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AHA/ACCF/HRS Recommendations for the Standardization and Interpretation of the Electrocardiogram

Part V: Electrocardiogram Changes Associated With Cardiac Chamber Hypertrophy

A Scientific Statement From the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society

Endorsed by the International Society for Computerized Electrocardiology

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The detection and assessment of cardiac chamber hypertrophy has long been an important objective of clinical electrocardiography. Its importance has increased in recent years with the recognition that hypertrophy can be reversed with therapy, and that by doing so, adverse clinical outcomes can be prevented or delayed (1,2).

(Note: This report uses the term *hypertrophy* rather than *enlargement*. The 1978 Bethesda Conference favored use of the term enlargement, but hypertrophy is more commonly used in recent research reports, although not necessarily in textbooks. Enlargement may be taken to imply an increase in chamber dimension, which may not be present in concentric hypertrophy. It is doubtful whether enlargement occurs without hypertrophy, at least in chronic stable syndromes. As discussed below, distinctive P-wave abnormalities may occur in the absence of atrial hypertrophy or dilation.)

The principal electrocardiogram (ECG) changes associated with ventricular hypertrophy are increases in QRS amplitude and duration, changes in instantaneous and mean QRS vectors, abnormalities in the ST segment and T waves, and abnormalities in the P wave. These changes have been correlated with direct or indirect assessments of ventricular size or mass to establish electrocardiographic criteria for the diagnosis of hypertrophy.

Originally, measurement of ventricular mass at autopsy or the clinical features of the patients were the reference standards used to establish ECG criteria. Later, the ECG

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changes were referenced against findings from various imaging modalities such as chest radiography or left ventriculography. In recent years, 2-dimensional echocardiography has become the favored reference standard, but it is now being challenged by 3-dimensional echocardiography, computerized tomography, and magnetic resonance imaging (3). Although these newer imaging techniques provide a more accurate assessment of ventricular myocardial mass than does the ECG, they do not obviate the clinical use of the ECG. The greater convenience and lower cost of the ECG continue to support its widespread use for the diagnosis of ventricular hypertrophy in clinical practice, epidemiological studies, and clinical trials. In addition, some ECG abnormalities have been shown to have independent clinical prognostic value.

The evolution of these new methods provides a compelling reason to reassess the role of the ECG in detecting cardiac hypertrophy and related abnormalities and to update our practice on the basis of new research findings and technological developments.

Left Ventricular Hypertrophy

Diagnostic Criteria Based on QRS Voltage

The most commonly used diagnostic criteria for left ventricular hypertrophy (LVH) are based on measurements of QRS voltages. The ECG criteria for LVH shown in Table 1 have evolved over the years. Criteria were originally based on R and S amplitudes in standard limb leads I and III, using clinical and autopsy data as reference standards (4–6). (Amplitudes of ECG complexes are referred to in millimeters, rather than millivolts. Using normal standardization, 10 mm equals 1 mV; 1 mm equals 0.1 mV.) Many other voltage criteria were introduced after the general acceptance of the standard 12-lead ECG, most notably by Sokolow and Lyon (7), who in 1949 introduced the widely used criterion based on the sum of S_{V1} and R_{V5} or R_{V6}. More recently, the sum of S_{V3} and R_{aVL}, referred to as the "Cornell voltage," has been used (8). The point score of Romhilt and Estes, introduced in 1968, incorporates abnormalities in QRS axis and duration, QRS onset-to-peak time, and P and ST-T morphology, in addition to QRS amplitude (9).

More recently, more complex criteria that are easily implemented with computerized recording and interpretation systems have been developed. These include indices based on products of voltage and QRS duration (10), computation of QRS area (11), composite use of several criteria (12), and indices based on scores derived from regression equations that incorporate multiple electrocardiographic and nonelectrocardiographic factors (13,14).

