Q	0.37	0.68	0	4.0	0.2	0.5	0	3.5	0.5	0.4	0	4.0
R	8.1	4.8	0	25.0	2.1	2.1	0	10.0	4.6	2.5	0.5	10.0
S	0.65	1.36	0	9.0	0.4	3.9	0	18.0	0.3	0.7	0	3.0
T	−0.51	1.37	−7.0	+2.5	0.53	1.26	−4.0	+6.0	1.0	0.81	−1.0	+2.5
aV_R												
Q	0.78	2.16	0	10.0	2.0	3.7	0	8.0	1.0	2.6	0	6.0
R	0.57	1.25	0	4.0	0.8	0.9	0	5.0	0.4	0.6	0	1.5
S	8.6	4.8	0	20.0	4.3	4.0	0	13.0	4.3	0.9	0	8.0
T	−0.36	1.42	−3.0	+5.0	−2.31	0.92	−5.0	+1.5	−2.2	0.67	−3.0	+1.0
aV_F												
Q	0.24	0.53	0	4.0	0.5	1.4	0	3.0	0.4	1.0	0	5.0
R	4.7	4.0	0	21.0	1.3	8.3	0	20.0	2.7	2.2	0	8.0
S	2.3	2.9	0	10.0	0.2	1.3	0	8.0	1.6	1.4	0	4.0
T	0.52	1.46	−4.0	+10.0	1.86	1.1	−0.5	+5.0	1.4	0.92	−0.5	+3.0

*ID = time of onset of intrinsic deflection from the beginning of the QRS complex.

ventricle (such as hypertension, aortic valvular lesions, coarctation of the aorta, patent ductus arteriosus) was present. Fifty-three patients were excluded from this study because they had received digitalis or quinidine, had angina pectoris or known coronary disease, or because their electrocardiograms exhibited bundle branch block or Q waves consistent with the possibility of myocardial infarction. One hundred forty-seven patients remained of whom 90 per cent had hypertension exceeding 155/95, with a mean blood pressure of 197/117, and a mean increase in the transverse diameter of the heart[20] of 15.8 per cent. Electrocardiographic studies of the patients in this group were made as will be described in detail later. As controls, 151 normal subjects, whose histories, physical examinations, electrocardiograms, and roentgenograms of the chest gave negative results, were studied similarly. This control group consisted of healthy nurses, medical students, members of the house staff, and flying personnel of a commercial airline. The mean age of the normal subjects was 35.1 years.

The standard limb leads were obtained first in each case. The augmented unipolar limb leads (aV_L, left arm; aV_R, right arm; and aV_F, left leg) and the unipolar precordial leads (V_1 through V_6) were then obtained by the method of Goldberger[17] in his modification of Wilson's central terminal. In addition, seven-foot chest films were taken in all but fourteen cases.

The electrocardiographic abnormalities considered to be particularly significant included the criteria previously noted in the literature,[5-11,18,19] as well as the variations in the unipolar leads to be described.

The electrocardiograms were analyzed in tabular form on master sheets, all waves of each record being carefully measured (with a magnifying lens if necessary). The amplitude of upright waves was measured from the upper edge of the base line to the peak of the wave; that of inverted waves, from the lower edge. Calibration corrections were applied, if necessary, for standardization (1.0 cm. = 1.0 mv.). Particular attention was paid to the voltage of the R and S waves in the precordial and unipolar extremity leads in order to calculate the ratios to be described. Gubner and Ungerleider[10] have emphasized the diagnostic importance of high voltage of the QRS complex in the standard limb leads in left ventricular hypertrophy. The data to be presented will aid in establishing the importance of high voltage in the precordial and extremity leads which had not been evaluated by the authors just mentioned. In addition, data on the total left ventricular potentials (the sum of the amplitudes of the R wave in Lead V_5 and the S wave in Lead V_1) of normal subjects were compared with those of patients with left ventricular hypertrophy.

RESULTS

Table I summarizes the statistical data obtained in the cases of left ventricular hypertrophy, in the entire normal group as well as in the subjects with left axis deviation included in the normal group. Table II summarizes the criteria obtained from a study of our data for the diagnosis of left ventricular hypertrophy when the changes occur in the presence of hypertension or a cardiac lesion putting strain on the left ventricle. Table III summarizes the frequency with which the various electrocardiographic abnormalities were encountered.

TABLE II. THE CRITERIA FOR THE DIAGNOSIS OF LEFT VENTRICULAR HYPERTROPHY

1. Standard limb leads
   (a) Voltage $R_1 + S_3 = 25$ mm. or more.
   (b) RS-T_1 depressed 0.5 mm. or more.
   (c) T_1 flat, diphasic, or inverted, particularly when associated with (b) and a prominent R wave.
   (d) T_2 and T_3 diphasic or inverted in the presence of tall R waves and (b).
   (e) T_3 greater than T_1 in the presence of left axis deviation and high voltage QRS complex in Leads I and III.
2. Precordial leads
   (a) Voltage of R wave in V_5 or V_6 exceeds 26 millimeters.
   (b) RS-T segment depressed more than 0.5 mm. in V_4, V_5, or V_6.
   (c) A flat, diphasic, or inverted T wave in Leads V_4 through V_6 with normal R and small S waves and (b).
   (d) Ventricular activation time in V_5 or V_6 = 0.06 second or more, especially when associated with a tall R wave.
3. Unipolar limb leads
   (a) RS-T segment depressed more than 0.5 mm. in aV_L or aV_F.
   (b) Flat, diphasic, or inverted T wave, with an R wave of 6.0 mm. or more in aV_L or aV_F and (a).
   (c) Voltage of R wave in aV_L exceeds 11.0 millimeters.
   (d) Upright T wave in aV_R.

TABLE III. THE FREQUENCY OF ABNORMALITIES IN UNIPOLAR LIMB AND PRECORDIAL LEADS IN LEFT VENTRICULAR HYPERTROPHY

Total number of cases		147
Total number of cases with normal or borderline standard leads		34
Abnormal RST-T findings		136
Lead I	69	
Lead II and/or III	20	
Leads V_4 through V_6	111	
Lead aV_L	88	
Lead aV_R	38	
Lead aV_F	40	
Abnormal voltage		67
$R_1 + S_3 = 25$ mm. or more	26	
R in V_5 or R in $V_6 = 26$ mm. or more	29	
R in $V_L = 11$ mm. or more	33	
R in V_5 + S in $V_1 = 35$ mm. or more	48	
Delayed onset of the intrinsic deflection (delayed ventricular activation time) 0.06 second or more in V_5 or V_6.		52

The cases of left ventricular hypertrophy were divided into three groups, depending on the size of the heart as determined by the method of Ungerleider and Clark.[20,21] In the group whose cardiac size fell within the normal range (± 10 per cent of the expected), it was found in some that the contour of the left border of the heart suggested left ventricular hypertrophy. For purposes of

uniformity these cases were classified in the group with no cardiac enlargement. No definite association was observed between cardiac size and electrocardiographic abnormalities. In many instances, significant electrocardiographic findings were noted in the absence of cardiac enlargement. The reverse was seen less frequently.

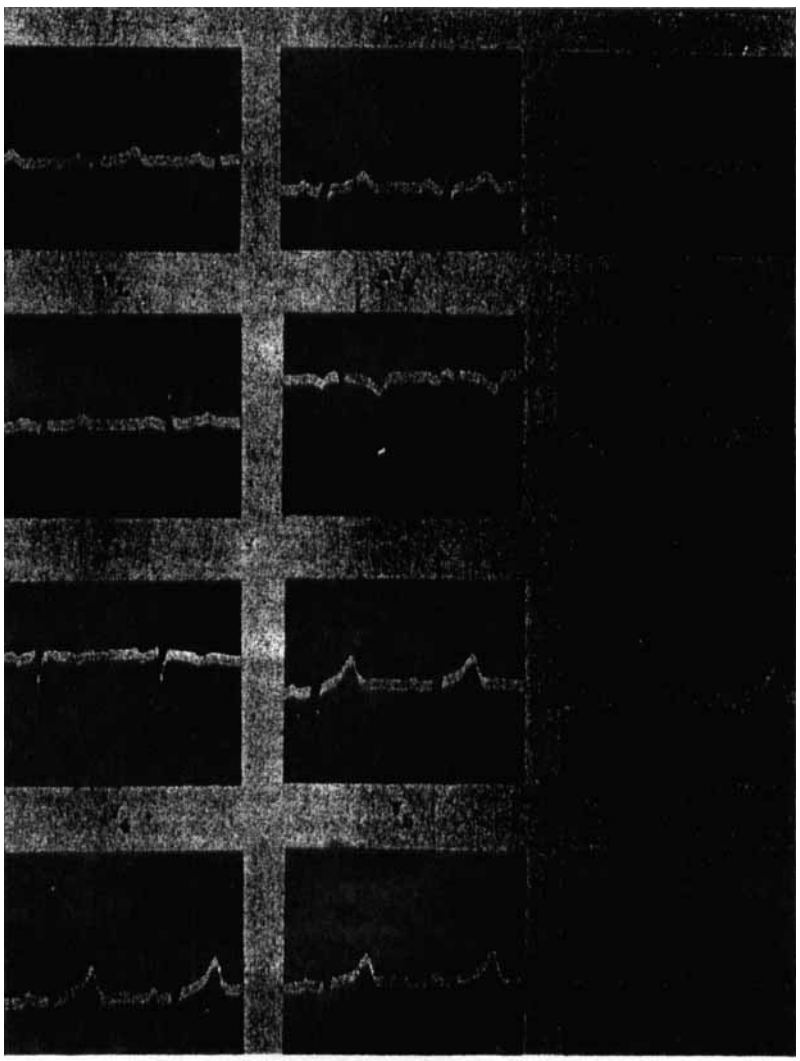

Fig. 1.—H. H., a 58-year-old man, U78776. Normal subject with horizontal position of the heart and left axis deviation (− 20°).

