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COOPERATIVE STUDIES

Criteria for Intraventricular Conduction Disturbances and Pre-excitation

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In an effort to standardize terminology and criteria for clinical electrocardiography, and as a follow-up of its work on definitions of terms related to cardiac rhythm, an Ad Hoc Working Group established by the World Health Organization and the International Society and Federation of Cardiology reviewed criteria for the diagnosis of conduction disturbances and pre-excitation. Recommendations resulting from these discussions are summarized for the diagnosis of complete and incomplete right and left bundle branch block, left anterior and left posterior fascicular block, nonspecific intraventricular block, Wolff-Parkinson-White syndrome and related pre-excitation patterns. Criteria for intraatrial conduction disturbances are also briefly reviewed.

The criteria are described in clinical terms. A concise description of the criteria using formal Boolean logic is given in the Appendix. For the incorporation into computer electrocardiographic analysis programs, the limits of some interval measurements may need to be adjusted.

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In recent years at international meetings and through concerted actions, several investigators (1–6) have made an appeal for standard rules of measurement, classification and description for electrocardiographic features. Such standards are desirable to improve patient care and disseminate medical knowledge and experience. Patient care can be improved by enhancing the consistency and quality of the electrocardiographic report and, thus, facilitating communication between the interpreter and the user. Since other groups specifically have addressed the problems of standardization of terminology (1–2) and measurement (5–6), the present Task Force was established to examine the possibility of standardization of diagnostic classification criteria.

At the 10th Bethesda Conference on Optimal Electrocardiography (1), it was proposed to categorize diagnostic electrocardiographic statements into three categories (Table 1):

1) *Type A statements* refer to an anatomic lesion or pathophysiologic state, such as hypertrophy, infarction, ischemia, pulmonary disease, drug and metabolic effects, and which can be verified by nonelectrocardiographic evidence;

2) *Type B statements* refer to an anatomic or functional disturbance, such as arrhythmias and conduction defects, and which are detectable mainly by the electrocardiogram itself; and

3) *Type C statements* refer to electrocardiographic features that do not fit into type A or B categories and often are merely descriptive, such as nonspecific ST-T changes, electrical axis deviation and low QRS voltage.

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JACC Vol. 5, No. 6
June 1985:1261–75

It is widely recognized that an optimal selection and evaluation of criteria for type A statements should be based on a library of cases in which the condition has been determined or excluded by independent nonelectrocardiographic techniques. Both type B and type C statements, however, are primarily detected on the electrocardiogram itself, either from surface or intracardiac leads. Since the elaboration of a data base for the evaluation of type A statements requires a large collaborative effort, this Task Force has assigned its objectives toward the standardization of Type B statements. As a follow-up to its work on rhythm statements (2), the aim of the Task Force was to reach a consensus on criteria for conduction disturbances and pre-excitation (Table 2).

Table 2. Categories of Intraventricular Block

Bundle branch block
Complete right
Complete left
Incomplete right
Incomplete left
Fascicular block
Left anterior
Left posterior
Bi- and trifascicular blocks
Nonspecific intraventricular block
Pre-excitation
Wolff-Parkinson-White pattern (syndrome)
Other pre-excitation patterns
Intraatrial block

Recommendations for Some Derived Electrocardiographic Measurements

The reader is referred to specific reports of the American Heart Association Committee on Electrocardiography (7) and the Working Party on Common Standards for Quantitative Electrocardiography (5–6) for recommendations on basic nomenclature for the P-QRS-T complex, wave definitions and primary electrocardiographic measurements. Some additional recommendations to these reports are needed with respect to some derived measurements.

Electrical axis of the QRS complex. An instantaneous electrical axis represents the direction of the electrical forces at any given instant, whereas the mean electrical axis refers to the average direction of the activation or repolarization process during the cardiac cycle. Instantaneous and mean electrical axes may be determined for any deflection (P, QRS, ST-T) in the three planes (frontal, transverse and sagittal) as well as spatially. The determination of the electrical axis of a QRS complex is useful for the diagnosis of certain intraventricular conduction disturbances.