The existence of many different criteria for diagnosing LVH makes clinical application more complex. The sensitivity of the various criteria is generally quite low (usually less than 50%), whereas the specificity is quite high (often in the range of 85% to 90%) (15). However, the sensitivity and specificity of each criterion is different. Thus, the diagnostic accuracy will depend on the specific criterion used. Because of these differences in sensitivity and specificity, patients who meet one set of criteria for LVH commonly do not meet other criteria. In a large group of patients with mild or moderate hypertension, only 11.2% of patients with LVH by either the Cornell voltage criterion or the Sokolow-Lyon criterion had LVH diagnosed by both criteria (16). In addition, the various criteria have different positive and negative predictive values in different patient populations (17), suggesting that the value of multiple criteria may be additive.

Published studies are currently insufficient to indicate whether any of the more recently proposed criteria are clearly superior to the others or are simply redundant. The data do suggest that interpretations should specify which criteria are used in making a diagnosis and that automated systems should apply multiple criteria. Furthermore, because the accuracy of the criteria is empirical, that is, dependent on correlations between specific ECG measurements and a reference standard, only ECG criteria that have been formally tested should be used without modification from the tested form.

One important issue in developing and applying diagnostic criteria for LVH based on QRS voltage is that QRS voltages are influenced by a variety of factors other than left ventricular size or mass. These factors include age, gender, race, and body habitus. Their effects may contribute to the limited accuracy of the ECG criteria. Day-to-day variability and variability resulting from variations in the sites of electrode placement also impact QRS voltages and, hence, the diagnostic value of ECG voltage criteria.

Age

Apart from the wide variation in the normal limits of QRS voltage in infants and children of various ages, there are important differences between adults of various ages, with QRS voltages tending to decline with increasing age. In general, the commonly used QRS voltage criteria apply to adults older than 35 years (15). Standards for the 16- to 35-year age group are not as well-established, and the diagnosis of LVH based on voltage alone has a low accuracy in this age group. The diagnosis of LVH in highly trained athletes is especially problematic.

Gender

Adult women have a slightly lower upper limit of QRS voltage than men do, although S_{V3} is the only measurement with a large difference (18). The difference persists after adjustment for body size and cardiac mass. Some criteria have been shown to improve their performance with gender adjustment, but the adjustment is not the same for all criteria (8,13,18–20).

Race

Normal values of QRS voltages vary by race. African-Americans have a higher upper normal limit of QRS voltage than do Euro-Americans, whereas Hispanic Americans have lower limits. In patients with mild or moderate hypertension, the Sokolow-Lyon criterion has a higher sensitivity and lower specificity in African-Americans than in Euro-Americans, whereas the Cornell voltage criterion shows lower sensitivity and higher specificity in African-Americans than in Euro-Americans (19–23).

Body Habitus

Obesity is associated with increased left ventricular mass by echocardiographic measurement but not with increased QRS voltage. This may be attributed to the insulating effect of