Position of the Heart.—The electrocardiographic position of the heart was determined in each case (Table IV). It will be seen that the majority of the patients had horizontal or semihorizontal hearts. Because of the horizontal position of the heart, left axis deviation was present. It will be shown later that

the electrocardiographic patterns of patients with horizontal hearts are those described in the literature as typical of left ventricular hypertrophy.[6,9] Some of the cases classified as intermediate in position may in fact have been semihorizontal because the major abnormalities in these cases were seen in Lead aV_L. Although the R wave was significantly upright in both Leads aV_L and aV_F, the contribution of the left ventricle was often seen to a greater degree in the changes in the left arm lead.

An attempt was made to differentiate the electrocardiographic findings in normal horizontal hearts with left axis deviation from the electrocardiographic abnormalities found in hearts with left ventricular hypertrophy and left axis deviation. Table I reveals that the findings in the electrocardiograms of normal subjects with left axis deviation do not differ significantly from the findings in the entire normal control group. No RST-T abnormalities or abnormal voltage of the R or S waves occurred in the standard, unipolar limb, and precordial leads in normal horizontal hearts (Fig. 1), although it is perhaps possible theoretically for very marked counterclockwise rotation to cause an inverted T wave in aV_L.[29] Furthermore, the time of onset of the intrinsic deflection was not found to be greater than 0.05 second in the normal individual.

TABLE IV. THE ELECTROCARDIOGRAPHIC POSITION OF THE HEART AS OBTAINED IN 147 CASES OF LEFT VENTRICULAR HYPERTROPHY

Horizontal	31
Semihorizontal	52
Intermediate	30
Semivertical	26
Vertical	6
Indeterminate	2
	147

The RST-T Pattern of Left Ventricular Hypertrophy in Horizontal Hearts.— The importance of abnormalities of the RS-T segment and T wave in the recognition of left ventricular hypertrophy is clearly seen from Table III, these changes being the most frequent of all the abnormal findings.

The patterns of left ventricular hypertrophy seen in horizontal and semihorizontal hearts (Figs. 2 through 5) show the "typical" changes in the standard leads because the position of the heart is horizontal. The left ventricular potentials (V_5 and V_6) are directed toward the left arm (aV_L) which in turn result in changes in Lead I. Hence, the typical RST-T variations of left ventricular hypertrophy in horizontal hearts may be seen in Leads I, aV_L, and V_4 through V_6. The precordial leads were usually the first to become abnormal, but occasionally the left arm lead revealed flat or inverted T waves when the RST-T changes in the precordial leads were borderline (Fig. 4).[10,11] This was true even though precordial leads were taken in the seven positions in both the third and fifth intercostal spaces. The abnormalities seen in Lead aV_L were usually more marked than those noted in Lead I but, as a rule, were less striking than those found in Leads V_5 and V_6.

Depending on the stage of evolution of the electrocardiographic pattern, the RST-T changes were minimal (Lead I in Fig. 3), moderate (Lead aV_L in Fig. 2), or marked (Lead V_5 in Fig. 5). In the well-developed pattern, the typical RST-T relationship, as previously emphasized by Rykert and Hepburn[5]

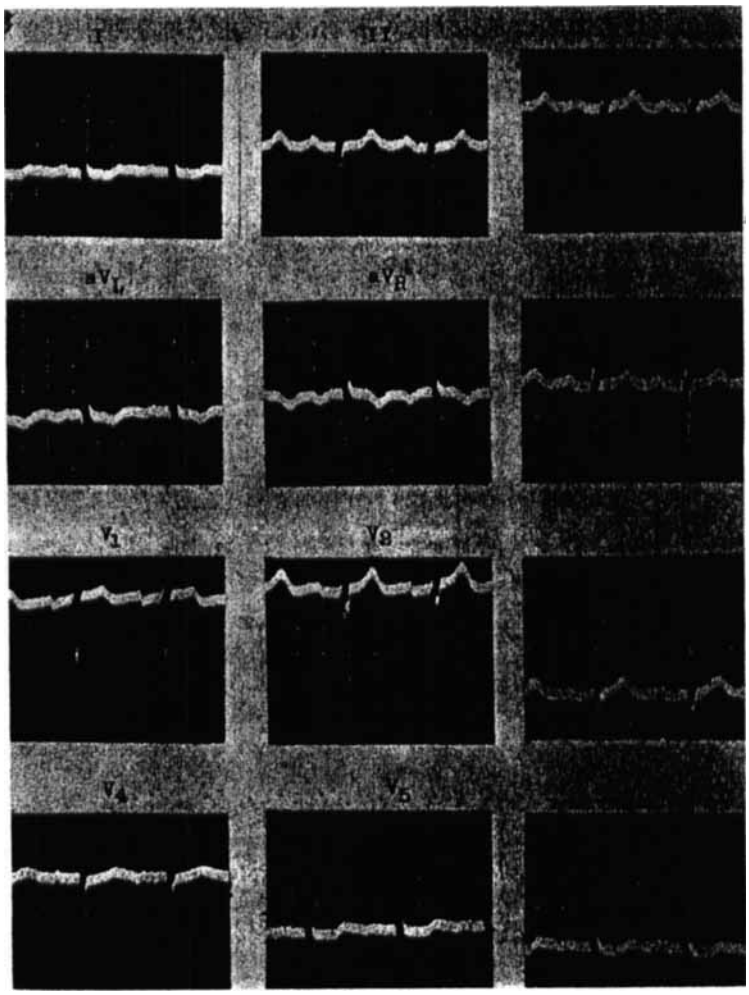

Fig. 2.—E. G., a 72-year-old woman, U132714. Hypertension. Left ventricular hypertrophy in a horizontal heart. Note the RST-T abnormalities in Leads I, aV_L, V_5, and V_6. In addition, the intrinsic deflection occurs in 0.06 second in V_6, the voltage of the R wave in aV_L equals 14 mm., and $R_1 + S_3$ equals 28 mm.

and by Kaplan and Katz,[9] was clearly seen in the left precordial leads and, depending on the position of the heart, these same abnormalities appeared in the left arm or the left leg lead. This contour was significant and was characterized in its typical form by an RS-T segment that was depressed and bowed upward and by a T wave that was inverted and asymmetric (V_5 in Fig. 5). The RS-T segment and T wave were both directed downward, in contrast to the usual appearance in coronary disease where the RS-T and T are in opposite directions

if the RS-T segment is elevated or depressed. In the early developing stages of left ventricular hypertrophy, the RST-T relationship was less clear. Often the T wave decreased in size before the changes in the RS-T segment appeared or slight depression of the RS-T segment was associated with a lowered T wave;

