Before the advent of noninvasive methods capable of accurately estimating the dimensions, mass, and wall motion of cardiac chambers, electrocardiography in association with chest radiography and physical examination played a prominent role in assessing the size of the cardiac chambers. The electrocardiographic theory underlying the recognition of hypertrophy or dilation rests on a number of sound physical principles that may lead to meaningful correlations with the tissue mass, chamber diameter, and intracardiac blood volume. There are, however, unavoidable limiting factors related to the variable orientation of the heart in the chest, variable properties of the volume conductor surrounding the heart, and nonspecificity of each depolarization and repolarization abnormality used for diagnosing hypertrophy or dilation.

As early as the 1950s and 1960s a wealth of autopsy and angiographic correlations firmly established the limits of the diagnostic accuracy of the electrocardiogram (ECG). With regard to left ventricular hypertrophy (LVH), it has been established that the ECG diagnosis is most accurate in patients with hypertensive and pure left-sided valvular heart disease in the absence of concomitant right ventricular hypertrophy (RVH), myocardial infarction, intraventricular conduction disturbances, and treatment with drugs altering depolarization and repolarization. Similar limiting factors play a role in the ECG diagnosis of RVH.

**Left Ventricular Enlargement**

*(Hypertrophy and Dilation)*

- Voltage
- Intrinsicsoid Deflection
- Repolarization Abnormalities
- Other ECG Changes in LVH
- Physiologic Factors Affecting the Reliability of Diagnostic Criteria
- Pathologic States Affecting the Diagnosis of LVH

**Assessment of the Severity of Valvular Lesions Causing LVH**

- Presence of LBBB

**Right Ventricular Hypertrophy and Dilation**

- Three Types of RVH
- RVH in Other Clinical Conditions

**Combined Ventricular Hypertrophy**

- Clinical and Anatomic Correlations

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**Voltage**

The increased voltage is attributed to one or more of the following factors: increased left ventricular mass, increased left ventricular surface, increased intracavitary blood volume, and close proximity of the enlarged ventricle to the chest wall. In general, ECG criteria for the diagnosis of left ventricular enlargement are increased QRS amplitude (voltage), intraventricular conduction delay manifested by delayed intrinsicsoid deflection in the precordial leads facing the left ventricle, widened QRS/T angle, and a tendency to left axis deviation.

**Left Ventricular Mass**

The increase in the left ventricular mass exaggerates the leftward and posterior QRS forces. An increase in voltage may be due to the increase in the number or size of the fibers in the hypertrophied ventricle. The importance of fiber size was shown in the computer-generated ECG model where the amplitude of the R wave increased by 36 percent when the cell radius was increased by 15 percent without increasing the number of cells. The enlarged cells and increased number of intercalated disks in the hypertrophied myocardium are believed to facilitate the intercellular current flow, which may be expected to increase the strength of the equivalent dipole generated by excitation of muscle layers. Consistent with this hypothesis was the finding of increased voltage recorded in the intramural electrograms from a hypertrophied human heart. In the presence of “pure” LVH (e.g., in children and young adults with isolated congenital
aortic stenosis), the maximum spatial QRS vector significantly correlated with the peak left ventricular systolic pressure. Because the QRS duration in this patient group was not increased, the increased voltage was believed to reflect an increased number of fibers in the hypertrophied myocardium. In middle-aged and elderly patients, a significant correlation was observed between the QRS voltage and the left ventricular mass estimated from angiograms and echocardiograms. Myocardial Surface

Hypertrophy increases the area of ventricular muscle in relation to the short-circuiting fluid surrounding the heart. Increased surface area and wall thickness may be expected to increase the solid angle subtended by the precordial electrodes (see Chapter 1).

In a study of left ventricular angiograms in 93 patients with LVH, wall thickening sufficient to result in an increased left ventricular mass did not result in increased QRS voltage indicative of LVH unless sufficient concurrent chamber dilation was present. This implied that there is a critical role for the geometric relation between wall thickness and chamber dilation, as would be expected from application of the solid angle theorem. However, a strict correlation between the left ventricular dimension and QRS voltage is not a universal finding. Thus Devereux et al. found that the QRS voltage correlated only weakly with the size of the left ventricular chamber; for a given left ventricular mass, it depended less on chamber dilation than on left ventricular weight, the depth of the left ventricle in the chest, and the patient’s age.

Intracavitary Blood Volume

The QRS amplitude may be expected to increase in the presence of an increased end-diastolic blood volume owing to a mechanism postulated by Brody. The Brody effect predicts that an increase in the intracardiac blood volume augments the initial (i.e., radial) QRS vectors and attenuates the late (i.e., tangential) QRS vectors. The validity of this principle was documented by correlating the R wave amplitude with the left ventricular volume, which was altered by pacing the heart at various rates. Numerous studies using ventricular angiography, nuclear imaging, and echocardiography have shown that the correlation of the QRS voltage with the left ventricular volume was either poor or not as good as the correlation with the left ventricular mass.

Proximity of the Heart to the Chest Wall

Feldman et al. have shown that the R wave amplitude increased as the left ventricular lateral wall moved closer to the V₃ and V₆ electrodes and that the proximity of the left ventricle to the anterior chest wall was a major determinant of the R wave amplitude. Moreover, echocardiographic studies showed that the correlation between the ECG criteria for LVH and left ventricular mass calculated from the echocardiogram...
could be improved by correcting for the distance of the center of the left ventricular mass from the chest wall.\textsuperscript{18}

**INTRINSICOID DEFLECTION**

In about 35 to 90 percent of LVH cases, the delayed onset of intrinsicoid deflections occurs \( \geq 45 \text{ ms} \) after the onset of the QRS complex,\textsuperscript{10} which may be due to late activation of the hypertrophied left ventricle. The finding is not specific, however, because late ventricular activation in subjects with LVH also correlated with right ventricular thickness.\textsuperscript{19}

**REPOLARIZATION ABNORMALITIES**

Deviation of the ST segment and the T wave in the direction opposite to the main QRS vector in the horizontal and frontal planes causes widening of the QRS/T angle. The combination of increased QRS amplitude and a wide QRS/T angle results in a pattern known as *left ventricular strain*. Even though the term “strain” is not appropriate to characterize an electrical event, use of this term has become entrenched in clinical practice. The ST and T changes in the left ventricular strain pattern are secondary to delayed propagation of the impulse in the conducting system, the hypertrophied myocardium, or both (see Chapter 23).

**Diagnostic Criteria for Patients Aged 40 Years or Older**

In most studies, high voltage in the precordial leads was the most sensitive criterion for the diagnosis of LVH. A recognition rate of up to 56 percent may be achieved, although it also was most frequently responsible for false-positive diagnoses.

If the duration of the QRS complex is \(<0.12 \text{ second}\), the following criteria are used for the diagnosis of LVH. They are based mainly on the studies of Sokolow and Lyon.\textsuperscript{20}

The limb lead criteria are as follows:
1. R wave in lead I + S wave in lead III \( >2.5 \text{ mV} \)
2. R wave in aVL \( >1.1 \text{ mV} \)
3. R wave in aVF \( >2.0 \text{ mV} \)
4. S wave in aVR \( >1.4 \text{ mV} \)

Precordial lead criteria are as follows:
5. R wave in V\textsubscript{5} or V\textsubscript{6} \( >2.6 \text{ mV} \)
6. R wave in V\textsubscript{6} + S wave in V\textsubscript{1} \( >3.5 \text{ mV} \)
7. Largest R wave + largest S wave in the precordial leads \( >4.5 \text{ mV} \)

The most commonly used *voltage* criteria in the more recent literature are as follows:

- S in V\textsubscript{1} + R in V\textsubscript{6} \( >3.5 \text{ mV} \)
- S in V\textsubscript{2} + R in V\textsubscript{6} \( >4.3 \text{ mV} \)
- S in V\textsubscript{1} \( >2.4 \text{ mV} \)
- S in V\textsubscript{6} \( >2.8 \text{ mV} \)
- R in aV\textsubscript{R} \( >1.3 \text{ mV} \)

In addition to the Cornell criteria of:
- R in aVL + S in V\textsubscript{3} \( >2.0 \text{ mV} \) for females
- \( >2.8 \text{ mV} \) for males

Supporting criteria include the following:
8. Onset of the intrinsicoid deflection in V\textsubscript{5} or V\textsubscript{6} \( >0.05 \text{ second} \)
9. ST segment depression and T wave inversion in the left precordial leads and in the limb leads in which major QRS deflection is upright in the presence of one or more of the above findings

Romhilt and Estes\textsuperscript{21} proposed a point-score system that uses a combination of the various findings listed in Table 3–1. LVH is considered