Table 1. Criteria for Left Ventricular Hypertrophy

	Amplitude	First Author of Study	Year of Study Publication
Limb lead voltage			
(R I–S I)+(S III–R III)	>16 mm	Lewis (5)	1914
R I+S III	>25 mm	Gubner (6)	1943
R I	>15 mm	Gubner (6)	1943
R aVL	>11 mm	Sokolow (7)	1949
R aVF	>20 mm	Goldberger (65)	1949
Q or S aVR	>19 mm	Schack (73)	1950
R+S in any limb lead	>19 mm	Romhilt (9)	1968
Precordial lead voltage			
S V_1	>23 mm	Wilson (76)	1944
S V_2	>25 mm	Mazzoleni (69)	1964
S V_1+R V_5	>35 mm	Sokolow (7)	1949
S V_2+R $V_{5,6}$	>45 mm	Romhilt (72)	1969
S $V_{1,2}$+R $V_{5,6}$	>35 mm	Murphy (54)	1984
S $V_{1,2}$+R V_6	>40 mm	Grant (66)	1957
R+S any precordial lead	>35 mm	Grant (66)	1957
R V_5: R V_6	>1.0	Holt (67)	1962
R, any precordial lead	>26 mm	McPhie (70)	1958
S V_2+R $V_{4,5}$	>45 mm	Wolff (77)	1956
R V_5	>33 mm	Wilson (76)	1944
R V_6	>25 mm	Wilson (76)	1944
Combinations of limb and precordial voltage			
RS aVF+V_2+V_6 (>30 years)	>59 mm	Manning (68)	1964
RS aVF+V_2+V_6 (<30 years)	>93 mm	Manning (68)	1964
S V_3+R aVL (men)	>28 mm	Casale (8)	1985
S V_3+R aVL (women)	>20 mm	Casale (8)	1985
Total 12-lead voltage	>175 mm	Siegel (74)	1982
Combinations of voltage and nonvoltage			
Voltage-STT-LAA-axis-QRS duration	Point score	Romhilt (9)	1968
(R aVL+S V_3)×QRS duration	>2436 mm/sec	Molloy (71)	1992
Total 12-lead voltage×QRS duration	>1742 mm/sec	Molloy (71)	1992
Criteria for use with left anterior fascicular block			
S V_1+R V_5+S V_5	>25	Bozzi (33)	1976
S $V_{1,2}$+R V_6+S V_6	>25	Bozzi (33)	1976
S III+max R/S any lead (men)	>30	Gertsch (32)	1988
S III+max R/S any lead (women)	>28	Gertsch (32)	1988
Criteria for use with right bundle-branch block			
Max R/S precordial lead (with LAD)	>29 mm	Vandenberg (75)	1991
S V_1	>2 mm	Vandenberg (75)	1991
R $V_{5,6}$	>15 mm	Vandenberg (75)	1991
S III+max R/S precordial (with LAD)	>40 mm	Vandenberg (75)	1991
R I	>11 mm	Vandenberg (75)	1991

Amplitudes are given in millimeters, where 1 mm=0.1 mV. LAD indicates left axis deviation.

adipose tissue and the greater distance from heart to the chest wall electrodes. The effect of obesity differs among the various ECG criteria. In a study of patients with mild or moderate hypertension, the Cornell voltage-duration product was more often in the LVH range in obese patients than in the nonobese, whereas the Sokolow-Lyon criterion was less often in the LVH range in obese patients (13,24–27).

The Diagnostic Role of QRS Duration

QRS duration is frequently increased in LVH. This is manifest by a diffuse increase in QRS duration or an increase in the time from onset of QRS to the R-wave peak in V_5 or V_6. The increased QRS duration may be attributed to the increased thickness of the left ventricle wall and to intramural fibrosis, which distorts and prolongs transmural impulse propagation.

When the electrocardiographic pattern of LVH with widened QRS is present, there may be loss of the septal Q wave, often with a slurred R-wave upstroke. In these cases, it is reasonable to diagnose associated incomplete left bundle-branch block, an entity that is commonly seen only in the presence of LVH. A progression from LVH alone to incomplete left bundle-branch block may be observed.

ST-T Abnormalities With LVH

The association of inverted T waves with increased work of the left ventricle was described in 1929 (28). The term "typical strain" was introduced in 1941 (29) and referred to a specific ST-T abnormality, which was attributed to an increased hemodynamic burden. It consisted of J-point depression, upwardly convex down-sloping depression of the ST segment, and asymmetrical inversion of the T wave. It is now appreciated that electrocardiographic LVH with ST-segment and T-wave abnormalities occurs in conditions that are not necessarily caused by increased hemodynamic work, as in patients with dilated or hypertrophic cardiomyopathies, and that lesser degrees of ST-T abnormalities than the "typical strain" pattern are associated with LVH. Thus, the terms "strain" and "typical strain" are discouraged, and the term "secondary ST-T abnormalities" is preferred. The presence of ST-T-wave abnormalities provides major support to a diagnosis of LVH that would otherwise be based only on increased QRS voltage, and there is evidence to suggest that the presence of ST-T abnormalities are associated with larger values for left ventricular mass and higher risks of cardiovascular complications and mortality than an increase in QRS voltage alone (30,31). However, the evidence is insufficient to indicate whether the "typical strain" pattern has more significant clinical implications than lesser ST-T abnormalities, whether ST-T abnormalities should be used to diagnose LVH in the absence of any QRS voltage criteria, or whether the presence of ST-T abnormalities should allow modification of QRS voltage criteria. These are important issues for further investigation.