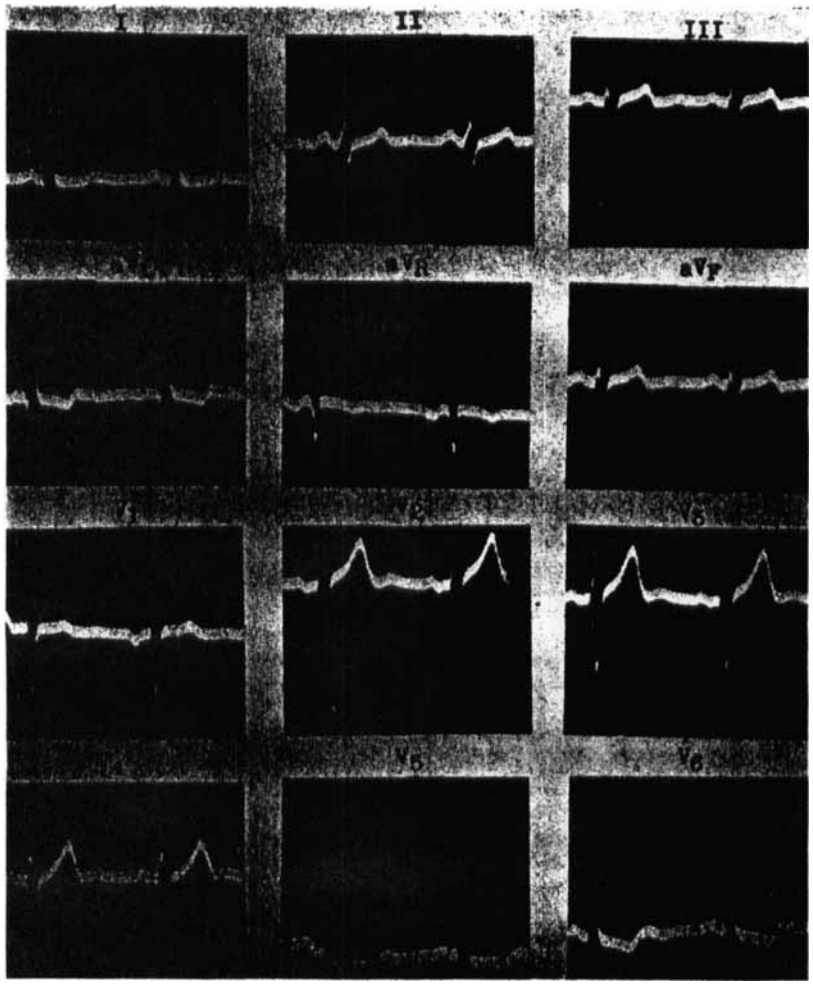

Fig. 3.—J. R., a 69-year-old man, U133478. Hypertension. Left ventricular hypertrophy in a horizontal heart. Note the characteristically abnormal RST-T contour in V_6 and aV_L with the early RST-T contour in Lead I. The voltage of $R_I + S_3$, of R in aV_L, and of R in $V_5 + S$ in V_1 are also abnormal.

in these cases the RS-T segment and T wave were not always in the same direction early in the disease (Lead I in Figs. 2 and 3). Some patients have the characteristic RST-T relationship of left ventricular hypertrophy in one lead (V_6 in Fig. 3) with the early relationship in another (Lead I in Fig. 3).

Low T waves were frequently observed in the left precordial leads in the patients with left ventricular hypertrophy, although no record was classified as abnormal on the basis of low T waves. To quantitate this finding, the ratio

of the amplitude of R to T was calculated in all the patients with upright T waves (those with flat, diphasic, or inverted T waves were excluded). Table IX summarizes the data obtained. The results were significant in that the *mean* R/T ratio in Leads V_5 and V_6 in the cases of left ventricular hypertrophy exceeded the *maximum* R/T ratio in these leads found in normal subjects. Fifty per cent of the patients with left ventricular hypertrophy with upright T waves

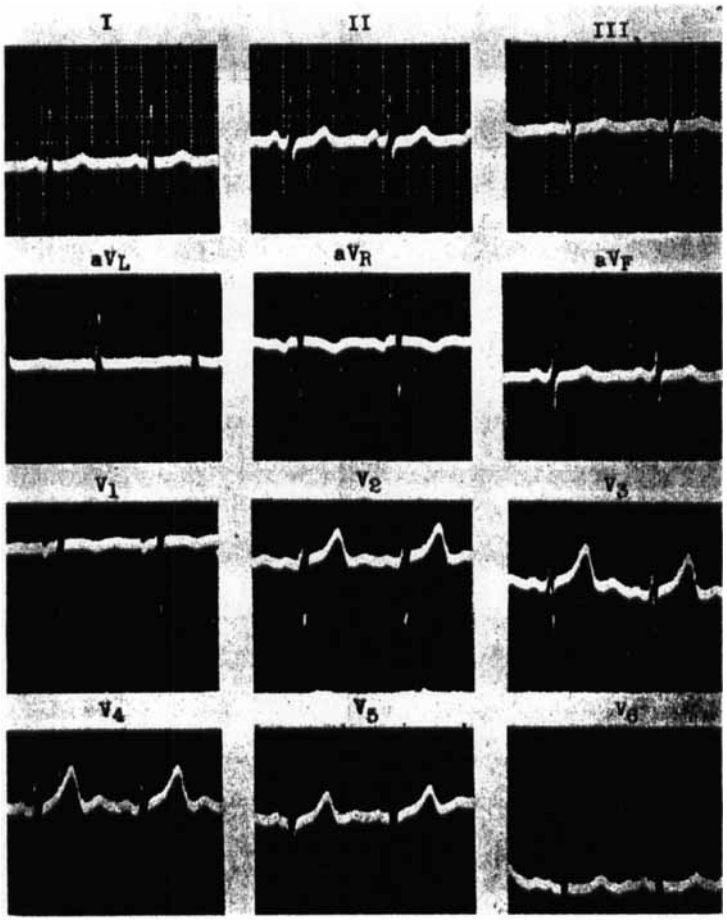

Fig. 4.—A. D., a 36-year-old woman. Hypertension of eight years' duration. Cardiac enlargement +25 per cent. Standard leads normal except that the ratio T_3/T_1 is 1 in presence of left axis deviation. A flat T wave in aV_L with an R wave of 9 mm. is the only abnormality except that the combined voltage of R in V_6 and S in V_1 is 44 millimeters. This record represents the earliest findings in left ventricular hypertrophy in a semihorizontal heart.

in V_5 and/or V_6 had an R/T ratio in these leads equalling or exceeding the maximum normal ratio of 10. It was of interest to note that the R wave was absent in Lead V_3 in only six of the 147 patients with left ventricular hypertrophy and in none in Leads V_4, V_5, or V_6.

The RST-T Pattern of Left Ventricular Hypertrophy in Electrocardiographically Vertical Hearts.—The electrocardiographic patterns of left ventricular hyper-

trophy in a vertical or semivertical heart (Figs. 6 and 7) illustrate that individuals with this type of hypertrophy do not have the changes in axis or in RST-T that have long been considered "typical" in the standard limb leads.[4-10] When the heart is electrocardiographically vertical the potential changes of the left ventricle are directed toward the left leg so that Leads II, III, and aV_F, but not Lead I, reveal the RST-T abnormalities originating in the left ventricle. Furthermore, the electrical axis may be directed toward the right. The precordial Leads V_5

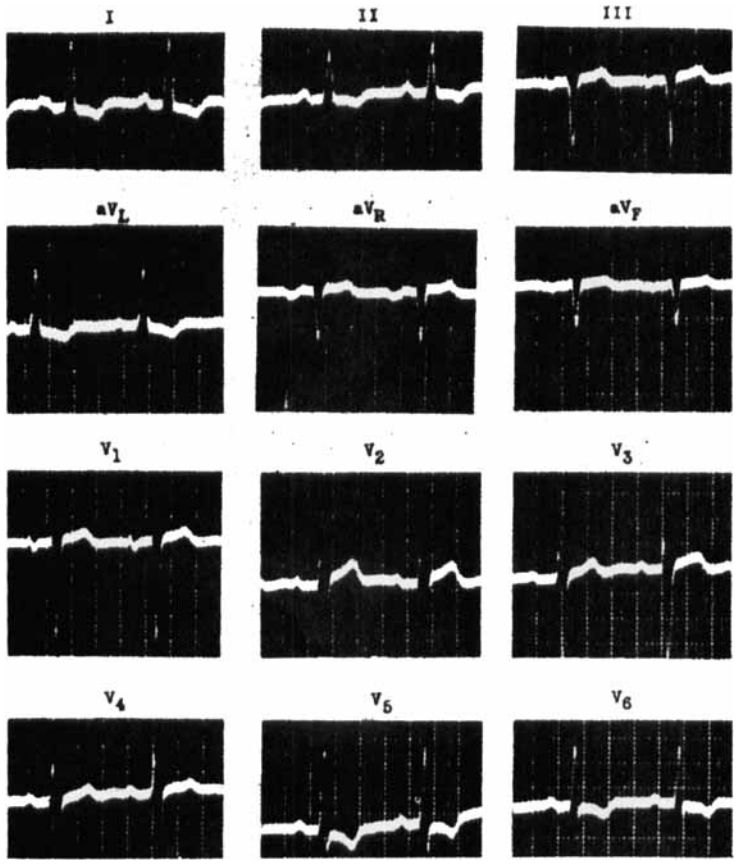

Fig. 5.—C. B., a 62-year-old woman. Hypertension. Typical left ventricular hypertrophy in a horizontal heart with the classic RST-T contour in Leads I, II, aV_L, V_5, and V_6. Note the upright T wave in aV_R. The voltage of $R_I + S_3$ and of the R wave in aV_L is just beyond the critical level. The onset of the intrinsic deflection is not delayed.

and V_6 show the same type of abnormalities in left ventricular hypertrophy, whether the heart is vertically or horizontally placed (Figs. 2 through 9). Variations in the standard leads in these cases merely reflect the electrocardiographic position of the heart, and the position determines whether the potential changes of V_5 and V_6 (representing the left ventricle) are transmitted to the left leg (and hence to Leads II and III), or to the left arm (and hence to Lead I).