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amplitude: any of the following</td>
<td>3</td>
</tr>
<tr>
<td>Largest R or S wave in the limb leads ( &gt;20 \text{ mm} )</td>
<td></td>
</tr>
<tr>
<td>S wave in V\textsubscript{1} or V\textsubscript{2} ( \geq 30 \text{ mm} )</td>
<td></td>
</tr>
<tr>
<td>R wave in V\textsubscript{3} or V\textsubscript{6} ( \geq 30 \text{ mm} )</td>
<td></td>
</tr>
<tr>
<td>ST segment and T wave changes (typical pattern of left ventricular strain with the ST segment and T wave vector shifted in a direction opposite to the mean QRS vector)</td>
<td>3</td>
</tr>
<tr>
<td>Without digitalis</td>
<td>3</td>
</tr>
<tr>
<td>With digitalis</td>
<td>1</td>
</tr>
<tr>
<td>Left atrial involvement: terminal negativity of the P wave</td>
<td>3</td>
</tr>
<tr>
<td>in V\textsubscript{1} is ( \geq 1 \text{ mm} ) in depth with a duration of ( \geq 0.04 \text{ second} )</td>
<td></td>
</tr>
<tr>
<td>Left axis deviation of (-30 \text{ degrees or more} )</td>
<td>2</td>
</tr>
<tr>
<td>QRS duration ( \geq 0.09 \text{ second} )</td>
<td>1</td>
</tr>
<tr>
<td>Intrinsicoid deflection in V\textsubscript{5} and V\textsubscript{6} ( \geq 0.05 \text{ second} )</td>
<td>1</td>
</tr>
</tbody>
</table>
present if the points total 5 or more and is probably present if the points total 4.

**Echocardiographic Criteria**

Casale et al. used the echocardiographically determined left ventricular mass measurements in 414 subjects as a standard to develop new ECG criteria for LVH and tested various correlations prospectively in an additional 129 subjects. The best criteria consisted of a combination of the sum of R amplitude in aVL and S amplitude in V3 to be >2.8 mV in men and >2.0 mV in women, associated with increased T wave amplitude in lead V1. This combination had a sensitivity of 49 percent and a specificity of 93 percent, which was better than the respective values of 33 percent and 94 percent for the Sokolow-Lyon voltage criterion and 30 percent and 93 percent for the Romhilt-Estes score of 4 points or more. In the same study, ECG criteria based on a multiple logistic regression equation developed in the learning series and tested prospectively achieved 51 percent sensitivity, 90 percent specificity, and 76 percent overall accuracy for diagnosis of LVH. The innovative feature of these criteria is the separation by gender. A more accurate correlation was found subsequently using the product of either the Cornell voltage or 12-lead voltage system and the QRS duration.

Framingham Heart Study investigators evaluated 10 ECG criteria for detecting LVH using ECG and echocardiographic measurements from 3351 adults. The best performer for evaluating ECG and echocardiographic measurements from 10 ECG criteria for detecting LVH using criteria, Scott and associates studied 100 cases of isolated LVH with increased heart weight and left ventricular wall thickness at autopsy. Using one or more of the criteria listed previously (except criterion 7), 85 percent of the cases were correctly diagnosed. In this study, however, ST segment and T wave changes alone were also considered as indicative of LVH. In two later investigations, the sensitivities were 60 percent and 85 percent, respectively.

To test the specificity of listed criteria (except criterion 7), Chou et al. reviewed the autopsy findings of 100 cases diagnosed as LVH by ECG less than 3 months before death. Cases with ST segment and T wave changes alone were not included. Taken together, 44 cases had isolated LVH, and 45 had combined ventricular hypertrophy. A false-positive diagnosis of LVH was made in 11 cases (11 percent). Selzer and associates reported a slightly higher incidence (15 percent) of false-positive diagnoses. The point-score system, tested in 360 autopsied hearts by the chamber dissection technique, gave a sensitivity of 54 percent, with only 3 percent false-positive diagnoses.

**Sensitivity and Specificity of Individual Criteria**

Mazzoleni and co-workers examined the reliability of several of the individual criteria in the autopsied hearts with the chamber dissection technique. In 185 unselected adult patients, anatomic LVH was recognized in 22 percent by the criterion RV5 or V6 + SV1 >3.5 mV and in 17 percent by the criterion RaVL >1.0 mV.

A group of Cornell investigators correlated antemortem ECGs with a left ventricular mass at autopsy in 220 patients. LVH was defined as an LV mass index >118 g/m² in men and >104 g/m² in women. They found that the product of the QRS duration and a 12-lead voltage (voltage-duration product) identified LVH more accurately than the voltage criteria alone, QRS duration criteria alone, or the Romhilt-Estes point score.

One of the most extensive anatomic correlation studies using the chamber dissection technique was conducted by Romhilt and associates. A total of 33 ECG criteria for LVH were evaluated. The most sensitive (45 percent) was the R + S amplitude >4.5 mV. In other studies, increased QRS voltage correlated to variable degrees with the sum of the thickness of the septum and the posterior wall, the posterior wall thickness alone, or the calculated left ventricular mass.

**ST Segment and T Wave Changes**

The two most commonly used repolarization criteria for the diagnosis of LVH are a QRS/T angle >100 degrees and a T wave that is upright in V3 and more negative than −0.1 mV in V6. The classic ST and T wave changes in LVH consist of ST segment depression with upward convexity and T wave inversion in the left precordial leads (Figures 3–1 to 3–4). Reciprocal changes are present in the right precordial leads with ST
segment elevation and a tall T wave. In the limb leads, the direction of the ST and T vectors is also directed opposite to the main QRS forces. Therefore ST segment depression and T wave inversion are seen in leads I and aVL when the QRS axis is horizontal and in leads II, III, and aVF when the QRS axis is vertical. These classic ST and T wave changes are usually found in patients with fully developed LVH.

If high QRS voltage and the secondary repolarization changes are both present, a false-positive diagnosis of LVH is seldom made. Less pronounced repolarization changes, such as slight ST segment depression or flat T waves in the left precordial leads, which usually precede frank ST segment deviation and T wave inversion during development of LVH, are also helpful, particularly when the voltage is increased.

A common diagnostic difficulty encountered in practice is recognition of the ST segment and T wave abnormalities caused by myocardial ischemia in the presence of the left ventricular strain pattern in patients with high-voltage QRS complexes suggestive of LVH. In theory, if the QRS area is strictly proportional to the ventricular mass and if the hypertrophy does not alter the
The morphology of the secondary T wave changes is distinctly different from that of the primary T wave changes and is suggestive of ischemia in about two thirds of the cases. In the remaining third of patients with documented LVH and normal coronary arteries, the negative T waves do not have the characteristic asymmetric configuration of secondary T waves (see Chapter 23) but instead resemble the primary terminally inverted T waves with an isoelectric or horizontally depressed ST segment, such as in the presence of subacute or chronic myocardial ischemia (see Figures 3–2 and 3–4). In such cases one cannot use repolarization criteria to support the diagnosis of LVH. Repolarization changes attributed to myocardial ischemia can be distinguished from the secondary repolarization abnormalities caused by LVH if, instead of the expected secondary ST segment elevation and upright T wave, one encounters ST segment depression and T wave inversion in leads V1 and V2 (see Chapters 7 and 8). Favoring a diagnosis of myocardial ischemia is the rapid appearance and disappearance of repolarization abnormalities in patients with coexisting coronary artery disease. It should be mentioned that transient or permanent T wave inversion in the right precordial leads in patients with LVH are occasionally caused by “right heart strain.”

OTHER ECG CHANGES IN LVH
Incomplete Left Bundle Branch Block
In patients with LVH, Q waves often decrease in size or are absent (Figures 3–5 and 3–6). The Q wave was absent in about 50 percent of autopsied hearts with LVH resulting from various causes (T.C. Chou, 1960, unpublished data). The absence of the Q wave in the left precordial leads is related to leftward displacement of the initial QRS forces, which is also partly responsible for the decrease in the R wave in the right precordial leads.

When high QRS voltage and ST and T wave changes in LVH are accompanied by an increased QRS duration up to 0.11 second, an absence of Q waves with a delay in the onset of the intrinsicoid deflection in the left precordial leads, and (occasionally) notching of the QRS in the mid-precordial lead, the often-raised question is whether incomplete left bundle branch block (LBBB) coexists with hypertrophy. The problem is seldom of practical importance because incomplete LBBB is nearly always associated with marked LVH and therefore has the same significance as the LVH pattern with secondary ST and T wave changes.
Abnormal Left Axis Deviation
When used alone, an abnormal left axis deviation of −30 degrees or more is a relatively insensitive, nonspecific sign of LVH. Grant found no correlation between heart weight and left axis deviation.