Left Atrial Abnormalities With LVH

P-wave abnormalities that are known to be associated with left atrial dilatation, hypertrophy, conduction delay, or elevated pressure are frequently associated with LVH and have been used as diagnostic criteria. P-wave changes occur frequently in patients with hypertension, and they may be the earliest electrocardiographic sign of hypertensive heart disease. However, similar P-wave abnormalities often occur in the absence of LVH. For this reason, and because adequate clinical studies assessing the accuracy of this criterion, either alone or in combination with other criteria, have not been reported, P-wave abnormalities should only be used as a supporting criterion.

Left Axis Deviation With LVH

Left axis deviation may be associated with LVH. However, it is not known whether left axis deviation results from hypertrophy itself, a degree of left anterior fascicular block, or other factors that may underlie the tendency toward a more leftward axis with increasing age, even in the absence of hypertrophy. Thus, this ECG finding, like others considered in this section, may be used to support a diagnosis of LVH rather than to make the diagnosis.

Prolonged QT Interval

LVH is often associated with slight prolongation of the QT interval, but it is not known whether QT-interval prolongation has independent value as an electrocardiographic criterion for LVH or is simply secondary to prolongation of QRS duration. A slightly prolonged QT interval is consistent with but not diagnostic of LVH. Such prolongation can reflect longer transmembrane action potentials because of alterations in ion channels as part of the hypertrophic process. Further studies testing the added value of QT-interval, QRS-axis, and P-wave changes in identifying LVH may be worthwhile.

Diagnosis of LVH in the Presence of Intraventricular Conduction Defects (Delays) and Bundle-Branch Block

Left ventricular hypertrophy commonly occurs in heart diseases that also cause intraventricular conduction defects or delays (IVCDs). As both LVH and IVCDs alter QRS patterns, the existence of an IVCD may impact the accuracy of ECG criteria for LVH.

Left Anterior Fascicular Block

In left anterior fascicular block, the QRS vector shifts in a posterior and superior direction, resulting in larger R waves in leads I and aVL and smaller R waves but deeper S waves in leads V_5 and V_6. R-wave amplitude in leads I and aVL are not reliable criteria for LVH in this situation. Criteria that include the depth of the S wave in left precordial leads improve detection of LVH in the presence of left anterior fascicular block (32–34).

Left Bundle-Branch Block

Studies of the electrocardiographic diagnosis of LVH in the presence of complete left bundle-branch block (LBBB) have yielded conflicting results (35–41). Some have concluded that the diagnosis should not be attempted in this setting (35–37), whereas others believe that the diagnosis can be made (38–41). Estimations of specificity are affected by the relatively high prevalence of anatomic LVH in patients with LBBB, especially in autopsy series, where it may be 90% or more. The variable results may also reflect differing definitions of LBBB. Strict definitions, which require monophasic notched or plateau-topped R waves in leads I, aVL, V_5, and V_6, tend to show low sensitivity for LVH criteria (42). Broader definitions, which require only a QRS duration greater than 120 ms, slurred predominant R in left precordial leads, and slurred predominant S wave in the right precordial leads, probably include cases that could be classified as LVH with associated intraventricular conduction delay rather than LBBB. Because "complete" LBBB may often be not truly complete, and because the QRS duration in LVH can probably be greater than 120 ms without a localized lesion in the left bundle, the distinction between these two entities may be difficult to define (43). A left atrial P-wave abnormality

(38,39,41) and a QRS duration greater than approximately 155 ms, as well as precordial lead voltage criteria (35,38–42), tend to have relatively high specificity for LVH in the presence of LBBB. In patients meeting these specific criteria, it is reasonable to diagnose LVH, even though the sensitivity is low. Otherwise, the ECG diagnosis of LVH should not be attempted when LBBB is present.