Lead aV_F was at times of definite diagnostic value when left ventricular hypertrophy occurred in a vertical heart, as illustrated in Fig. 8. In this particular case the changes in Leads II and III were not diagnostic and the only abnormalities in V_5 and V_6 were slight depression of the RS-T segment. Possibly Leads V_7 or V_8 would have shown greater abnormalities if the transitional zone had been displaced farther to the left. Nevertheless, with the usual records, the significant abnormalities were seen mainly in Lead aV_F. This particular record

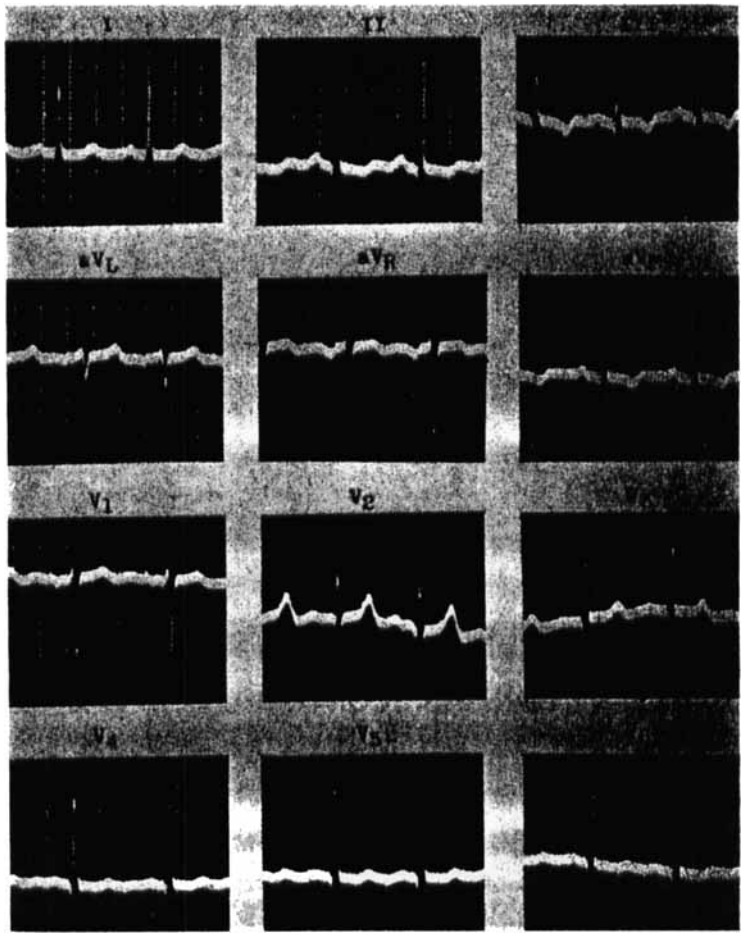

Fig. 6.—T. M., a 39-year-old woman, U136786. Hypertension. Left ventricular hypertrophy in a semivertical heart. Note the RST-T abnormalities in Leads II, III, and aV_F, and to a lesser extent, in V_4 through V_6.

was interpreted as representing left ventricular hypertrophy when the daily records were being routinely read; no history of the patient was available. When the patient was seen later, it was found he had hypertension.

Intrinsic Deflection.—Wilson and his associates[11] emphasized the importance of the time of appearance of the intrinsic deflection in the recognition of ventricular hypertrophy and bundle branch block. They stated that the time from the onset

of the QRS to the peak of the R wave or the beginning of the abrupt downstroke of the QRS represents the time interval required for the passage of the impulse through the ventricle to the epicardium underlying the exploring electrode. This time interval will be referred to in this paper as the "ventricular activation

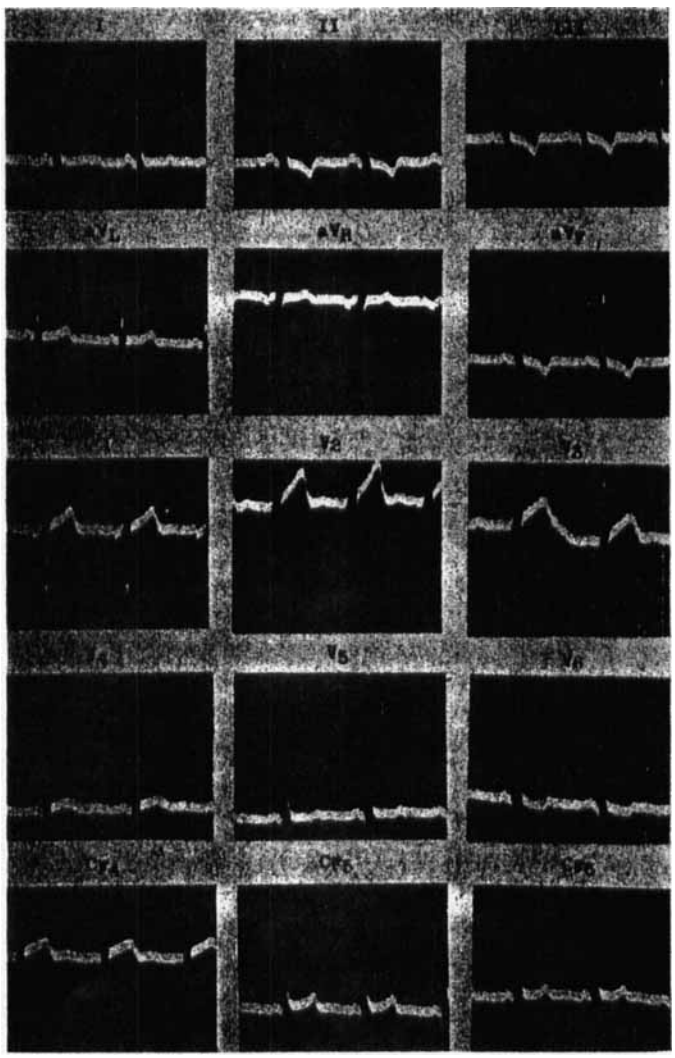

Fig. 7.—D. H., a 26-year-old man, U125144. Coarctation of the aorta. Cardiac enlargement, 20 per cent. Typical left ventricular hypertrophy in a vertical heart with abnormalities in Leads II, III, aVF, V_5, and V_6.

time." An increased mass of myocardium, as present in left ventricular hypertrophy, would be expected to delay this time interval required for the passage of the impulse to the epicardium. In three different series of normal subjects, comprising 280 cases,[16,22,23] the onset of the intrinsic deflection (ventricular activation time) in Leads V_5 or V_6 was less than 0.06 second. Kossmann and

Johnston[22] stated that the time of onset of the intrinsic deflection in the normal individual averages 0.02 second in Lead V_1 and 0.04 second in V_5. Sodi-Pallares and his associates[16] in their study of 100 normal subjects found the maximum ventricular activation time in V_5 to be 0.05 second. In the present control series of normal subjects (Table I) 0.03 second in V_1 and 0.05 second in V_5 or V_6 was the upper limit of normal found.[23] Sixteen (10 per cent) of our normal subjects had a figure of 0.05 second in Leads V_5 or V_6.