Poor R Wave Progression in Precordial Leads
Poor progression of the R wave in the precordial leads occurs commonly with LVH and is associated with a leftward shift of the transitional zone in the precordial leads (i.e., R/S ratio <1 in V₃). Occasionally, R waves...
are absent in leads V1, V2, and even V3, resulting in a QS deflection in these leads that mimics anteroseptal myocardial infarction (see Figures 3–2 and 3–3; also see Chapter 9).

**Abnormal Q Wave in Inferior Leads**

Occasionally, an abnormal Q wave is recorded in leads III and aVF and less often in lead II, mimicking inferior myocardial infarction. Examples are shown in Figures 3–1 and 3–5. The mechanism of this pseudoinfarction pattern is not clear.

**Notching and Prolongation of QRS Complex**

In some patients the normal QRS complex is slightly prolonged and notched, especially in the mid-precordial leads, probably as a result of an intraventricular conduction defect (see Figure 3–3).

**U Wave**

The amplitude of a normal positive U wave may be increased in the right precordial leads, but the increase appears to be proportional to the overall increase in the amplitude of the ventricular complex. The U wave is often negative in the left precordial leads in patients with LVH, particularly when the latter is caused by systemic hypertension or regurgitation of the mitral or aortic valve (see Figure 3–3).42

**Anatomic Correlations**

Reichek and Devereux43 found that the left ventricular mass estimated from M-mode echocardiography correlated well with the postmortem left ventricular weight in 34 subjects, and the respective sensitivities of the Romhilt-Estes point score and the Sokolow-Lyon voltage criteria were 50 percent and 21 percent, with a specificity of 95 percent for both criteria.

Several echocardiographic studies confirmed that the sensitivity and the specificity of the left ventricular strain pattern or of the Romhilt-Estes point score (particularly when the score was >5) was superior to the voltage criteria alone. The point score of Romhilt and Estes correlated with an increased left ventricular mass and was associated with a sensitivity of 57 percent and a specificity of 81 percent.44 In two other studies45,46 the left ventricular strain pattern had a sensitivity of 52 percent and a specificity of 95 percent.

Crow et al.47 tested the performance of eight ECG criteria with the echocardiographic left ventricular mass index in a biracial population of men and women enrolled in the Treatment of Mild Hypertension Study. The ECG LVH sensitivity at 95 percent specificity was <34 percent. The Cornell voltage criteria showed the highest average sensitivity (17 percent). The ECG correlations with the LV mass index were consistently improved by including non-ECG variables, such as blood pressure and body mass index. The authors47 thought that further refinement of the ECG criteria alone in Caucasian men was unlikely to improve its relation to the left ventricular mass. More recently, Budhwani et al.48 correlated several traditional ECG criteria with the increased left ventricular mass estimated by echocardiography in 608 patients. Increasing number of the ECG criteria was associated with a greater mean left ventricular mass, but increased wall thickness and ventricular diameter did not influence significantly the frequency of any of the ECG criteria.

Echocardiographic studies exposed the shortcomings of the ECG in its ability to differentiate among concentric hypertrophy, eccentric hypertrophy, and dilatation without hypertrophy and demonstrated that the QRS voltage may be increased in the presence of an increased left ventricular diastolic diameter and normal thickness of the left ventricular wall.34 It appears that none of the tested criteria, such as the magnitude of the maximal QRS vector in the horizontal plane,35 the QRS amplitude in the scalar ECG, or T wave changes, can distinguish reliably between concentric LVH and isolated left ventricular dilatation. Echocardiography also showed that for a diagnosis of asymmetric LVH, the presence of prominent abnormal Q waves attributed to septal hypertrophy was a poor predictor of increased septal thickness or the septal wall/free wall ratio31,49 (see Chapter 12). The echocardiogram is also superior to the ECG for detecting LVH in the presence of a ventricular conduction defect.

**Systolic and Diastolic Overload of the Left Ventricle**

Cabrera and Monroy introduced the concept of systolic and diastolic overload of the left ventricle.50 Later writers used the terms pressure overload and volume overload. The ECG pattern of left ventricular systolic overload includes high voltage of the R wave and the classic secondary ST segment and T wave changes in the left precordial leads (see Figures 3–3 and 3–5). It occurs with such conditions as aortic stenosis, systemic hypertension, and coartation of the aorta when the left ventricle contracts against increased resistance. With diastolic overload of the left ventricle, which is seen in patients with aortic insufficiency,
mitral insufficiency, and patent ductus arteriosus, the ECG usually exhibits high voltage of the R waves with prominent Q waves in the left precordial leads (Figure 3–7). The ST segment is usually elevated, with an upright, peaked T wave.

Although the concept of systolic and diastolic overload has enjoyed some popularity, its clinical application often has been disappointing, especially in patients with advanced acquired heart disease, severe dilatation, and hypertrophy. In young patients with congenital heart disease, correlation of the hemodynamic state and the ECG pattern usually is better, particularly in patients with a ventricular septal defect without pulmonary hypertension whose ECG often shows a prominent Q wave followed by a tall R wave and an upright T wave (Figure 3–8).

**PHYSIOLOGIC FACTORS AFFECTING THE RELIABILITY OF DIAGNOSTIC CRITERIA**

Most of the present ECG criteria for LVH have been developed for populations with a high
prevalence of heart disease. The Bayes theorem predicts that these criteria would result in a high incidence of false-positive ECG interpretations in a population with a low prevalence of heart disease. Indeed, in such populations none of the ECG signs of LVH are specific, and no QRS voltage criteria have more than 46 percent accuracy for the diagnosis of LVH.51

A common vexing problem stems from variations in precordial voltage. Such variations may be due to changes in electrode position but may also occur in the same individual at 24-hour intervals, even independent of electrode position, perhaps due to changes in respiration or heart rate.52 The ST and T wave abnormalities are even less specific than the voltage. Moreover, the combination of increased voltage and a wide QRS/T angle can be present without hypertrophy due to delayed conduction in the left bundle branch system. This can be shown by applying early premature atrial stimuli, which causes a delay in the left bundle branch. The LVH pattern produced in this manner can be normalized by delaying conduction in the right bundle branch.53

Age

Age is one of the most important factors to be considered in the ECG diagnosis of LVH. Most of the voltage criteria were derived and tested against the older population. It is well known, however, that the QRS voltage is higher in adolescents and young adults than in older individuals (Figure 3–9). For example, from age 20 to 29 years, the normal 99th percentile for

\[ SV_1 + RV_5 \text{ or } V_6 \text{ is } 5.3 \text{ mV}. \]

The voltage of \[ SV_2 + RV_5 \text{ is more than } 3.5 \text{ mV} \] in 32 percent of normal men 20 to 39 years of age (T.C. Chou, 1960, unpublished data). It has been estimated that the amplitude of the maximum spatial QRS vector decreases by 6.5 percent for each decade of life from age 20 to 78.53

During routine interpretation of the ECG, LVH must be diagnosed with caution in patients under age 40 if only the voltage criteria are met, unless the amplitude is extremely high. Even the combination of high QRS voltage and ST and T wave changes, which is reliable in older subjects, cannot be applied with equal confidence in young subjects. The ST and T wave abnormalities may be due to other causes in the young patient, and the high amplitude of the QRS may be normal for the particular body build. It is therefore advisable to look for some other supporting evidence in the QRS complex such as abnormal left axis deviation, a delay in the onset of the intrinsicoid deflection in leads \( V_5 \) and \( V_6 \), an increase in QRS duration to 0.11 second, the presence of notching of the QRS complex in the mid-precordial leads, and especially poor progression of the R wave in the right and mid-precordial lead. Because most normal individuals have taller R waves in lead \( V_5 \) than in lead \( V_6 \), reversal of this order also is suggestive of an abnormality.