An average axis is meaningful when the QRS complex has one dominant deflection and when other deflections are of small amplitude. Whenever the QRS complex consists of two or more deflections of comparable amplitude in more than one standard limb lead, an axis should be determined for each of these deflections.

Table 1. Three Categories of Electrocardiographic Statements*

1. Type A, documentable by nonelectrocardiographic means
Infarction/injury
Hypertrophy/enlargement/overload
Chronic obstructive lung disease/pulmonary emphysema
Metabolic/digitalis/other drug effects
Ischemia (possibly documentable)
2. Type B, detectable primarily by electrocardiography
Rhythm disturbances including pacemaker rhythms and artifacts
Conduction disturbances
3. Type C, morphologic descriptive statements
Axis deviation
Low or increased QRS voltage
Nonspecific ST-T changes, strain, large T waves

*Adapted from Rautaharju PM, et al. (4) with permission.

Area method. The net amplitude and direction of the QRS complexes in any two of the standard bipolar leads (I, II, III) or augmented leads (aVR, aVL, aVF) has been used most often for the determination of the mean electrical axis in the frontal plane (8–10). However, to be accurate, one should utilize the net area rather than the amplitude of the various components of the QRS complex (11–13). The areas of these deflections are first added algebraically and subsequently projected on the sides of the Einthoven triaxial or hexaxial reference system. The axis is then determined at the intersection and expressed in polar coordinates as specified in the American Heart Association report (7). All computer electrocardiographic analysis programs should utilize the area method. For the calculation of the areas, the same QRS group onset and offset should be used for each lead, with the QRS onset being defined as the onset of the earliest deflection and the QRS offset as the latest end in any of the simultaneously recorded three or more leads. Because of Einthoven's law, which stipulates that leads I + III = II, the same mean QRS axis should theoretically be obtained from any pair of simultaneously recorded bipolar standard leads. However, to increase reproducibility in the presence of noise or low voltage, averaging of axes determined by different lead combinations may be recommended for computer processing.

Intrinsicoid deflection versus R peak time. The definition and interpretation of the term "intrinsicoid deflection" have changed over the years. Initially, only the term "intrinsic deflection" was used to indicate the instant at which the area of cardiac muscle immediately below a unipolar epicardial electrode was completely depolarized (8). Later, this concept was extended to the precordial leads, for which the term intrinsicoid deflection was introduced. Some authors (13) have also applied the term to the limb

leads. They regard the intrinsicoid deflection as representing the turning point of the cardiac vector along the lead axis. However, since by definition the intrinsicoid deflection can only be measured on unipolar precordial leads, its use in the limb leads should be discouraged. For all practical purposes in the evaluation of conduction disturbances, the term "R peak time" is preferred both for the limb and precordial leads.

The R peak time in a specific lead is the interval from the earliest onset of the QRS complex, preferably determined from multiple simultaneously recorded leads, to the peak (maximum) of the R wave or R′ if present (Fig. 1). If the R peak is notched, the R peak time is measured to the second peak, eventually following the maximum of the R wave. This definition deviates from the recommendations given in the Minnesota Code (10). In the right-sided precordial leads V_1 or V_2, this interval is normally 0.04 second or less, whereas in the left-sided leads V_5 or V_6, it usually does not exceed 0.05 second in adults and 0.04 second in children younger than 14 years of age.

Axis deviation. Various arbitary ranges have been reported for the definition of axis deviations (14). Some authors considered values from 0 to $+90°$ as being normal for adults, from 0 to $-90°$ as left-axis deviation and from $+90$ to $-90°$ as right-axis deviation. Other values suggested for left-axis deviation were from $+30$ to $-120°$. At present, left-axis deviation is generally diagnosed when there is an axis shift in the frontal plane of the scalar electrocardiogram from -30 to $-90°$; right-axis deviation is diagnosed from $+90$ to $+180°$. Between -90 and $+180°$, the axis deviation is undetermined.