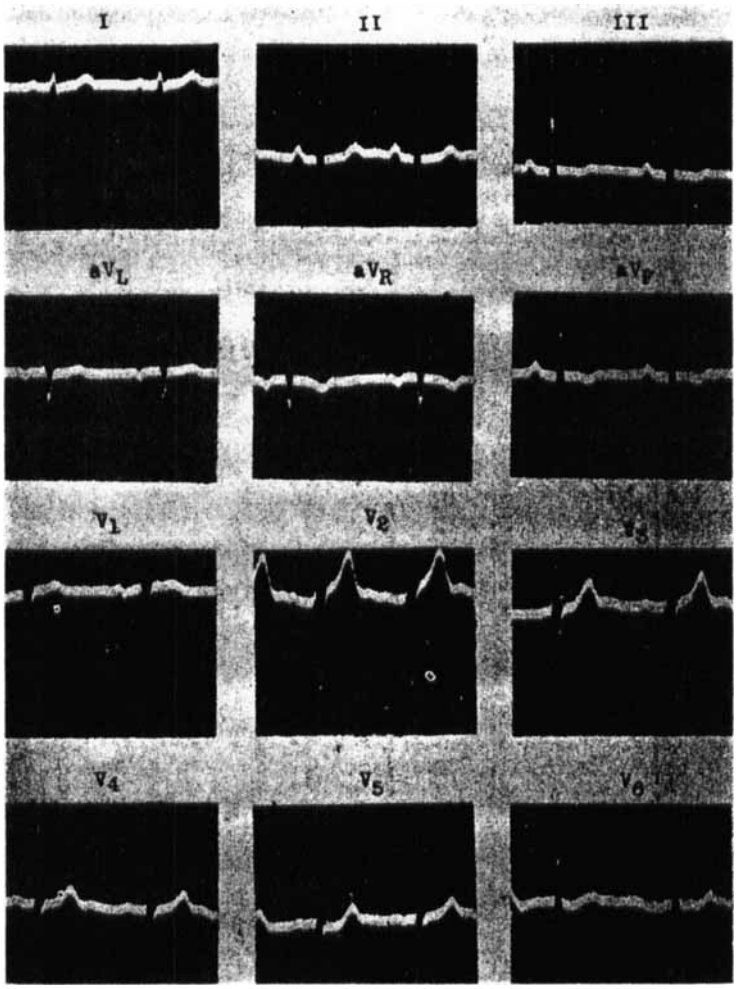

Fig. 8.—E. V. Y., a 29-year-old man. Hypertension. Left ventricular hypertrophy in a vertical heart. The major abnormality is present in Lead aV_F with minor RS-T segment depression in V_5 and V_6. The standard and unipolar precordial leads alone would not have been sufficient for a proper diagnosis.

In the present series of 147 cases of left ventricular hypertrophy, 85 (58 per cent) had a ventricular activation time* in V_5 or V_6 of 0.05 to 0.08 second.

*The term "ventricular activation time" refers to the time in seconds from the beginning of the QRS complex to the onset of the intrinsic deflection.

In fifty-two cases (35 per cent), the left ventricular activation time was 0.06 second or greater (but less than 0.08 second). The mean ventricular activation time in V_5 or V_6 was 0.05 second in contrast to 0.04 second in the normal group. In 40 per cent of the cases of left ventricular hypertrophy, the time of onset of the intrinsic deflection was normal even though other typical findings, such as a

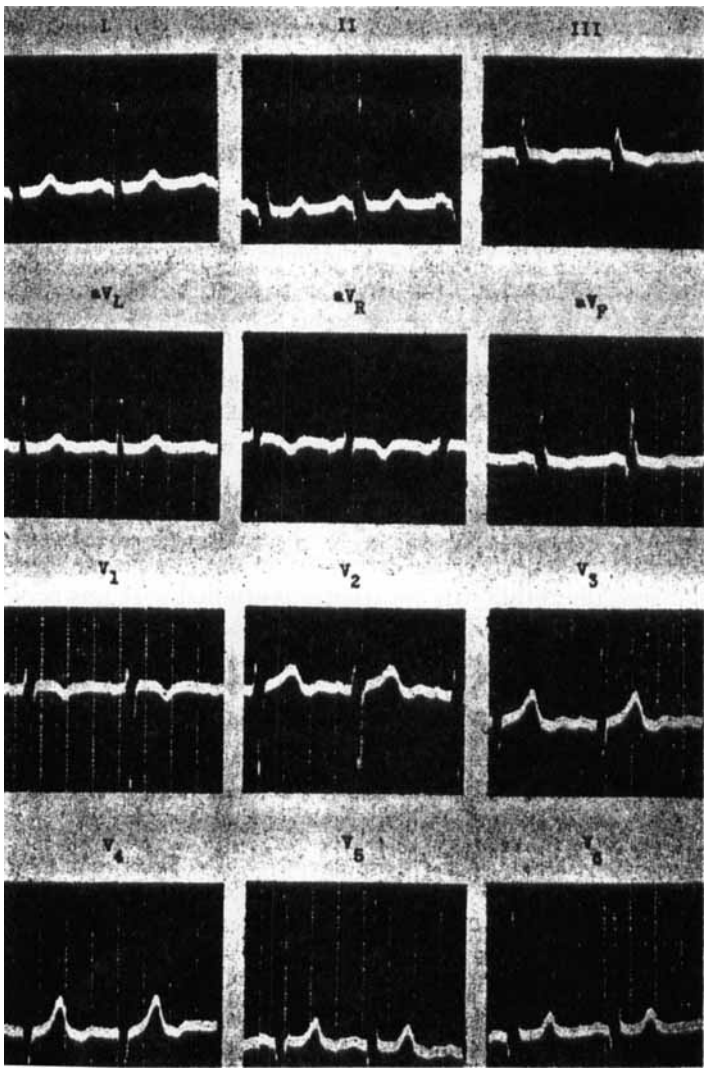

Fig. 9.—M. C., a 39-year-old woman, U128549. Hypertension. The voltage of R in V_5 and S in V_1 equals 49 millimeters. The ventricular activation time equals 0.06 second in V_5 and V_6. Early RST-T abnormalities are seen in Lead aV_F.

depressed R-ST segment and inverted T waves, were present (Fig. 5). In some patients, however, with long-standing hypertension and roentgenologic evidence of left ventricular enlargement, a delayed intrinsic deflection in Lead V_5 or V_6 was the only abnormal electrocardiographic sign. Is one justified in diagnosing

left ventricular hypertrophy solely by the presence of an intrinsic deflection that is delayed in V_5 or V_6? No normal individual in the three available series (total of 280 cases)[16,22,23] had a ventricular activation time of as much as 0.06 second. The majority of patients with left bundle branch block (Wilson's criteria[11]) had a ventricular activation time between 0.08 and 0.14 second; none was found to be shorter.[26] In contrast, approximately 50 per cent in Sodi-Pallares' series[16] and fifty-two of 147 (35 per cent) in the present series of left ventricular hypertrophy had a ventricular activation time of 0.06 second or longer. It must be concluded that such a finding, even as an isolated abnormality, should weigh heavily in favor of a diagnosis of left ventricular hypertrophy, especially if, in addition to a delayed intrinsic deflection, the R wave is tall and the T wave is relatively low in the same lead.

It was considered of interest to note the association between size of the heart (as obtained from the transverse diameter of the heart and the table of Ungerleider and Clark[20]) and the ventricular activation time. Thirteen (25 per cent) of the fifty-two patients in the series of 147 who had a ventricular activation time of 0.06 second had no cardiac enlargement. Of the ninety-five persons in whom this time interval was less than 0.06 second, thirty (31 per cent) had no cardiac enlargement. Thus, no association between a delayed intrinsic deflection and transverse cardiac diameter could be demonstrated. Cardiac hypertrophy may be present, however, without roentgenologic evidence of cardiac enlargement.

Intraventricular Conduction.—The relationship between the ventricular activation time and the total QRS duration was studied. It has been previously shown that in left ventricular hypertrophy the total QRS duration may exceed 0.10 second, often being 0.12 second without the electrocardiographic pattern of left bundle branch block being present.[11] This was confirmed by a study of the patients in this series in whom the QRS duration was 0.11 or 0.12 second. This duration was present in eighteen (12 per cent) of the 147 cases under discussion. In typical left bundle branch block, the left ventricular activation time almost always exceeded 0.08 second, whereas it very rarely reached this figure in left ventricular hypertrophy. Furthermore, in left ventricular hypertrophy with a QRS duration of 0.12 second and a delayed intrinsic deflection of 0.06 or 0.07 second, the peak of R in the ventricular complex usually was found to be tall and sharp. In left bundle branch block, the peak of R in Leads V_5 or V_6 is broad topped, notched, or "M" shaped, reflecting the delay in the spread of the impulse through the left ventricle. There was no constant relationship between the time of onset of the intrinsic deflection in Lead V_5 and the total QRS duration. Some patients had a normal activation time with a QRS of 0.12 second, while others had a delayed intrinsic deflection with a QRS of 0.10 second. The significance of these findings is not yet clear.

Voltage.—The importance of the voltage of the QRS complex was adequately demonstrated in the present series (Table III). In some cases the increased voltage was seen months or years before unequivocal RST-T changes occurred and as such was a valuable early diagnostic finding (Figs. 10 and 11).