Right Bundle-Branch Block

Right bundle-branch block (RBBB) reduces the amplitude of the S wave in the right precordial leads and tends to reduce the sensitivity of electrocardiographic criteria for LVH. The ancillary features of left atrial abnormality and left axis deviation have enhanced value for the diagnosis of LVH in the presence of RBBB (44–47). Several criteria have been proposed for use specifically in the presence of RBBB, including S_{V1} greater than 2 mm (0.2 mV), $R_{V5,6}$ greater than 15 mm (1.5 mV), and QRS axis to the left of −30, with S III+largest R/S in a precordial lead greater than 30 mm (3.0 mV). These criteria were reported to have sensitivities of 46% to 68% and specificities of 57% to 71% (47).

Issues of Terminology

Estimates of Probability

Qualifying diagnostic terms such as *probable* or *possible* or *consider* are subject to multiple interpretations and may be used to indicate that some criteria for LVH are met, but that the accuracy of these criteria is limited, or that the criteria almost meet the threshold values, but that LVH is still strongly considered because of other contravening variables, such as obesity. Each interpretation has different meanings to the reader and to the user of the ECG. Hence these terms should be used and interpreted with caution. Additional studies would be worthwhile to propose specific criteria for their use.

Diagnostic Terms

Over years of use, electrocardiographers have adopted various terms for certain ECG findings, many with limited usefulness and accuracy. The terms *systolic (pressure) overload* and *diastolic (volume) overload* have limited accuracy in patients with congenital heart disease and in adults, and their use is not recommended. As discussed above, the term *strain* originated in an older concept of an ST-T abnormality that was considered to reflect ventricular overwork but not necessarily hypertrophy. Its use should also be discontinued.

Special Issues in Children

Electrocardiographic detection of ventricular hypertrophy in children is largely based on QRS voltage abnormalities. The standards for QRS voltage are derived from studies of populations of clinically normal children. Studies are relatively few and do not always include referencing to body size, gender, or race. Correlation with echocardiograms is also limited, and reference standards from autopsy or magnetic resonance imaging are not available.

Standards derived from a population of Canadian children (48) are widely used in North America. More recent studies in Scottish children using a digital sampling rate of 500 samples per second (49) and in Dutch children using a sampling rate of 1200 samples per second (50) showed higher upper-normal voltage limits. When the higher sampling rates are used, the amplitude criteria in children should be adjusted.

Gender and racial differences in QRS voltage similar to those in adults exist in children older than 10 years. Adjustment for body habitus has not been adequately investigated.

The sensitivity of ECG criteria for LVH is low in children, as it is in adults. The ECG is best used in pediatrics as a screening tool to be correlated with other measurements for the assessment of hypertrophy.

Other Considerations

Several other factors influence the value of the ECG in detecting LVH. The sensitivity and specificity of various ECG criteria reflect issues related to the types of heart disease, anatomic patterns of LVH, and degrees of hypertrophy present in different patient populations. Okin et al (16) noted that in patients with mild or moderate hypertension, an increase in the product of the $S_{V3}+R_{aVL}$ voltage and the QRS duration characterized older patients who were obese and female, whereas an increase in the sum of S_{V1} and R_{V5} was more characteristic of patients who were younger, male, black, and nonobese. The accuracy will also be different in populations in which LVH is unlikely (with most positive tests being false positives) than in populations in which LVH is more likely, for example, groups of patients with significant hypertension, in which more negative results will be false negatives. It is also important to recognize that the characteristics of patient groups in whom the criteria were established may be different from those in whom the criteria are applied.

Recommendations

1. Interpretation of ECGs for LVH should use only validated criteria, without deviation from the validated formulas.
2. No single diagnostic criterion can be recommended for use compared with the others.
3. Computer systems should use all criteria that are supported by valid evidence for identifying LVH.
4. Interpretations should specify which diagnostic criteria were used and which were abnormal (and thereby, by exclusion, which were examined but not found to be abnormal).
5. Criteria should be adjusted for factors known to alter accuracy, including gender, race, and body habitus, when such criteria have been validated.
6. The terms strain, systolic, and diastolic should not be used in diagnostic statements related to LVH.
7. The terms probable, possible, and borderline should be used with caution.
8. Because the evidence is conflicting, the diagnosis of LVH in the presence of complete LBBB should be made with caution.