The correlation between the ECG and the left ventricular wall thickness in the elderly was determined in 671 autopsies of patients 65 to 116 years of age. The correlation was not affected by age in subjects younger than 85 years but was blunted in those older than 85.56

Figure 3–9 ECG with high QRS voltage recorded from a healthy 22-year-old male medical student. \( RV_5 + SV_1 = 46 \text{ mm} \); \( RV_5 + SV_2 = 49.5 \text{ mm} \).
Body Habitus

The voltage is reduced in persons with large breasts. A significant increase in R amplitude in leads V1–V5 occurred after left mastectomy and in leads V3R and V4 after right mastectomy.57 On the basis of increased voltage, LVH would erroneously diagnosed in nearly 50 percent of women after mastectomy.57 Other factors that may alter voltage in the absence of hypertrophy are hematocrit, intraventricular conduction disturbance, and perhaps myocardial ischemia. A low hematocrit increases the QRS voltage and a high hematocrit decreases it because of changes in blood resistivity.58

Kilty and Lepeschkin51 examined the effect of body build on the QRS voltage of the ECG. Using the ponderal index (height in inches divided by the cube root of weight in pounds) as a measure of body build, they found a highly significant correlation between body build and the voltage of R1 + S3, largest R + S in the precordial leads, and largest R + S in a single precordial lead. The precordial lead criterion R + S >4.5 mV underdiagnoses LVH in obese people and overdiaognoses it in thin people. Others have also noted that most false-positive diagnoses of LVH occur in emaciated individuals.51 The increased amount of adipose or muscle tissue in the chest wall affects the voltage as a result of an increased distance between the precordial electrode and the heart. An accumulation of adipose tissue around the heart of overweight persons may produce a similar effect59 (see Figure 1–23).

The Cornell investigators60 found that the ability of ECG criteria to detect LVH differs depending on the method used to index the left ventricular mass for body size and with the presence or absence of obesity. When the left ventricular mass was indexed to the body surface area or to height, the sensitivity of the Cornell product criteria increased from 39 to 52 percent with a matched specificity of 95 percent.

Gender and Race

Gender has a significant effect on the amplitude of the QRS complex. Men have higher amplitudes than women in both the limb leads and precordial leads, but especially in the latter.59 In women it is sometimes important to know whether lead V4 was recorded with the electrode on or underneath the breast. A significant reduction in voltage may occur if the electrode is placed on a large breast (Figure 3–10).

African-American subjects are reported to have higher QRS voltage than Caucasian subjects. In a study of 114 healthy adolescents61 the upper limit for the S wave amplitude in lead V1 was 3.4 mV in black males, 3.2 mV in white males, 2.6 mV in black females, and 1.6 mV in white females. The corresponding values for the R amplitude in lead V6 were 3.0, 2.4, 2.2, and 2.4 mV.62

In another study of 15- to 19-year-old adolescents61 the sum of S in V1 and R in V6 amplitudes averaged 3.69 mV in black males and 3.13 mV in white males. No significant differences were found between black and white females. The echocardiograms showed that the left ventricular posterior wall was thicker and the distance between the anterior chest wall and the mid-left ventricle was shorter in black males than in white males. No such racial differences were observed in females.

The Chicago Heart Association Detection Project in Industry collected data from 1391 black men and 19,216 white men.63 The prevalence of an ECG LVH pattern was significantly higher in black men than in white men in each age group. The difference remained significant after adjustment for all possible risk factors. Examination of the data from National Health and Nutrition Surveys64 also showed that black racial background was associated with a more than threefold excess of LVH by the Cornell voltage criteria.

In a smaller study of 196 African-Americans, ages 54 to 100 years, Arnett et al.65 found that the application of ECG algorithms was associated with misclassification of LVH and overestimation of the left ventricular mass by echocardiography. The diagnostic accuracy of the Cornell criteria in African-Americans was comparable to the accuracy reported in white subjects.

PATHOLOGIC STATES AFFECTING THE DIAGNOSIS OF LVH

The lung is a poor electrical conductor and attenuates the QRS voltage. In patients with chronic obstructive pulmonary disease (COPD), the voltage of the QRS complex may be markedly reduced. The presence of LVH often is not recognized in such patients. Pericardial effusion may also mask a ventricular hypertrophy pattern because of the short-circuiting effect of the pericardial fluid. Pulmonary edema may reduce the body surface potential by its short-circuiting effect.66 A reduction of the amplitude of the complexes is also seen in patients with pleural effusion, generalized anasarca, or pneumothorax.

Myocardial damage in patients with coronary artery disease, secondary myocardial disease such as amyloidosis, or scleroderma-associated heart disease is often accompanied by low
voltage, which makes diagnosing LVH difficult. The presence of myocardial infarction did not affect ECG recognition of LVH by the voltage criteria in an autopsy series,\textsuperscript{67} although changes in QRS amplitude during the acute stage are often present (see Chapter 7).

In most patients with RVH secondary to left ventricular disease, the effect is minimal and the diagnosis of LVH is not affected. When RVH is severe, however, the LVH pattern may not be apparent because of the canceling effect of the rightward forces.

ASSESSMENT OF THE SEVERITY OF VALVULAR LESIONS CAUSING LVH

Most attempts to correlate the ECG with the severity of a valvular lesion have been made in patients with valvular aortic stenosis. Hugenholtz and associates\textsuperscript{68} studied 95 patients (ages 6 weeks to 20 years) with congenital aortic stenosis. About three fourths of the patients with severe lesions and one fourth of those with mild lesions had evidence of LVH. Braunwald and associates\textsuperscript{69} reported hemodynamic and ECG findings in 100 patients with congenital aortic stenosis. Their ages ranged from 2 to 51 years, with 36 patients younger than 10 years. No single ECG change was found to be reliable for determining the severity of the obstruction. The ECG was more helpful in patients younger than age 10 than in older individuals.

Roman and associates\textsuperscript{70} correlated the ECG, echocardiographic, and radionuclide angiographic findings in 95 adults with severe, pure, chronic aortic insufficiency and no evidence of coronary
artery disease. The correlation between the QRS voltage and the degree of left ventricular dilatation and dysfunction was weak. In patients with chronic aortic regurgitation and massive cardiomegaly (heart weighing 1 kg or more), the usual QRS criteria for LVH were often absent.

In patients with mitral insufficiency, the correlation between the severity of the mitral lesion and the ECG changes is generally poor. In a series of 65 cases of dynamically significant mitral insufficiency without significant mitral stenosis confirmed at surgery or autopsy, the ventricular complex was normal in 50 percent and suggestive of LVH in 30 percent, RVH in 15 percent, and combined ventricular hypertrophy in 5 percent of cases.

**LVH and Systemic Hypertension**

In the early reports from the Framingham Study, the ECG was normal in almost half, borderline in one fifth, and definitely abnormal in one third of a group of 154 patients with a clinical diagnosis of hypertensive cardiovascular disease. During the follow-up, the incidence of an LVH ECG pattern over a 14-year period of observation rose in proportion to the blood pressure elevation in all age groups. About half the patients with systolic pressures above 200 mmHg had or developed an LVH ECG pattern during the 14 years.

Ashizawa and associates reported a 26-year follow-up study of 601 patients with hypertension and ECG changes of LVH with both high QRS voltage and ST segment and T wave changes. The patients were examined at 2-year intervals. In 60 subjects the ECG signs of LVH developed during the observation period. In 37 of the subjects, high QRS voltage appeared first, followed by ST and T wave changes. In 10 patients the ST and T wave changes occurred first; in the other 13 patients the QRS and ST segment and T wave changes appeared simultaneously. In 72 of the 601 subjects the LVH pattern regressed. The QRS voltage became normal in 36, ST segment and T wave changes disappeared in 25, and both high QRS voltage and ST segment and T wave changes disappeared in 11 patients. In about half of these 72 subjects, regression of the abnormal ECG findings, usually the QRS voltage, was associated with lowering the blood pressure. Regression of the ECG abnormalities during antihypertensive therapy was associated with lower morbidity and mortality.

**Prognostic Value of ECG Changes**

The ECG is of limited value for predicting the severity of the hypertension in individual patients. It is believed, however, that in the presence of both ST segment and T wave changes and high QRS voltage, patients usually have a higher blood pressure than those with high QRS voltage alone.

In a study of 227 patients with untreated diastolic hypertension before the advent of echocardiography, the left ventricular mass was predicted by combining the surface area, gender, age, the S voltage in leads V1 and V4, and the duration of the terminal P wave component in lead V1. In the Framingham study, the mortality of hypertensive patients with a definitive LVH pattern (tall R wave, ST segment depression, flattened or inverted T wave, increased ventricular activation time in the left precordial leads) was much higher than in subjects without such findings, even though the blood pressure was the same. In an analysis of 4824 patients by Sullivan et al., the ECG pattern of LVH had an independent adverse effect on survival, even in patients without coronary artery disease.

In a study of 923 Caucasian untreated hypertensive patients, in whom the prevalence of echocardiographic LVH was 34 percent, the specificity of ECG for LVH was >90 percent and the sensitivity varied between 9 percent and 33 percent. The highest sensitivity was found when three highly specific criteria (Cornell voltage, Romhilt-Estes score, left ventricular strain pattern) were combined. Combination of these criteria was also most predictive of cardiovascular events in 1717 Caucasian hypertensive patients followed prospectively for up to 10 years. In a long-term Losartan Intervention for Endpoint Reduction (LIFE) in Hypertension study of 8696 hypertensive subjects, ECG LV strain pattern identified patients at increased risk of developing congestive heart failure and dying, even in the setting of aggressive blood pressure lowering.