The voltage of R and S in the unipolar leads can be seen in Table I. The mean height of the R wave in V_5 in left ventricular hypertrophy was 18.9 mm., and in normal subjects with horizontal hearts 11.8 mm. with a standard deviation of ± 5.4 millimeters. The maximum amplitude of R in V_5 in normal subjects was found to be 26 millimeters. This amplitude was exceeded in twenty-nine cases (20 per cent) of left ventricular hypertrophy. The amplitudes of the waves in the augmented unipolar extremity leads used in this study are 50 per cent greater than those obtained in the unipolar limb leads according to the

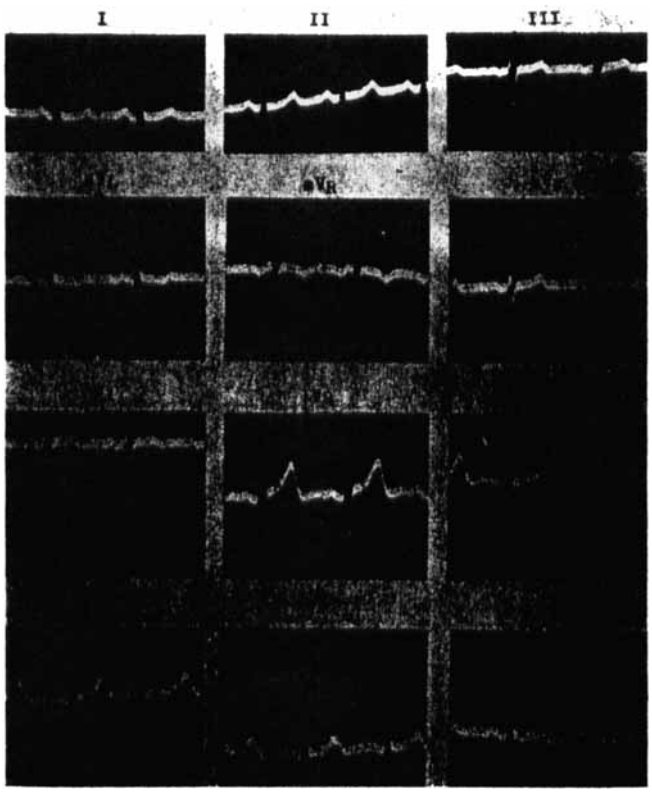

Fig. 10.—C. S., a 45-year-old woman, US6287. Hypertension. Sept. 6, 1946. The major abnormalities are seen in aV_L with high voltage of R (11.5 mm.) and early RST-T changes. The voltage of the R wave in Lead I is high, 16 millimeters.

method of Wilson. Also of value was the voltage of R in aV_L. The amplitude of this wave in normal horizontal hearts was found to be 4.6 mm. with a standard deviation of ± 2.5 mm., in contrast to the cases of left ventricular hypertrophy in which the corresponding figure was found to be 8.1 ± 4.8. In normal horizontal hearts, 99 per cent of the subjects may be expected to have an R wave in aV_L of less than 11.1 millimeters. In normal subjects the maximum R in aV_L was found to be 10.5 millimeters. This voltage was exceeded in thirty-three cases (22 per cent) of left ventricular hypertrophy. The diagnostic value of the

voltage of the R wave in Lead aV_F was less because the normal height of this wave is often great (up to 20 mm.).

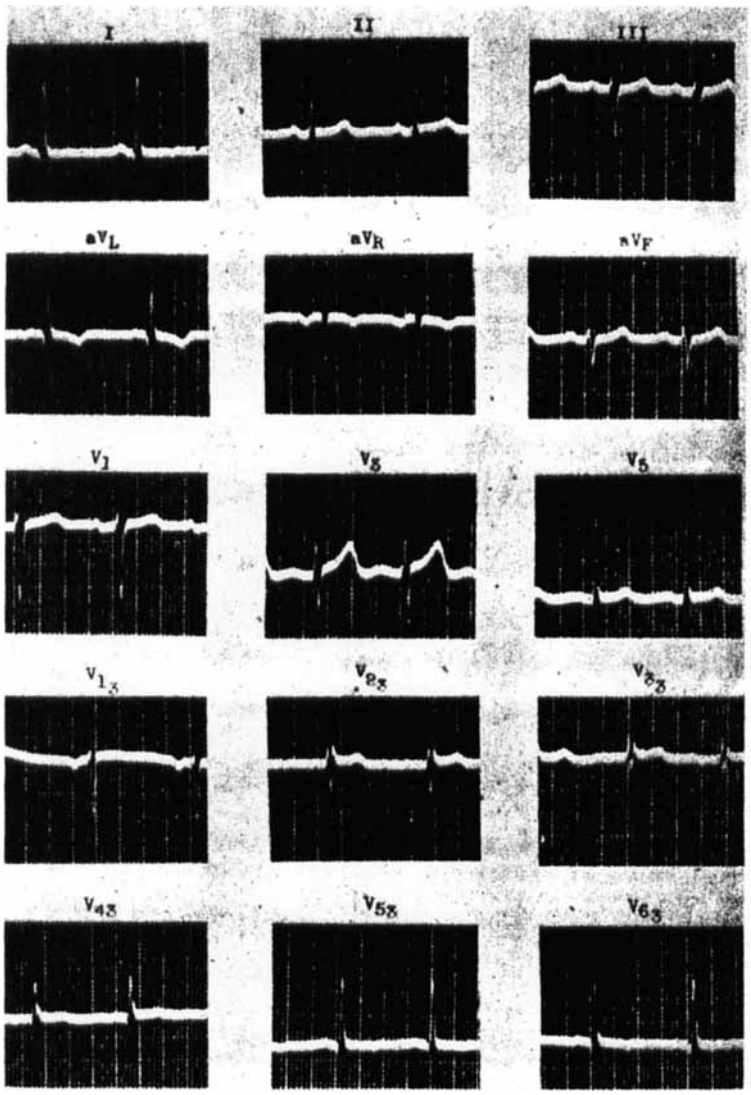

Fig. 11. Same patient as in Fig. 10. June 6, 1947. Progressive abnormalities have appeared in aV_L and now Lead I and also Lead V_5 recorded in the third intercostal space are abnormal. Note that V_5 recorded in the third intercostal space is more abnormal than the conventional V_5 recorded in the fifth intercostal space.

In addition to the absolute value of the height of the R wave, the relationship of the R to the S wave in Leads V_1 and V_5 was found to be quite different in the group with left ventricular hypertrophy as compared with the normal individuals (Table V). The differences between the two groups were more strikingly evident when the ratio of $\frac{R/S \text{ in } V_5}{R/S \text{ in } V_1}$ was determined (Table VI). Of the

forty-two cases of left ventricular hypertrophy in which this ratio could be determined (indicating an R wave in V_1 and an S wave in V_5), fourteen cases (33 per cent) exceeded the maximum normal ratio of 100.

TABLE V. THE R/S RATIO IN THE UNIPOLAR PRECORDIAL LEADS OF CASES OF LEFT VENTRICULAR HYPERTROPHY AS COMPARED WITH NORMAL SUBJECTS

	NORMAL			LEFT VENTRICULAR HYPERTROPHY		
	MEAN	ST. DEV.	RANGE	MEAN	ST. DEV.	RANGE
R/S ratio						
V_1	0.3	0.3	(0-1.0)	0.08	0.11	(0-0.6)
V_2	0.2	1.2	(0.1-13)	0.65	2.05	(0-20)
V_3	1.4	1.4	(0.1-10)	2.6	6.1	(0-56)
V_4	4.1	3.8	(0.2-19)	5.8	6.6	(0.2-38)
V_5	7.3	4.7	(1.0-24)	10.7	9.2	(1.6-50)
V_6	9.0	5.0	(2.3-22)	15.7	11.2	(4.2-38)

TABLE VI. THE RESULTS OF THE R/S RATIO IN V_5 DIVIDED BY THE R/S RATIO IN V_1 IN NORMAL SUBJECTS AND THOSE WITH LEFT VENTRICULAR HYPERTROPHY

	NORMAL	LEFT VENTRICULAR HYPERTROPHY
R/S in V_5		
R/S in V_1		
Mean	32.0	98
Standard deviation	26.9	91.5
Range	3.1–100	13–400

The sum of the total left ventricular potentials (S wave in V_1 plus R wave in V_5 or R wave in V_6) proved to be of definite diagnostic importance (Table VII). The mean sum in the normal subjects was 19.9 ± 5.6 mm.; in only six (4 per cent) of 150 normal individuals did the sum exceed 30 and none exceeded 35 millimeters. This is in contrast to the findings obtained in the 147 patients with left ventricular hypertrophy in seventy-two (49 per cent) of whom the sum of the S wave in V_1 and the R wave in V_5 or V_6 exceeded 30 mm. and in forty-eight (32 per cent) of whom this sum exceeded 35 millimeters. In approximately one-third, therefore, of the cases of left ventricular hypertrophy the sum of R in V_5 and