Recommendations for Further Study

Issues that require additional study before recommendations can be made include the following:

1. Development and testing of adjustments of major diagnostic criteria for gender, race, age, and body habitus;
2. Adjustment of major criteria for specific populations with varying prevalences of LVH, including (semi) quantitation of modifying terms such as possible and probable;
3. Development and testing of criteria for specific indications, for example, prognosis, screening, follow-up of therapy, and so on;
4. The added clinical value of the ECG when used in addition to other diagnostic methods;
5. The possible use of ST-T abnormalities typical of LVH to diagnose LVH in cases where voltage criteria of LVH are not met;
6. The utility of voltage and other criteria, such as QRS axis, left atrial abnormality, and QRS duration, in diagnosing LVH in the presence of LBBB;
7. Identification of criteria that consistently outperform other criteria and those that are only redundant;
8. For pediatric patients, possible improvement of criteria based on current sampling technology, wider demographic groups, and the use of more leads; and
9. The effect of day-to-day variation of voltage and other criteria on the validity of LVH criteria.

Right Ventricular Hypertrophy

Right ventricular hypertrophy (RVH) causes a displacement of the QRS vector toward the right and anteriorly and often causes a delay in the R-wave peak in right precordial leads. However, considerable degrees of RVH are often required to change the balance of right and left ventricular vectors, because the vector of left ventricular activation dominates the balance in the normal heart and even more so in the setting of LVH. Thus, the ability of the ECG to detect RVH may be expected to be low.

Numerous criteria mostly derived from the amplitude of R and S in leads I, V_1, V_6, and the R-wave peak time in V_1 have been proposed and are shown in Table 2. They have been correlated primarily with autopsy data, although some are based on clinical and hemodynamic identification of conditions that impose increased workloads on the right ventricle (51–54). The echocardiogram has also been used as a reference standard, but it is less definitive than in LVH because of the complex 3-dimensional shape of the right ventricle and the frequent difficulty of measuring the thickness of the right ventricular free wall.

Although the sensitivity of the electrocardiographic criteria for RVH is generally low, some criteria have high specificity and can be used to advantage in diagnostic schemes or to derive continuous variables (54,55). The greatest accuracy is in congenital heart disease, with intermediate accuracy in acquired heart disease and primary pulmonary hypertension in adults. The lowest accuracy occurs in chronic lung disease.

Electrocardiographic RVH, particularly in congenital heart disease, has often been classified on the basis of contrasting ECG patterns. One pattern is similar to that of incomplete RBBB, suggesting volume overload, and a second pattern consists of predominantly tall R waves (as part of Rs, R, or Qr complexes) in right precordial leads, suggesting pressure overload. Both patterns are associated with right axis deviation. Both are also frequently associated with ST depression and T-wave inversion in right precordial leads; as with LVH, these ST-T abnormalities are better referred to as "secondary ST-T abnormality" than as "strain." In patients with chronic nonobstructive lung disease, there is often right axis deviation and deep S waves in the precordial leads.

Chronic obstructive pulmonary disease often causes a characteristic electrocardiographic pattern that reflects mainly the low diaphragm resulting from the increased lung volume. This pattern includes low voltage in the limb leads; a frontal plane QRS axis that is rightward, superior, or indeterminate; a rightward P-wave axis (i.e., greater than 60 degrees); persistent S waves in all precordial leads; and low R-wave amplitude in V_6 (56). RVH is suggested, in the presence of the chronic obstructive pulmonary disease pattern, only if R-wave amplitude in V_1 is relatively increased.

Right axis deviation and prominent anterior forces in the right precordial leads should be required for the electrocardiographic diagnosis of RVH in nearly all cases. On the other hand, such features occur for various reasons other than RVH, including a not-infrequent normal variant. The use of ancillary clinical information, therefore, plays a greater role in the appropriate use of the ECG for the purpose of recognizing RVH than it does in the case of LVH or the atrial abnormalities.