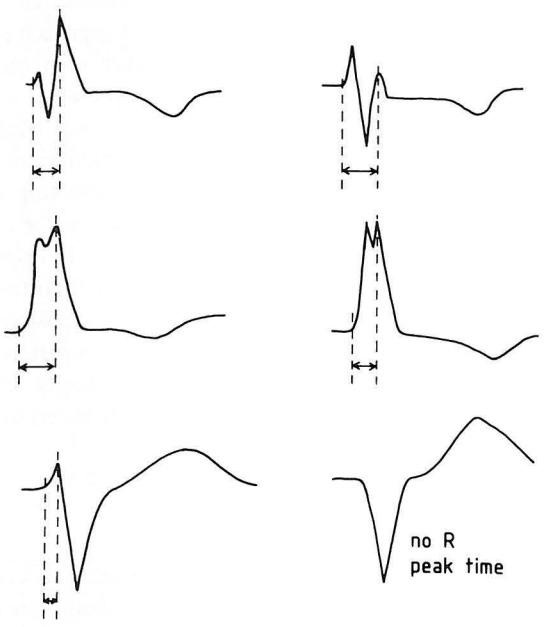

Figure 1. Illustration of measurement of the R peak time.

Brief Review of Normal Intraventricular Conduction

Although controversy persists regarding the anatomy (15) and function of the intraventricular conduction system, knowledge of the former is essential. When this is combined with what we know of the time course of the excitatory process in the normal heart, it is also helpful in the understanding of electrocardiographic findings in conduction disturbances (16–19).

Trifascicular conduction system. In light of the work of others (20–22) over preceding decades, Rosenbaum et al. (17) stressed the clinical relevance of the concept that the intraventricular conduction network can be regarded as a trifascicular system consisting of the right bundle branch and two divisions of the left bundle branch. The cord-like right bundle branch is the continuation of the bundle of His, and proceeds subendocardially along the right side of the interventricular septum until it terminates in the Purkinje plexuses of the right ventricle. Although the left bundle branch also courses subendocardially but on the left side of the septum, it is composed of multiple fascicles which Rosenbaum et al. (17) grouped into two main divisions, namely, the anterosuperior division and the posteroinferior division, respectively. A third medial or centroseptal division supplies the mid-septal area of the left ventricle and arises either from the main left bundle branch or from the anterior or posterior radiations, or both (18); these three subdivisions of the left bundle are usually extensively interconnected.

Endocardial activation. The three left-sided terminations correspond closely to the endocardial areas synchronously excited early (0 to 5 ms) after the commencement of the left ventricular cavity potential, as demonstrated in the isolated human heart by Durrer et al. (23). These investigators showed that endocardial activation of the right ventricle started near the insertion of the anterior papillary muscle, 10 ms after the onset of the left ventricular cavity potential.

Septal and ventricular activation. Septal activation is not only from left to right, but also predominantly from apex to base, and the inner layers of both ventricles are normally excited very soon after depolarization of the septum by the rapid spread of conduction through the Purkinje network. Further depolarization occurs centrifugally from endocardium to epicardium as well as tangentially. The earliest epicardial breakthrough occurs in the right ventricle in the area pretrabecularis, from which there is, overall, radial spread toward apex and base, the last part to be excited being the atrioventricular sulcus and pulmonary conus. Overall, the posterobasal paraseptal region or a more lateral location of the left ventricle is the last part of the heart to be depolarized.

JACC Vol. 5, No. 6
June 1985:1261–75

Vectorial approach. The sequence of ventricular activation has been approximated by a vectorial approach (11). The initial portion of the complex, whether seen on the electrocardiogram or vectorcardiogram, represents septal activation and is always directed anteriorly and, usually, rightward and superiorly. The larger left ventricular muscle mass results in left ventricular potentials dominating over those of the right ventricle in the body of the vector loop. Thus, the mean direction of the QRS vector is oriented leftward, inferiorly and posteriorly. The terminal portion of the loop, representing later activation, is directed posteriorly and either slightly to the left or the right, and superiorly or inferiorly. Deflections recorded in any lead simply reflect the degree to which the cardiac vectors are projected on the axis of that lead.