TABLE VII. THE SUM OF THE AMPLITUDES (IN MILLIMETERS) OF THE R WAVE IN V_5 AND THE S WAVE IN V_1 IN NORMAL SUBJECTS AND SUBJECTS WITH LEFT VENTRICULAR HYPERTROPHY

R IN V_5 + S IN V_1	NORMAL	LEFT VENTRICULAR HYPERTROPHY
Mean	19.9	30
Standard deviation	± 5.6	± 10.4
Range	0–35	12–65

S in V_1 exceeded the maximum sum obtained in normal subjects. In some cases, abnormal voltage of R in V_5 or V_6 and S in V_1 and minor T-wave changes were the only abnormalities (Fig. 4). Re-evaluation of some of the electrocardiograms of patients with hypertension not included in the present study because the records were interpreted as normal revealed a number that would have been classified as abnormal if the data on voltage here presented had been used. The single measurement of the sum of the R waves in V_5 or V_6 (whichever is larger) and the S wave in V_1 is an apparently reliable criterion of left ventricular hypertrophy (Table III).*

The variety of data on abnormally high voltage proved helpful because the high voltage was seen in different combinations in different cases. Fig. 2 illustrates high voltage of the R wave in aV_L (14 mm.) and high voltage in the sum of $R_1 + S_3$ (31 mm.); the sum of S in V_1 and R in V_5 is within normal limits (31 mm.). In Fig. 4 the voltage of $R_1 + S_3$ (21 mm.) and of R in aV_L (10 mm.) is within the normal range, yet S in V_1 + R in V_6 is definitely abnormal (45 mm.). In Fig. 3 all three measurements of voltage are high.

Q Waves.—The evaluation of the Q waves in the unipolar precordial and limb leads is no less difficult than in the standard limb leads. The criteria suggested for abnormality of the Q waves vary widely.[24,25,26] A Q wave 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, a Q wave may normally be seen in aV_L as well as in Leads V_4 through V_6; in vertical hearts, Q waves can be found in aV_F and in Leads V_4 through V_6. Q waves were commonly seen in our cases of left ventricular hypertrophy, being found in approximately one-third of the cases in the left precordial leads and in Lead aV_L (Table VIII). Rarely did the Q waves exceed

TABLE VIII. THE Q/R RATIO IN PATIENTS WITH LEFT VENTRICULAR HYPERTROPHY AS COMPARED WITH SUBJECTS WITH NORMAL HEARTS

LEAD	NORMAL					LEFT VENTRICULAR HYPERTROPHY				
	NO.	MEAN	ST. DEV.	MIN.	MAX.	NO.	MEAN	ST. DEV.	MIN.	MAX.
V_1	0	0	0	0		0	0	0	0	
V_2	0	0	0	0		4	0.06	0.05	0	0.13
V_3	2	0.025	0.002	0	0.03	5	0.072	0.059	0	0.14
V_4	16	0.04	0.032	0	0.1	21	0.043	0.002	0	0.1
V_5	49	0.07	0.039	0	0.16	48	0.055	0.038	0	0.23
V_6	65	0.087	0.043	0	0.21	51	0.072	0.045	0	0.25
aV_L	20	0.238	0.165	0	0.75	43	0.147	0.178	0	1.0
aV_R	43	4.97	2.96	0	14.0	17	6.02	4.75	0	20.0
aV_F	58	0.1	0.06	0	0.28	38	0.15	0.14	0	0.77

*A recent patient followed to autopsy illustrates the diagnostic value of the voltage of the precordial leads. A 16-year-old boy with coarctation of the aorta had a normal-sized heart by x-ray study. The electrocardiogram was entirely normal except that the sum of the R wave in V_5 and the S wave in V_2 equalled 50 millimeters. The S wave in V_2 was 36 millimeters (the maximum obtained in our normal subjects was 29 mm.). At autopsy the left ventricular wall measured 2.0 cm. in thickness. There was no coronary disease or myocardial fibrosis. The heart weighed 300 grams.

3.0 mm. in depth in left ventricular hypertrophy, and when the Q waves were of that depth, the R waves in the corresponding lead were tall. The maximum Q/R ratio in the left precordial leads in left ventricular hypertrophy was found to be 25 per cent (Table VIII).

The interpretation of Q waves, especially in Lead aV_L in semivertical hearts, is extremely difficult. When the Q is wide (0.04 second), when it represents 50 per cent of the QRS complex, and when it is followed by a convex elevated RS-T segment and a late inversion of T, a lateral myocardial infarction should be suspected.[27] Unipolar leads made in the second and third intercostal spaces should be taken if the routine precordial leads are not diagnostic in order to recognize a high anterior lesion. However, in some cases, no further electrocardiographic support for myocardial infarction can be elicited by exploratory precordial leads. In the normal subjects of our series in whom Q waves in aV_L represented 50 per cent of the R wave, the total QRS complex was small (less than 5.0 mm.).

TABLE IX. THE RATIO OF THE AMPLITUDES OF THE R AND T WAVES (R/T RATIO) IN PATIENTS WITH LEFT VENTRICULAR HYPERTROPHY AS COMPARED WITH SUBJECTS WITH NORMAL HEARTS. THE RATIO IS CALCULATED ONLY WHEN THE T WAVE IS UPRIGHT; FLAT, DIPHASIC, OR INVERTED T WAVES ARE EXCLUDED

LEAD	NORMAL (151 CASES)				LEFT VENTRICULAR HYPERTROPHY (147 CASES)			
	NO.	MEAN	ST. DEV.	RANGE	NO.	MEAN	ST. DEV.	RANGE
V_1	59	1.4	0.9	(0.3–7)	68	1.0	1.0	(0.1–4)
V_2	145	1.4	1.4	(0.2–12)	123	1.3	1.4	(0.1–7)
V_3	150	1.9	1.6	(0.3–13)	114	3.0	3.2	(0.1–20)
V_4	150	3.1	2.3	(0.3–9)	99	6.1	3.9	(0.6–23)
V_5	151	3.5	1.6	(1.0–9)	68	10.9	7.4	(2.7–44)
V_6	151	4.1	1.9	(1.7–10)	60	11.1	6.6	(2.7–34)
V_L	91	2.6	1.9	(0.1–10)	40	8.1	7.4	(0.3–28)
V_F	142	4.6	3.2	(0.3–14)	81	4.4	4.5	(0.3–28)
V_R		0	0	0		0	0	0

ASSOCIATED CORONARY DISEASE AND MYOCARDIAL INFARCTION

Coronary arteriosclerosis is commonly found at autopsy in patients with hypertension and left ventricular hypertrophy. It is not surprising, therefore, that changes typical of myocardial infarction or coronary insufficiency frequently coexist with signs of left ventricular hypertrophy. The factors of left ventricular hypertrophy and coronary insufficiency cannot be adequately separated when the RS-T contour and T-wave changes are characteristic of coronary insufficiency.[8] Probably myocardial infarction can be diagnosed in conjunction with left ventricular hypertrophy when the Q, RS-T segment, and T-wave changes typical of myocardial infarction are present concurrently with signs of left ventricular hypertrophy, such as high voltage and typical RST-T changes. One of the major values of unipolar precordial and limb leads is their ability to

uncover an unsuspected myocardial infarction (usually anteroseptal and old) when the standard limb leads are either normal or the abnormalities are nonspecific. Many instances of myocardial infarction are clinically occult, and unipolar studies in patients in whom myocardial infarction is common (for example, patients with hypertension) will allow unsuspected myocardial infarction to be recognized occasionally. Despite the fact that myocardial infarction clinically may not be typical, it is rarely completely silent. In a recent study of thirty patients with hypertension in whom a previously unsuspected diagnosis of myocardial infarction was made from the unipolar leads, practically all had some episode in the past history compatible with the diagnosis.[27] Sudden weakness, sunstroke, sudden cardiac failure, hemiplegia, pulmonary embolism, and sudden paroxysmal nocturnal dyspnea were frequently noted in the relatively recent past history of these patients. Therefore, unipolar leads are valuable not only in delineating the characteristic features of left ventricular hypertrophy, but in excluding or establishing the presence of associated coronary insufficiency or myocardial infarction.