Table 2. Criteria for Right Ventricular Hypertrophy

	Amplitude	First Author of Study	Year of Study Publication
Tall R V_1	>6 mm	Myers (78)	1948
Increased R:S ratio V_1	>1.0	Myers (78)	1948
Deep S V_5	>10 mm	Myers (78)	1948
Deep S V_6	>3 mm	Myers (78)	1948
Tall R aVR	>4 mm	Sokolow (7)	1949
Small S V_1	<2 mm	Myers (78)	1948
Small R $V_{5,6}$	<3 mm	Myers (78)	1948
Reduced R:S ratio V_5	<0.75	Myers (78)	1948
Reduced R:S ratio V_6	<0.4	Myers (78)	1948
Reduced R:S V_5 to R:S V_1	<0.04	Sokolow (7)	1949
(R I+S III)–(S I+R III)	<15 mm	Lewis (5)	1914
Max R $V_{1,2}$+max S I, aVL–S V_1	>6 mm	Butler (51)	1986
R V_1+S $V_{5,6}$	>10.5 mm	Sokolow (7)	1949
R peak V_1 (QRS duration <0.12 sec)	>0.035 sec	Myers (78)	1948
QR V_1	Present	Myers (78)	1948
Supporting criteria			
RSR V_1 (QRS duration >0.12 sec)	Present		
S>R in I, II, III	Present		
S I and Q III	Present		
R:S V_1>R:S $V_{3,4}$	Present		
Negative T-wave V_1 through V_3	Present		
P II amplitude	>2.5 mm		

Amplitudes are given in millimeters, where 1 mm=0.1 mV.

Table 3. Pediatric Criteria for Left Ventricular Hypertrophy (Age-Related)

	Voltage (mm)				
	Age 0–7 d	Age 7 d–1 y	Age 1–3 y	Age 3–5 y	Age >5 y
RV_6	>12	>23	>23	>25	>27
SV_1	>23	>18	>21	>22	>26
SV_1+R V_6	>28	>35	>38	>42	>47

Based on Davignon et al (48). Amplitudes are given in millimeters, where 1 mm=0.1 mV.

Table 4. Pediatric Criteria for Right Ventricular Hypertrophy (Age-Related)

	Voltage (mm)				
	Age 0–7 d	Age 7 d–1 y	Age 1–3 y	Age 3–5 y	Age >5 y
R V_1	>27	>22	>18	>18	>13
S V_6	>10	>10	>7	>6	>4
R V_1+S V_6	>37	>43	>30	>24	>17

Based on Davignon et al (48). Amplitudes are given in millimeters, where 1 mm=0.1 mV.

Recommendations

1. No single criterion or limited set of criteria can be recommended for use exclusive of other validated criteria. The effect of using larger numbers of criteria on sensitivity and specificity should be further studied.
2. Criteria should be adjusted for age, gender, race, and body habitus.
3. Probability estimates for RVH should be adjusted in the light of available clinical diagnoses suggesting congenital heart disease, valvular heart disease, or chronic pulmonary disease. Incorporation of such clinical diagnoses into computer algorithms should be explored.

Biventricular Hypertrophy

Hypertrophy of both the right and left ventricle is relatively common in patients with heart disease of many types. Its recognition by ECG has a particularly low sensitivity, explained at least in part by the cancellation of increased QRS vectors of both RVH and LVH. In the presence of ECG criteria for LVH, the presence of prominent S waves in V_5 or V_6, right axis deviation, unusually tall biphasic R/S complexes in several leads, and signs of right atrial abnormality are useful signs that RVH may also be present (57,58).

In patients with congenital heart defects and RVH, the presence of combined tall R waves and deep S waves in leads V_2 to V_4, with combined amplitude greater than 60 mm (6.0 mV), suggests the presence of LVH.

Recommendations

1. Biventricular hypertrophy should be suggested on the basis of the presence of accepted criteria for both RVH and LVH. The low sensitivity of such patterns should be noted.
2. Right axis deviation in the presence of electrocardiographic LVH and tall biphasic R/S complexes in several leads should be recognized as suggestive of biventricular hypertrophy.