Definition of Terms

The following terminology has been generally accepted (1,2) with respect to ventricular conduction disturbances:

Block. This refers to a delay or failure of impulse propagation. Conduction disturbances of various degrees may occur in different locations within the heart, and may reflect delay or failure of propagation in either the anterograde or retrograde direction, or both.

Bundle branch block. This is considered to be a delay or failure of conduction within one of the bundle branches. It may be complete or incomplete, permanent, transient or intermittent in one or more branches. Bundle branch block can only be diagnosed in the presence of supraventricular rhythm and in the absence of pre-excitation patterns.

Complete bundle branch block. This pattern indicates the absence of conduction in a bundle branch, or conduction delay of such magnitude that ventricular activation occurs largely or exclusively through the contralateral bundle. There is absolutely no way to determine if bundle branch block is complete or not. However, as in previous reports by the Task Forces of the American College of Cardiology (1) and the World Health Organization and International Society and Federation of Cardiology (2), both the terms "complete" and "incomplete" bundle branch block will be used in the present report to describe the patterns of conduction delay. These definitions refer to electrophysiologic and electrocardiographic deductions and do not always indicate an organic pathologic lesion because defects may be electrical rather than structural, especially when intermittent. We concede the importance of better pathologic correlation with electrocardiographic appearances, but in their absence, we refer to the various types of block in electrocardiographic terms.

So-called complete bundle branch block causes widening of the QRS complex to 0.12 second or more in adults. The limit of 0.12 second is rather arbitrary, but has been used for a long time to diagnose complete bundle branch block. It is the result of pragmatic reading of the electrocardiogram, that is, it equals 3 mm at the conventional recording speed of 25 mm/s which can easily be distinguished on the millimeter paper grid. The QRS duration usually exceeds 0.14 second in most patients with complete bundle branch block. It may even exceed 0.20 second in the presence of myocardial disease, electrolyte disturbance or use of certain drugs. In young children, a QRS complex with a duration of less than 0.12 second may indicate "complete" block of a bundle branch. On the other hand, several investigators (24–26) have demonstrated that a QRS duration of 0.12 second in adults may indicate "incomplete" block.

Incomplete bundle branch block. This pattern indicates a delay in activation of a ventricle, resulting from delayed conduction within the ipsilateral bundle branch. The involved ventricle may be partially activated by the impulse from the contralateral bundle.

Fascicular block. This is an electrocardiographic (electrophysiologic) concept that ascribes certain abnormal waveforms of the QRS complex to particular patterns of disturbed intraventricular conduction. Accepted electrocardiographic patterns exist for left anterior and left posterior fascicular block. Although prominent anterior forces are thought to be suggestive, there still is no agreement on specific electrocardiographic features that would permit a reliable diagnosis of lesions of the mid-septal fascicle (27–29).

Nonspecific (unspecified) intraventricular block. This applies to any pattern of intraventricular conduction disturbance that cannot be ascribed to block in a specific portion (bundle branches or fascicles) of the specialized conduction system.

Wolff-Parkinson-White pattern. This applies to preexcitation of the ventricles by means of an additional anatomic atrioventricular connection. The Wolff-Parkinson-White syndrome requires an association of Wolff-Parkinson-White pattern with tachycardias (reentrant tachycardias or atrial flutter/fibrillation) (1,2).

Other terms with respect to ventricular conduction disturbances exist, but it is recommended (1,2) that they be avoided because they are either nonprecise or controversial.