**PRESENCE OF LBBB**

Klein et al. used echocardiograms to develop criteria for diagnosing LVH in patients with LBBB. They found that the sum of the S amplitude in V3 and the R amplitude in V6 exceeding 4.5 mV had an 86 percent sensitivity and 100 percent specificity for LVH, and that the diagnosis of LVH was supported by the findings of left atrial enlargement and QRS duration >160 ms.

**Right Ventricular Hypertrophy and Dilatation**

Right ventricular forces normally are directed anteriorly and rightward but are mostly masked by the dominant left ventricular potential. An
increase in the right ventricular muscle mass modifies the resultant forces proportional to the severity of the hypertrophy. In mild cases, no apparent change may be detected in the ECG. If the RVH is severe, the normally dominant left posterior QRS forces may be replaced by prominent right anterior forces, and the QRS patterns in the left and right precordial leads are reversed. Intermediate degrees of abnormality may be observed when the hypertrophy is moderate. Because lead V_1 is more proximal to the right ventricular mass, it is the most sensitive lead for recording the changes. Tall R wave, small S wave, or a change in the R/S ratio may be observed. The onset of the intrinsicoid deflection in this lead may be delayed because of delayed activation of the right ventricular epicardium, but the delay is seldom sufficiently pronounced to be of diagnostic usefulness.

In some instances the increased rightward forces are directed posteriorly instead of anteriorly. In such cases no apparent abnormality is seen in lead V_1, but the left precordial leads may reveal deep S waves, and the increased rightward forces may be recognized in the limb leads as right axis deviation.

Secondary T wave change often occurs. The T waves are directed opposite to the main QRS vector (i.e., inverted in the right precordial leads and upright in the left precordial leads). These secondary T wave abnormalities may be accompanied by secondary deviations of the ST segment. In some instances, the ST and T changes may be seen without apparent QRS abnormalities and are attributed to myocardial ischemia of the right ventricle, or right ventricular strain.

THREE TYPES OF RVH

It is useful to subdivide the ECG manifestations of RVH and right ventricular dilation into three types: (1) typical RVH pattern with anterior and rightward displacement of the main QRS vector; (2) incomplete right bundle branch block (RBBB); and (3) posterior and rightward displacement of the main QRS axis, predominantly in patients with chronic lung disease.

Typical RVH Pattern

The typical RVH pattern is a mirror image of the LVH pattern, with right axis deviation in the frontal plane, tall R waves in the right precordial leads, deep S waves in the left precordial leads, and a slight increase in QRS duration (Figure 3–11). This pattern is characteristically present in patients with congenital pulmonary stenosis, tetralogy of Fallot, primary pulmonary hypertension, and other conditions in which the right ventricular mass tends to approach or exceed the left ventricular mass. The earliest portion of the QRS complex is usually unchanged because septal activation is normal. The anterior displacement of the main QRS vector manifested by tall R waves in the right precordial leads is attributed to a longer activation time of the hypertrophied right ventricular free wall.

Intraoperative epicardial mapping of patients with RVH undergoing pulmonary thromboendarterectomy showed that right ventricular activation was delayed by an average of 36 ms. In addition, the latest right ventricular epicardial activation occurred significantly later than the latest left ventricular activation (i.e., average of 75 ms vs. average of 64 ms). In some cases there was no discrete early right ventricular strain.

Figure 3–11 Typical right ventricular hypertrophy pattern with right axis deviation and negative T waves in leads V_1–V_4 in a 26-year-old man who had undergone corrective surgery for transposition of the great vessels during childhood.
breakthrough, but in these cases the latest right ventricular activation also occurred significantly later than the left ventricular activation.

**Sensitivity and Specificity of Criteria for RVH**

The sensitivity and the specificity of various criteria are generally inversely related. The early criteria of Myers and associates and Sokolow and Lyon included the following:

1. \( \text{R in } V_1 > 0.7 \text{ mV} \)
2. \( \text{S in } V_1 < 0.2 \text{ mV} \)
3. \( \text{S in } V_5 \text{ or } V_6 > 0.7 \text{ mV} \)
4. \( \text{Sum of R in } V_1 \text{ and S in } V_5 \text{ or } V_6 > 1.05 \text{ mV} \)
5. \( \text{R in } V_5 \text{ or } V_6 < 0.5 \text{ mV} \)
6. \( \text{R/S ratio in } V_5 \text{ or } V_6 < 1 \)
7. \( \text{R in } aV_R > 0.5 \text{ mV} \)
8. \( \text{R/S ratio in } V_5 \text{ divided by R/S ratio in } V_1 < 0.4 \)
9. \( \text{R/S ratio in } V_1 \text{ (or } V_3R) > 1 \)
10. \( \text{qR pattern in } V_1 \text{ (or } V_3R) \)

Supporting criteria include the following:

11. Onset of intrinsicoid deflection in \( V_1 \) later than 0.04 second
12. Negative T wave in \( V_1 \) in the presence of \( R > 0.5 \text{ mV} \)
13. Right axis deviation >110 degrees

These criteria have high specificity but low sensitivity. In the autopsy series in which the anatomic diagnosis was based mainly on right ventricular wall thickness, the ECG met one or more of the criteria in 23 to 100 percent. Roman and colleagues examined 118 hearts with an ECG diagnosis of RVH. There was a false-positive diagnosis in 60 percent of the cases. The criteria of \( R \) in \( V_1 + S \text{ in } V_5 \text{ or } V_6 > 1.05 \text{ mV} \) and \( S \text{ in } V_5 \text{ or } V_6 > 0.7 \text{ mV} \) were responsible for most of the erroneous diagnoses. Chou et al. studied 97 patients in whom the presence of RVH was supported by hemodynamic data. The sensitivity of the ECG was 66 percent.

Burch and de Pasquale found that none of the RVH criteria were satisfied in 40 percent of cases of autopsy-proven RVH due to cor pulmonale. Kilcoyne et al. found that only 28 percent of 81 patients with cor pulmonale had an RVH pattern on the ECG.

Milnor modified the Sokolow and Lyon criteria and limited the variables to: QRS duration <0.12 second and a frontal axis of +110 to +180 degrees or -91 to ±180 degrees; or an R/S or R/or S ratio in \( V_1 > 1.0 \), with R or S amplitude >0.5 mV. These criteria have the advantage of applicability in the presence of incomplete RBBB. They predicted RVH correctly in 24 of 32 autopsy-proven cases. A 79 percent sensitivity and 73 percent specificity for diagnosing RVH was reported by Holt et al. using the 12-dipole ECG derived from records made at 126 body sites.

Behar et al. evaluated the usefulness of the Butler-Legget criteria for diagnosing RVH. These criteria are as follows: (1) P wave amplitude >0.25 mV in any of leads II, III, aVF, \( V_1 \), or \( V_2 \); (2) R wave amplitude = 0.2 mV in lead I; (3) \( A + R - PL = 0.7 \text{ mV} \), where \( A \) (anteriorly directed deflection) is derived from lead \( V_1 \) or \( V_2 \), \( R \) (rightward deflection) is the S amplitude in lead I or \( V_6 \), and \( PL \) (posterolateral deflection) is the S amplitude in \( V_1 \). In patients with pulmonary hypertension, these criteria achieved 66 percent sensitivity for mitral stenosis, 97 percent sensitivity for pulmonary arterial obstructive disease, and 79 percent sensitivity for pulmonary disease. The specificity of these criteria was enhanced by ruling out posterior myocardial infarction using the Selvester QRS scoring system (see Chapter 8).

The explanation for the marked differences in the results probably lies in the patient population sampled. The lower sensitivity was obtained mostly from unselected adult patients in a general hospital. In these patients, RVH most commonly develops as a result of left ventricular disease. The degree of hypertrophy usually is mild and likely to be masked by the dominant left ventricle. The higher sensitivity values mostly came from centers with large numbers of patients with congenital heart disease and a high incidence of severe RVH.

Flowers and Horan used the chamber dissection technique to examine 819 apparently unselected hearts, including 178 with RVH. The sensitivity and specificity of the individual criteria are summarized in Table 3–2, which indicates that the sensitivity of the individual criterion is low, generally less than 20 percent. The more sensitive signs, such as those based on changes in the left precordial leads, usually are associated with a larger number of false-positive diagnoses. By comparison, criteria based on abnormalities in lead \( V_1 \) are more specific but less sensitive.