Atrial (P-Wave) Abnormalities

Abnormalities in the P wave that are related to anatomic or physiological abnormalities in the right or left atrium have been recognized since the early years of electrocardiography. The terms *P-mitrale*, *P-congenitale*, and *P-pulmonale* were later replaced by *left atrial enlargement* and *right atrial enlargement*, as it was realized that different clinical conditions caused similar abnormalities. However, other terms such as *atrial hypertrophy*, *atrial overload*, *atrial strain*, and *interatrial (or intraatrial) conduction defect* have also been used, reflecting the fact that atrial dilatation, atrial muscular hypertrophy, elevated atrial pressure, impaired ventricular distensibility, and delayed intraatrial conduction all seem to play a role in causing P-wave abnormalities. Because the effects of these several factors on the P wave may often appear in combination and may not be distinguishable, the less specific terms *left atrial abnormality* and *right atrial abnormality* are preferable.

Left Atrial Abnormality

Left atrial abnormality usually involves prolongation of the total atrial activation time, because left atrial activation begins and ends later than right atrial activation. Delay in the left atrial activation tends to cause a double-peaked or notched P wave, because the right and left atrial peaks that are normally nearly simultaneous and fused into a single peak become more widely separated. Activation of the left atrium has a more leftward and posterior vector than that of the right atrium. The product of the amplitude and the duration of the terminal negative component of the P wave in lead V_1 (the P terminal force) has been used most frequently of the various criteria for left atrial abnormality, but the P-wave duration (120 ms or more) and widely notched P wave (40 ms or more) appear to have equal value. Several other criteria, including left axis of the terminal P wave (−30 to −90), and possibly the P-wave area, are also useful (59,60). A purely negative P wave in V_1 is suggestive but can occur without an increased P terminal force.

Prolonged activation time of the atrium, indicated by the total duration of the P wave of 120 ms or more, is present in a large majority of patients with electrocardiographic signs that are considered to represent left atrial abnormalities (61). Conduction delay is more closely linked to left atrial abnormality than to right atrial abnormality, probably because it often represents delay in the specialized interatrial pathway (Bachmann's bundle) (62,63) and possibly within the left atrial myocardium as well. The more general term *intraatrial* is therefore preferable to *interatrial*, even though the delay might in fact be primarily interatrial.

Right Atrial Abnormality

Right atrial abnormality is typically manifested as an increase in amplitude of the P wave and a tendency to rightward shift of the P-wave vector (64). A tall upright P wave in lead II (greater than 2.5 mm) is characteristic, often with a peaked or pointed appearance that presumably reflects summation of the enhanced right atrial component with the simultaneous left atrial component. Right atrial abnormality increases the amplitude of the initial P-wave forces, contrasting with increase in the later P-wave forces that can result from left atrial abnormality (pseudo-P pulmonale). Prominent initial positivity of the P wave in V_1 or V_2 (1.5 mm [0.15 mV] or more) also indicates right atrial abnormality. Rightward axis of the P wave and a peaked form without increased amplitude are supportive signs. Total P-wave duration is usually normal, but an exception occurs in patients with surgically repaired congenital heart disease (especially those with single-ventricle physiology) where significant P-wave prolongation occurs and is a risk factor for the development of atrial tachyarrhythmias.

Combined Atrial Abnormality

Combined atrial abnormality is indicated essentially by the presence of some of the features of both right atrial and left atrial abnormality. However, little evidence is available regarding the accuracy of ECG criteria for combined atrial abnormality.

Recommendations

1. Abnormal P waves should usually be referred to as right or left "atrial abnormality" rather than enlargement, overload, strain, or hypertrophy.
2. Multiple electrocardiographic criteria should be used to recognize atrial abnormalities.
3. Intraatrial conduction delay should be recognized as a category of atrial abnormality applicable particularly to instances where P-wave widening is not accompanied by increased amplitude of right or left atrial components.

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*Modest.

†Significant.

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*Modest.

†Significant.

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