Criteria for Complete Right Bundle Branch Block

Sequence of ventricular activation. In right bundle branch block, the delay or failure of impulse propagation occurs in the right bundle, causing delay of activation in the right ventricle, whereas the left ventricle is activated normally and early septal activation takes place in the normal left to right direction. The site of block may vary (11–13,19,30–34). Right ventricular activation proceeds for a large part by slow muscle to muscle conduction. This causes an increase in QRS duration to 0.12 second or longer due to widening and slurring of the terminal QRS deflections.

The initial part of the QRS complex remains unchanged in complete right bundle branch block. Changes in the QRS complex become manifest in the middle part, especially when left ventricular activation is near completion and the delayed abnormal activation of the right ventricular free wall begins. Late QRS forces resulting from activation of the right ventricle are no longer cancelled by left ventricular potentials, and produce slowly inscribed QRS vectors that are predominantly directed rightward and anteriorly. The abnormal sequence of ventricular activation is accompanied by a change in the course of repolarization, and generally also proceeds in a left to right direction. The ST and T vectors are, therefore, directed leftward and opposite to the terminal part of the QRS complex (19).

Diagnostic criteria. The diagnosis of uncomplicated complete right bundle branch block is made when the following criteria are met (see Appendix):

1) Prolongation of QRS to 0.12 second or more.

2) An rsr′, rsR′ or rSR′ pattern in lead V_1 or V_2. The R′ is usually greater than the initial R wave. In a minority of cases, a wide and notched R pattern may be seen.

3) Leads V_6 and I show a QRS complex with a wide S wave (S duration is longer than the R duration or greater than 40 ms in adults).

4) The R peak time should be greater than 0.05 second in lead V_1 and should be normal in leads V_5 and V_6.

Of these criteria the first three should be present for the diagnosis to be made. When a notched dominant R pattern is present in V_1, criterion 4 should be satisfied as well.

ST-T changes. In uncomplicated right bundle branch block, the ST-T segment is depressed and the T wave inverted or biphasic (− +) in leads V_1 and V_2, and the T wave is upright in leads I, V_5 and V_6. However, these ST-T changes should not be used as criteria for right bundle branch block.

Criteria for Incomplete Right Bundle Branch Block

When the electrocardiographic criteria for right bundle branch block 2 to 4, as just listed, are met and the QRS duration is less than 0.12 second, the diagnosis of incomplete right bundle branch block will be made. There is no minimal QRS duration for incomplete right bundle branch block.

Site of conduction delay. This pattern has been attributed to causes other than conduction delay in the right bundle branch (19,31–33). The suggested sites of delay include the right ventricular free wall, in particular in the presence of hypertrophy of the crista supraventricularis, conduction delay in the terminal right ventricular Purkinje network or physiologic variability of the thickness and distribution of right ventricular mass (33). The pattern of incomplete right bundle branch block may occur in right ventricular hypertrophy due to congenital or acquired heart disease and chronic lung disease. It may also be observed as a normal variant in patients with left anterior fascicular block, posterobasal myocardial infarction and some skeletal deformities, such as pectus excavatum or straight back syndrome.

Prevalence in normal subjects. Hiss and Lamb (35) reported a prevalence of 2.4% in a large series of normal young subjects. Raunio et al. (36) observed an rsr′ in lead V_1 in 2.9% of children, 1.4% of young adults and 0.6% of middle-aged and elderly subjects in the absence of cardiopulmonary disease. When the right precordial leads are recorded one intercostal space lower, the R′ wave may diminish or disappear (37).

Criteria for Complete Left Bundle Branch Block

Sequence of ventricular activation. In complete left bundle branch block, the sequence of ventricular activation is changed from its onset (24,25,38–40). Due to block in the left bundle, excitation first appears low on the right septal surface near the base of the anterior papillary muscle. The initial activation is septal from right to left, making the left ventricular cavity potential initially positive. From this point, early activation spreads over the endocardial surface of the right ventricle and upward over the right septal surface. The resulting initial forces are directed leftward and most often anteriorly and inferiorly. As the right to left septal activation continues and proceeds slowly from muscle fiber to muscle fiber rather than through the specific Purkinje system and over to the left septum, the forces generated will be oriented leftward, posteriorly and inferiorly. The leftward direction of the ventricular forces remains as the activation process proceeds in the free wall of the left ventricle. There are different opinions (24–26,38–42) on the exact pattern of activation and the site of the conduction delay in the left ventricle.