**Echocardiographic Correlations**

Good correlations were found between the measurements of right ventricular wall thickness on the echocardiogram and autopsy findings. The echocardiogram was reported to be more sensitive for detecting RVH than the ECG. For increased right ventricular wall thickness, the sensitivity and the specificity of the echocardiogram were 93 percent and 95 percent, respectively. The corresponding values for the ECG
were 31 percent and 85 percent, respectively.\textsuperscript{103} When the ECG and the echocardiogram were compared in 134 patients with RVH and in 78 patients without RVH, the ECG had 27 percent sensitivity and 88 percent specificity. In more than half the patients the diagnosis of RVH was difficult to establish on the ECG because of conduction disturbances or old myocardial infarction.\textsuperscript{103}

### Differential Diagnosis

#### Abnormal Right Axis Deviation

Other than RVH, the situations in which right axis deviation may be seen are as follows:

1. Normal young or slender adults
2. Chronic obstructive pulmonary disease (COPD) without cor pulmonale
3. Lateral myocardial infarction
4. Left posterior fascicular block

Right axis deviation occurs normally in infants and children. The mean QRS axis during the first 4 weeks of life is $+110$ degrees or more.\textsuperscript{104} After 1 month the average axis is less than $+90$ degrees (although a significant number of children still have a QRS axis of up to $+110$ degrees). In the adult population, tall and slender subjects tend to have a rightward QRS axis. Hiss and colleagues\textsuperscript{105} reported that 2 percent of normal subjects 20 to 30 years of age have an axis of $+105$ degrees. A frontal plane QRS axis of more than $+110$ degrees in older individuals is uncommon, however, and usually suggests abnormality. Even an axis within the range from $+90$ to $+110$ degrees may indicate an abnormality in older patients, particularly if other ECG abnormalities coexist.

In patients with COPD the frontal plane axis may be within a range from $+90$ to $+110$ degrees in the absence of pulmonary hypertension. The recognition or exclusion of RVH in these patients is difficult. In patients with chronic lung disease without RVH, the amplitude of the entire QRS complex in lead I tends to be small.

In patients with lateral wall myocardial infarction, the loss of leftward forces may result in a rightward shift of the QRS vector. In these patients the initial R wave in lead I is usually absent, however, and abnormal Q waves are often observed also in the left precordial leads. Inverted T waves in leads I, aV\textsubscript{L}, V\textsubscript{5}, and V\textsubscript{6} are often present in patients with lateral myocardial infarction but are uncommon in patients with pure RVH.

### Tall R Wave, Small S Wave, Increased R/S Ratio in Lead V\textsubscript{1} in Conditions Other than RVH

One or more of the findings of a tall R wave, small S wave, and increased R/S ratio in lead V\textsubscript{1} in conditions other than RVH are:

\begin{table}[h]
\centering
\begin{tabular}{|c|c|c|c|}
\hline
Criterion & Sensitivity (%) & Specificity (False-Positives) (%) & Correctness (%) \\
\hline
Right axis deviation $\geq 110$ degrees & 12 & 4 & 78 \\
R/S V\textsubscript{1} (or V\textsubscript{3R}) $> 1$ & 6 & 2 & 78 \\
RV\textsubscript{1} $\geq 7$ mm & 2 & 1 & 78 \\
SV\textsubscript{1} $\leq 2$ mm & 6 & 2 & 78 \\
qR in V\textsubscript{1} & 5 & 1 & 79 \\
RV\textsubscript{1} + SV\textsubscript{5} or V\textsubscript{6} $> 10.5$ mm & 18 & 6 & 77 \\
R/S V\textsubscript{5} or V\textsubscript{6} $< 1$ & 16 & 7 & 77 \\
OID in V\textsubscript{1} (or V\textsubscript{3R}) $= 0.035–0.055$ second & 8 & 6 & 76 \\
RSR$^1$ in V\textsubscript{1} with R’ $> 10$ mm & 0 & 0 & 78 \\
aVR $\geq 11.5$ mm & 0 & 0 & 78 \\
RV\textsubscript{5} (or RV\textsubscript{6}) $< 5$ mm & 13 & 13 & 71 \\
SV\textsubscript{5} (or SV\textsubscript{6}) $\geq 7$ mm & 26 & 10 & 76 \\
\hline
\end{tabular}
\caption{Sensitivity, Specificity, and Correctness of ECG Criteria for Diagnosis of Right Ventricular Hypertrophy}
\end{table}


OID = onset of intrinsicoid deflection.
wave, a small S wave, or an increased R/S ratio in lead V1 may be seen in the following:

1. Normal young adults
2. True posterior infarction
3. Intraventricular conduction disturbances attributed to left septal fascicular block
4. Displacement of the heart due to pulmonary disease
5. Wolff-Parkinson-White pattern

A tall R wave with or without a small S wave in lead V1 is a frequent finding in normal children. The average amplitude of the R wave in V1 is more than 0.7 mV in children younger than 8 years. The amplitude exceeds 0.7 mV in 20 percent of children between ages 8 and 12 and in 11 percent of those between ages 12 and 16. An amplitude of up to 1.6 mV may be seen in normal adolescents. The R/S ratio in V1 is more than 1 in most children under 1 year of age, but the ratio progressively decreases as age increases. In adults an R/S ratio of 1 or more was reported in fewer than 1 percent of the normal population. Personal observations suggest that such a pattern is much more common, particularly among the younger and the obese populations. In lead V2 an R/S ratio of >1 was observed in as many as 10 percent of adults.

In patients with true posterior myocardial infarction, the polarity of the T wave in V1 often is helpful in the differential diagnosis. Although exceptions do occur, an inverted T wave usually is seen in patients with RVH (Figures 3–11 and 3–12; see also Figure 2–4), whereas an upright T wave in V1 is likely to be associated with posterior myocardial infarction. The coexistence of abnormal right axis deviation in the frontal plane favors RVH. Occasionally, however, right axis deviation is observed in patients with posterior myocardial infarction when it is complicated by left posterior fascicular block. Because true posterior myocardial infarction is seldom encountered in the absence of inferior or lateral wall infarction, the differential diagnosis is usually not difficult.

An R wave of 0.7 mV or more occasionally is seen in lead V1 in patients with autopsy-proven isolated LVH. In such cases the S wave amplitude in lead V1 is usually large. The cause of the tall R wave is unclear. In some cases it is associated with hypertrophic cardiomyopathy. These subjects often have a prominent R wave in the right precordial leads and a small S wave in lead V1, resulting in an R/S ratio of >1. Deep Q waves are usually present in the left precordial leads and in leads II, III, and aVF. Hypertrophy of the interventricular septum is probably responsible for these abnormal forces.

Rightward displacement of the heart as a result of disease of the lung or pleura, such as massive pleural effusion or pneumothorax, may be accompanied by a tall R wave in lead V1 (Figures 3–13 and 3–14) because lead V1 is believed to record left, rather than right, ventricular potential. In the absence of structural heart disease, the T wave in V1 is upright in the case of a displaced heart, whereas in patients with RVH the T wave in this lead is often inverted.

In patients with the Wolff-Parkinson-White pattern, the anterior direction of the delta wave resulting in an R or Rs pattern in lead V1 is present in most subjects with posterior and leftward insertions of the atroventricular bypass tract.

Figure 3–12  Severe mitral stenosis in a 47-year-old man with severe mitral stenosis proved at surgery. On the ECG the P waves are consistent with biatrial enlargement. The abnormal right axis deviation with an R/S ratio of >1 in lead V1 and the T wave inversion in the right precordial leads are consistent with right ventricular hypertrophy.
**qR Pattern in Lead V1.** The qR complex in V1 is one of the most specific signs of severe RVH (see Figure 3–14). When the earliest part of the QRS complex is isoelectric, abnormal depolarization of the interventricular septum probably is responsible for the initial negativity of the complex. It is possible that the right septal forces are now greater than those on the left, and the resultant vector is directed toward the left (opposite to the normal orientation). It has been also suggested that this complex is a manifestation of an enlarged right atrium that transmits to the right precordial leads the intracardiac potential from the right ventricle. Indeed, in patients with this pattern the right atrium is frequently enlarged, and tricuspid regurgitation is commonly present. Others have ascribed this qR morphology to the extreme rotation of the heart such that the forces arising from the left ventricle are now recorded in the V1 position.

Some normal adults have a QS rather than an rS deflection in lead V1. If such an individual develops RBBB, a qR rather than an rSR pattern will be recorded. If the QRS duration is 0.12 second or more, the conduction defect usually can be readily recognized. If the bundle branch block is incomplete with a QRS duration of 0.11 second or less, the differentiation becomes difficult. A similar problem exists in patients with anterior myocardial infarction and RBBB. The qR complex in these patients usually is accompanied by abnormal Q waves in the adjacent precordial leads.