DISCUSSION

The electrocardiographic diagnosis of left ventricular hypertrophy depends upon proper evaluation of two particular problems in so far as the standard limb leads are concerned: (1) the differentiation of "normal" from "abnormal" left axis deviation; (2) the interpretation of RST-T changes (especially in Leads II and III) in the absence of left axis deviation and in the absence of abnormal RST-T changes in Lead I. As far as the first problem is concerned, the diagnosis is fairly simple when the typical RST-T changes of left ventricular hypertrophy occur in association with high voltage QRS waves and left axis deviation. When the RS-T segment and T wave in Lead I are essentially normal and the voltage of R_1 and S_3 is not abnormal, the problem is more difficult. It is then necessary to study the precordial and unipolar extremity leads in order to differentiate transverse position of the heart or counterclockwise rotation of the heart on its longitudinal axis[28] from left ventricular hypertrophy. Left axis deviation and the associated transverse position of the heart in young people should immediately arouse suspicion of abnormality, especially if the individual is of average build. Horizontal or semihorizontal hearts with left axis deviation were infrequently found in normal subjects under the age of 40 years in the absence of obesity or conditions, such as pregnancy, that elevate the diaphragm.[23] Many persons who were overweight did not have horizontal hearts as might be expected.[23] In older and stout individuals, left axis deviation and transverse hearts were more commonly seen without left ventricular hypertrophy, and additional substantiation was required from the precordial and unipolar limb leads in order to establish a diagnosis. These leads were of particular value because it was not uncommon for typically abnormal RST-T signs to be found in Leads V_5 or V_6 or aV_L when they were absent or not characteristic in Lead I (Fig. 3). At times, because of unknown factors, possibly rotation, the abnormal left ventricular potentials may be transmitted to the left arm, and Lead aV_L

may be more abnormal than V_5 or V_6 (Figs. 10 and 11); this, however, was unusual. Because Lead I reflects the difference in potential between the left and right arms, an abnormal T wave in the left arm may be neutralized by the potential of the right arm, and hence Lead I may be normal and Lead aV_L abnormal. The deviation of the T wave to the right, with T_3 becoming equal to or greater than T_1 in the presence of a horizontal or semihorizontal heart, reflects the abnormality of the T wave in aV_L. Such progressive "rightward deviation" of the T wave in the standard leads occurs coincidentally with progressive lowering of the T wave in aV_L in cases of hypertension with horizontal hearts (Figs. 10 and 11) and we have found the observations of Proger and Minnich[18] and of Ashman and Hidden[19] to be of value. Even when both Lead I and Lead aV_L are abnormal, the degree of abnormality is usually greater in the left arm lead (Fig. 11). Therefore, the unipolar left arm lead (aV_L), as well as the unipolar precordial leads (especially V_5 and V_6), are very helpful in evaluating the significance of left axis deviation in the standard leads in the presence of normal RS-T segments and T waves.

In the interpretation of RST-T changes in the standard leads in the absence of left axis deviation, or in the presence of right axis deviation, unipolar precordial and extremity leads are even more valuable (Fig. 7). As already noted, the typical pattern of left ventricular hypertrophy with left axis deviation and abnormal RST-T in Lead I occurs in individuals with horizontal hearts (Fig. 5). Because of the transmission of the left ventricular potential to the left leg in persons with vertical hearts, the characteristic RST-T changes of left ventricular hypertrophy will occur in the left leg lead and in standard Leads II and III (Fig. 7). The precordial leads, however, in left ventricular hypertrophy in vertical hearts are similar to those obtained in left ventricular hypertrophy in horizontal hearts. The dissimilarity in the standard leads in vertical and horizontal hearts (the absence of left axis deviation and the presence of the major abnormalities in Leads II and III in the former) merely reflects the position of the heart. An appreciation of this fact will allow the ready recognition of the RST-T changes of left ventricular hypertrophy, no matter in what lead they occur. Recourse to the precordial leads will resolve the diagnostic dilemma, especially when right axis deviation is present. Since right axis deviation with inversion of T_2 and T_3 may occur in both right and left ventricular hypertrophy, a study of the precordial leads may reveal the characteristic changes in voltage of the QRS, in the RST-T waves, and in the ventricular activation time in the left precordial leads if left ventricular hypertrophy is present. The typical findings of right ventricular hypertrophy will be noted in Leads V_1 and V_2 if the abnormalities in the standard leads are due to right ventricular hypertrophy.

The data presented on voltage of the QRS complexes in the precordial leads should prove helpful in providing supportive evidence of left ventricular hypertrophy in the early stages of the developing pattern. This is especially true in patients in whom the heart is not horizontal (Fig. 9). The data on voltage provided by Gubner and Ungerleider[10] will be adequate in most cases of left ventricular hypertrophy with horizontal heart. In these patients the voltage

is often equally abnormal in both standard and precordial leads, although exceptions in both directions may occur; the various data on voltage are complementary. In hearts that are not horizontal, even in semihorizontal hearts, we have found abnormal voltage of the QRS in the unipolar left arm lead and/or in the precordial leads (S in V_1 + R in V_5) and yet the voltage in the standard leads (R_1 + S_3) was not abnormal. In these cases, the additional information offered by the data on voltage presented here was of definite diagnostic value.*

SUMMARY AND CONCLUSIONS

1. A statistical study is presented of the unipolar precordial and augmented limb leads in 147 cases of left ventricular hypertrophy.

2. The patterns of left ventricular hypertrophy are described with particular attention to the early abnormalities found (depressed RS-T segment with flat or low diphasic T waves, abnormally high voltage of the QRS complex, and delayed onset of the intrinsic deflection).

3. The characteristic and diagnostic changes in the precordial leads found in the cases of left ventricular hypertrophy studied include, in order of frequency:

(a) A depressed RS-T segment and asymmetric inversion of the T wave in Lead V_5 or V_6. In early cases, the T wave may be low and diphasic or flat in association with depression of the RS-T segment.

(b) Abnormalities in voltage of the QRS complex in which the R wave in V_5 or V_6 exceeds 26 mm. and/or the sum of the R wave in V_5 and the S wave in V_1 exceeds 35 mm.

(c) The onset of the intrinsic deflection (the ventricular activation time) exceeds 0.05 second in Lead V_5 or V_6.

4. The same characteristics noted in V_5 and V_6 often appear in aV_L in horizontal hearts and in aV_F in vertical hearts. The changes in these unipolar extremity leads usually are less striking but occasionally may be more abnormal than the changes in the precordial leads.

5. Abnormalities in the left arm lead (aV_L) usually are reflected in Lead I, and the pattern of left ventricular hypertrophy in the standard leads described as "typical" in the literature occurs in individuals with horizontal hearts. The abnormalities seen in Lead aV_L usually are more striking than those found in Lead I.

6. Abnormalities in the left leg lead (aV_F) usually are reflected in Leads II and III, but to a lesser degree. Individuals with abnormalities in these leads have been shown to have vertical hearts and the standard leads will disclose no axis deviation or right axis deviation, and the pattern described in the literature as "atypical" will appear.

7. The diagnostic significance of the voltage of the left ventricular potentials as reflected by the sum of the R wave in V_5 or V_6 and the S wave in V_1 is empha-

*Since this paper was submitted for publication, twenty-two patients in whom the diagnosis of left ventricular hypertrophy was made by the electrocardiographic criteria here presented have been examined at autopsy. In twenty patients the left ventricular thickness equalled or exceeded 1.5 cm. and in the remaining two patients the heart weights were 500 grams and 420 grams respectively. The left ventricular thickness in these two patients was 1.2 centimeters.

sized. Thirty-two per cent of patients with left ventricular hypertrophy had the sum of these two potentials exceed 35 mm., whereas in no normal person did this sum exceed 35 mm.; in 96 per cent of normal individuals the sum was found to be below 30 millimeters.

8. The voltage of the R wave in V_5 and in aV_L was helpful in the diagnosis of left ventricular hypertrophy. In 20 per cent of the patients with left ventricular hypertrophy, the voltage of R exceeded 26 mm. in V_5 and in 22 per cent this voltage exceeded 10 mm. in aV_L the maximum values found in normal subjects according to our data.

9. The importance of the time of onset of the intrinsic deflection (ventricular activation time) is discussed. In 35 per cent of patients with left ventricular hypertrophy the ventricular activation time was 0.06 second or more, in contrast to the fact that this delayed time was not encountered in any of 150 normal subjects.

10. In the evaluation of left axis deviation and RST-T abnormalities in the standard limb leads, unipolar extremity and precordial leads are confirmatory and often of critical diagnostic importance.

11. Horizontal or semihorizontal hearts were found in eighty-three (56 per cent) while vertical or semivertical hearts were found in thirty-two (22 per cent) of the patients with left ventricular hypertrophy in this series.

12. Low T waves were frequently noted in Leads V_5 and V_6 in association with tall R waves, resulting in a high R/T ratio. Fifty per cent of the patients in this series had an R/T ratio in V_5 and/or V_6 exceeding the maximum ratio of 10 found in the normal subjects.

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