**Deep S Wave, Small R Wave, R/S Ratio < 1 in Leads V5 and V6.** In patients with COPD, the left precordial leads often display small R waves, deep S waves, and an R/S ratio of <1. These changes may be observed in the absence of pulmonary hypertension or cor pulmonale. When the lung disease is not associated with RVH,
however, the amplitude of the QRS complexes in the left precordial leads tends to be low.

A similar pattern in the left precordial leads may be seen in normal subjects and with various conditions associated with cardiomegaly and marked leftward shift of the transition zone. An rS pattern in leads V5 and V6 may be seen in patients with anterior myocardial infarction\(^{110}\) (see Figure 4–7) and in those with left anterior fascicular block.\(^{111}\)

**ST Segment and T Wave Changes in RVH.** With RVH, ST segment depression and T wave inversion are seen most commonly in the right precordial leads (see Figures 3–12 and 3–14). These changes also may be seen in leads II, III, and aVF. If the T waves are biphasic in the right precordial leads, it is useful to note whether the configuration is of the positive-negative or negative-positive type. A negative-positive biphasic T wave is abnormal and often is seen in patients with RVH\(^ {112,113}\) (Figure 3–15), whereas the positive-negative configuration may be normal.

**Systolic and Diastolic Overload Patterns.** Cabrera and Monroy\(^ {50}\) called attention to the different ECG changes during systolic and diastolic overloading of the right ventricle. With systolic overloading, which was called pressure overloading by later investigators, lead V1 exhibits a tall monophasic R wave or a diphasic RS, Rs, or qR complex. The T wave usually is inverted in this lead. This pattern typically is seen in patients with pulmonary stenosis, tetralogy of Fallot, or pulmonary hypertension (see Figures 3–14 and 3–15). With diastolic, or volume, overload of the right ventricle, lead V1 usually shows an rSR\(^ {1}\) pattern. This is the typical QRS complex in patients with an atrial septal defect, partial anomalous pulmonary venous return, or tricuspid insufficiency. The anatomic alteration consists mainly of a dilated right ventricle instead of hypertrophy as in the case of systolic overload.

The clinical application of this concept is of questionable value. As a rule, the hemodynamic correlation of these two ECG overload patterns is more satisfactory in congenital than acquired heart disease.\(^ {114}\)

**S\(_1\)S\(_2\)S\(_3\) Pattern as a Manifestation of RVH.** The S\(_1\)S\(_2\)S\(_3\) pattern is a frequently used descriptive term during ECG interpretation. It is seen in normal individuals and in patients with pulmonary emphysema or RVH. Chou et al.\(^ {112}\) proposed the criterion of an R/S ratio of <1 in leads I, II, and III or the S waves in these leads that exceed the upper limits of normal for the various age groups as defined by Simonson.\(^ {115}\) S waves of low amplitude are more likely to be seen in normal subjects and deeper S waves in patients with RVH (Figure 3–16). Additionally, in patients with RVH and the S\(_1\)S\(_2\)S\(_3\) pattern, the prominent late QRS forces are directed rightward and superiorly. The S wave amplitude is usually greater in lead II than in lead III.

\(^{115}\)Upper limits of the normal amplitude of the S waves in leads I, II, and III:
- Age 20–29: S\(_1\), 0.4 mV; S\(_2\), 0.5 mV; S\(_3\), 0.6 mV
- Age 30–39: S\(_1\), 0.4 mV; S\(_2\), 0.4 mV; S\(_3\), 0.9 mV
- Age 40–49: S\(_1\), 0.3 mV; S\(_2\), 0.4 mV; S\(_3\), 0.8 mV

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**Figure 3–15** Severe pulmonary stenosis in a 19-year-old man. The right ventricular peak systolic pressure was 132 mmHg. On the ECG, the findings suggestive of right ventricular hypertrophy are an abnormal right axis deviation and an R/S ratio >1 in V1. The R wave in lead aVR is >5 mm. Note the negative-positive biphasic T wave in lead V1.
Incomplete RBBB

Incomplete RBBB, manifested by the rSR\textsuperscript{1} pattern in the right precordial leads, is attributed to delayed activation of the hypertrophied right ventricular outflow tract (see Chapter 5). This pattern is most frequently due to factors other than RVH. It can signify hypertrophy, dilation, or overload of the right ventricle, perhaps most commonly in mitral valve disease with pulmonary hypertension (Figure 3–17) and atrial septal defect (see Chapter 12). In patients with pulmonic valvular stenosis, the R\textsuperscript{1} voltage correlates with the severity of stenosis\textsuperscript{116} (Figure 3–18). The prompt disappearance of this pattern observed in many cases within days after corrective surgery suggests that incomplete RBBB may result from slowing of intraventricular conduction due to stretching of the peripheral conducting system in the dilated ventricle (see Chapter 5).

In addition to RVH, an rSR\textsuperscript{1} pattern in lead V\textsubscript{1} with the duration of the QRS complex <0.12 second is also seen in: (1) normal individuals; (2) acute right ventricular dilatation; (3) true posterior myocardial infarction; and (4) extracardiac abnormalities such as pectus excavatum. Further differential diagnoses of the incomplete RBBB pattern is discussed in Chapter 5.
Patterns in the Presence of Chronic Lung Disease

The characteristic ECG pattern in patients with COPD is attributed to changes in the spatial orientation of the heart and the insulating effect of the overaerated lungs.96 The changes induce peaked P waves in leads II, III, and aVF; a low R wave amplitude in all leads; and a late QRS vector oriented superiorly and to the right resulting in a wide, slurred S wave in leads I, II, III, V4, V5, and V6.96,118,119 Pathognomonic of emphysema in the absence of myocardial infarction is low voltage with a posteriorly and superiorly oriented QRS vector and an axis of the P wave >60 degrees in the limb leads.117 Atrial repolarization may become more prominent, as evidenced by depression of the Ta wave in lead II.121 In the presence of an rSr1 pattern in the right precordial leads, a slurred S wave in the left precordial leads and a prominent R wave in lead aVR may indicate superimposed RVH.117,120

Severity of Chronic Lung Disease

The best criteria for judging the severity of COPD are (1) R in V6 <0.5 mV; (2) R/S in V6 <1.0; and (3) increased P wave amplitude in leads II and III122 (Figure 3–19). In the orthogonal leads, low R wave amplitude and low R/S amplitude in the X lead, low voltage in the X and Y leads, and a rightward shift of the P axis identified COPD correctly in 75 percent of patients, with only 8 percent being
false-positive diagnoses. The best reported indicators of deteriorating pulmonary function in patients with COPD are (1) progressive reduction of the R wave and the R/S ratio in orthogonal lead X (may be applied to lead I), (2) progressive shift of the QRS axis in the superior direction, and (3) rightward shift of the P wave axis.

In a study of 263 cases of COPD followed for 13 years after an exacerbation of respiratory failure, Incalzi et al. identified the strongest predictors of death to be an S1S2S3 pattern, “right atrial overload” (defined as a P wave axis of +90 degrees or more), and an alveolar-arterial O2 gradient >48 mmHg. The median survival of patients having either of these two ECG signs was 2.7 years; of those having both ECG signs, 1.33 years.

**Chronic Cor Pulmonale**

In an effort to detect early cor pulmonale, Kilocoyne and co-workers surveyed 200 patients with COPD. One or more of the following ECG trends were suggested as indicators of right ventricular abnormality or dilation: (1) shift of the mean QRS axis to the right of +30 degrees; (2) T wave inversion in the right precordial leads; (3) ST segment depression in leads II, III, and aVF; and (4) transient appearance of RBBB. These ECG manifestations usually were associated with an arterial oxygen saturation of less than 85 percent and a mean pulmonary arterial pressure of 25 mmHg or more.

The sensitivity of the ECG for diagnosing RVH in patients with chronic autopsy-proven cor pulmonale is about 60 to 70 percent, whereas in clinical studies the rate of recognition may be only 28 percent. With moderate RVH the precordial leads display deep S waves in leads V5 and V6, but the R waves in the right precordial leads are not prominent. In the most severe cases of cor pulmonale, the QRS forces are oriented anteriorly and rightward, and tall R waves appear in the right precordial leads (Figure 3–20). Patients with cor pulmonale secondary to pulmonary thromboembolism, idiopathic pulmonary hypertension, or obesity hypoventilation syndrome are more likely to have a tall R wave in V1 than are those with pulmonary emphysema (Figures 3–21 and 3–22). The latter, however, more often have deep S waves in leads V4, V5, and V6.

**RVH IN OTHER CLINICAL CONDITIONS**

**Pulmonary Hypertension in Mitral Stenosis**

In the absence of RVH secondary to pulmonary hypertension, the only ECG abnormality in patients with mitral stenosis is left atrial enlargement. In the experience of Chou, the criteria for an RVH diagnosis, in descending order of frequency, are an R/S ratio in V1 > 1; a delay of onset of the intrinsicoid deflection in lead V1; RV1 + 5V5,6 > 1.05 mV; S wave in V1 < 0.2 mV; R in V1 > 0.7 mV; R/S ratio in V5 or V6 ≤ 1; right axis deviation ≥ +110 degrees; rSR’ in V1; RaVR ≥ 0.5 mV; and qR in V1. Although exceptions often occur, there is correlation between the degree of pulmonary hypertension in mitral stenosis and the appearance of RVH on the ECG. Fowler and associates found that when the mean pulmonary arterial pressure exceeded 42 mmHg, nearly all the patients exhibited a hypertrophy pattern, as indicated by an abnormal R/S ratio with delayed intrinsicoid deflection in lead V1. Such evidence was not found in patients with a pulmonary artery pressure < 28 mmHg. Others have shown

![Figure 3–20](image-url)
that when the ECG displayed evidence of RVH, the mean pulmonary arterial pressure was uniformly 33 mmHg or higher, and the total pulmonary vascular resistance exceeded 800 or 1000 dynes·sec$^{-1}$·cm$^{-5}$. The finding of a mean QRS axis that exceeded $+90$ degrees is thought by some to indicate moderate or severe pulmonary hypertension, whereas incomplete RBBB or an rSR' pattern is not a reliable indicator of the severity of the vascular obstruction. A
monophasic R wave or qR complex in lead V1 with T wave inversion is uncommon in patients with mitral stenosis, but its presence signifies advanced disease.

**Congenital Heart Disease**

The ECG diagnosis of RVH in congenital heart disease is generally accurate in more than 90 percent of cases. The higher rate of RVH recognition in congenital heart disease than in acquired heart disease is due mainly to a higher right ventricular systolic pressure, greater right ventricular thickness, and the frequent absence of LVH in the congenital heart disease group.

The relation between the ECG findings of RVH and the hemodynamic findings has been studied in patients with isolated pulmonary stenosis by several investigators. According to Burch and DePasquale, the ECG is normal in about 50 percent of patients with mild pulmonary stenosis and a peak right ventricular systolic pressure <60 mmHg. When the right ventricular pressure is elevated moderately or severely, most patients have an RVH pattern. A monophasic R wave or qR complex with T wave inversion in V1 usually is seen in patients with severe lesions, whereas the less typical rSR1 pattern is encountered more often in those with mild disease (see Figure 3–18).

With an atrial septal defect, the basic pattern of rSR1 in lead V1 is common in patients who have mean pulmonary arterial pressures <20 mmHg, whereas most patients with a qR or rSR1S3 complex have higher pressures. Additional discussion of the ECG findings with an atrial septal defect is included in Chapter 12.

**Combined Ventricular Hypertrophy**

Partial cancellation of oppositely directed forces generated by LVH and RVH may result in a normal ECG pattern. For example, the development of RVH due to pulmonary hypertension sometimes obscures the previous LVH pattern. More often, however, recognition of the concurrent presence of LVH and RVH is possible because of the asynchrony of ventricular depolarization and because the semidirect precordial leads preferentially reflect the local potentials underlying the respective electrodes. A combined pattern of RVH and LVH is frequently present in patients with ventricular septal defect or patent ductus arteriosus in the presence of pulmonary hypertension (Eisenmenger syndrome). In such cases, tall R waves may be present in both left and right precordial leads with a tall biphasic QRS in the mid-precordial leads (Katz-Wachtel pattern).

Right axis deviation in the frontal plane in the presence of an LVH pattern suggests associated right ventricular enlargement. A less reliable indicator of possible right ventricular dilation in the presence of an LVH pattern is a shift of the transition zone in the precordial leads to the left. In adults with rheumatic heart disease, biventricular hypertrophy may be suspected in the presence of tall R waves in the left precordial leads and disproportionately small S waves (i.e., <1 mV in lead V5) or inverted T waves in the right precordial leads. Such a pattern is characteristically present in patients with mitral stenosis and pulmonary hypertension who also have mitral regurgitation or aortic valve disease.

**Clinical and Anatomic Correlations**

One of the following ECG criteria has been applied in a limited number of correlation studies:

1. ECG pattern meets one or more of the diagnostic criteria for isolated RVH or LVH.
2. Precordial leads show signs of LVH, but the QRS axis in the frontal plane is more than +90 degrees.
3. The R wave is greater than the Q wave in lead aVR, and the S wave is greater than the R wave in lead V5, with T wave inversion in lead V1 in conjunction with signs of LVH.

In general, these criteria have low sensitivity for recognizing combined ventricular hypertrophy. In 172 cases of anatomic combined ventricular hypertrophy studied by four groups of investigators, the ECG was diagnostic in only 17 percent of cases. It showed signs of isolated LVH or RVH in 28 percent of the cases. The secondary RVH in patients with left-sided heart disease is usually masked by the dominant LVH (Figure 3–23). Conversely, LVH, which is often seen in patients with chronic cor pulmonale, usually is not detectable on the ECG (Figure 3–24).

The specificity of the ECG diagnosis of combined ventricular hypertrophy is also limited. In a small autopsy series examined by Chou, only 10 of 22 cases (45 percent) with an ECG diagnosis of combined hypertrophy had anatomic combined ventricular hypertrophy, with
the remainder showing isolated LVH. Jain et al.\textsuperscript{147} studied 69 patients with biventricular hypertrophy identified by echocardiography. Among them, 17 (25 percent) had ECG findings compatible with biventricular hypertrophy, 25 (36 percent) had an LVH pattern, and 14 (20 percent) had an RVH pattern. An S wave in leads V\textsubscript{5} and V\textsubscript{6} >0.7 mV was the most frequent finding in the 17 patients with the ECG criteria for biventricular hypertrophy. The sensitivity of the ECG criteria for biventricular hypertrophy was 24.6 percent and the specificity was 86.4 percent. Figures 3–23 to 3–26 illustrate the spectrum of ECG changes seen in patients with anatomic combined ventricular hypertrophy.

Figure 3–23 Anatomic combined ventricular hypertrophy with a left ventricular hypertrophy (LVH) pattern on the ECG in a 40-year-old man with advanced hypertensive cardiovascular disease and severe congestive heart failure. At autopsy the heart weighed 550 g, with marked hypertrophy and dilatation of all the heart chambers. The ECG shows left atrial enlargement and LVH. No evidence is seen of right ventricular hypertrophy.

Figure 3–24 Combined ventricular hypertrophy with a right ventricular hypertrophy (RVH) pattern on the ECG in a 25-year-old extremely obese woman (400 lb). She was believed to have the pickwickian syndrome with chronic cor pulmonale and recurrent pulmonary embolism. At autopsy the heart weighed 615 g, with marked RVH and moderate left ventricular hypertrophy (LVH). The right ventricular wall was 9 mm thick, and the left ventricular wall was 19 mm thick. There also was marked right atrial hypertrophy and dilatation. On the ECG the P waves are consistent with right atrial enlargement. An abnormal right axis deviation with deep S waves in the left precordial leads suggests RVH. There is no evidence to suggest coexisting LVH.
Figure 3–25  Rheumatic heart disease with combined ventricular hypertrophy in a 29-year-old man with severe aortic and mitral valve disease proved at cardiac catheterization and surgery. On the ECG the P waves are consistent with biatrial enlargement. The frontal plane QRS axis is +90 degrees. This finding, in the presence of signs of left ventricular hypertrophy, suggests coexisting right ventricular hypertrophy.

Figure 3–26  Combined ventricular hypertrophy demonstrated on the ECG in a 63-year-old man with severe mitral and aortic stenosis and mild aortic insufficiency. At autopsy the heart weighed 850 g, with hypertrophy and dilatation of all chambers. The left ventricular wall was 20 mm thick, and the right ventricular wall was 9 mm thick. Coronary arteries were patent, and there was no evidence of myocardial infarction. The ECG shows atrial fibrillation. The abnormal right axis deviation and the RSR' pattern in lead V1 suggest right ventricular hypertrophy. The voltage of the R waves and the ST segment and T wave changes in the left precordial leads suggest left ventricular hypertrophy. Some of the ST and T changes are the result of a digitalis effect.

REFERENCES