The leftward orientation of the initial forces explains the absence of the normal ''septal'' Q wave in lead I and the left precordial leads. Slurring and notching of the R wave in these leads is due to the abnormal slow conduction in the left ventricle. The increase in time required for the completion of the activation process causes prolongation of the QRS duration to 0.12 second and more.

Diagnostic criteria. The electrocardiographic criteria for the diagnosis of uncomplicated complete left bundle branch block can be summarized as follows (39,43):

1) The QRS duration is 0.12 second or more.

2) Left-sided precordial leads, V_5 and V_6, as well as lead I and aVL show broad and notched or slurred R waves. Occasionally, an RS pattern may occur in leads V_5 and V_6 in uncomplicated left bundle branch block associated with posterior displacement of the left ventricle. An R pattern

JACC Vol. 5, No. 6
June 1985:1261–75

may then be seen if leads V_7 and V_8 are recorded in these patients (19).

3) With possible exception of lead aVL, Q waves are absent in the left-sided leads, specifically in leads V_5, V_6 and I.

4) The R peak time is prolonged to more than 0.06 second in lead V_5 or V_6, but is normal in leads V_1 and V_2 when it can be determined.

5) In the right precordial leads V_1 and V_3, there are small initial r waves in the majority of cases, followed by wide and deep S waves. The transition zone in the precordial leads is displaced to the left. Wide QS complexes may be present in leads V_1 and V_2 and rarely in lead V_3.

Although these criteria are considered to be typical of left bundle branch block (as in complete right bundle branch block), one cannot be sure about the actual site of block. Thus, the pattern of left bundle branch block may be closely mimicked by conduction delay in the peripheral left ventricular Purkinje system or myocardium, or both.

ST-T changes. In uncomplicated left bundle branch block, the ST segments are usually depressed and the T waves inverted in left precordial leads V_5 and V_6 as well as in leads I and aVL. Conversely, ST segment elevations and positive T waves are recorded in leads V_1 and V_2. Only rarely is the T wave upright in the left precordial leads in an uncomplicated case of left bundle branch block.

Criteria for Incomplete Left Bundle Branch Block

The existence of incomplete left bundle branch block as an electrocardiographic entity has long been debated (44–46).

Diagnostic criteria. The generally accepted criteria are:

1) A QRS duration of more than 0.10 second, but less than 0.12 second.

2) Prolongation of the R peak time to 0.06 second or more in the left precordial leads.

3) Absence of a Q wave in the left precordial leads (V_5 and V_6) and lead I.

4) The presence of notching or slurring, or both, of the ascending limb of the R wave in the left precordial leads increases the likelihood of the diagnosis of incomplete left bundle branch block.

It is fairly common to see these abnormalities in patients with left ventricular hypertrophy. As a result, the question may be raised whether this conduction defect coexists with or whether the changes are secondary to left ventricular hypertrophy (19). However, increased QRS voltages have been observed with the onset of incomplete left bundle branch block (45,47). Further lines of clinical investigation designed to resolve this problem have been proposed by Schamroth and Bradlow (44) and Barold et al. (45).

Progression of conduction delay. According to Schamroth and Bradlow (44), to establish convincing clinical evidence of incomplete left bundle branch block in a patient, it is necessary to demonstrate: 1) tracings with normal ventricular conduction, 2) subsequent tracings that show various degrees of incomplete left bundle branch block, 3) tracings that eventually show complete left bundle branch block, and 4) transitions occurring during a short-term interval. These criteria may, indeed, establish a firm diagnosis of incomplete left bundle branch block, but they occur so rarely in a single patient as to be of little clinical use.

Barold et al. (45) stated that any recognizable degree of incomplete left bundle branch block is characterized by the disappearance of the small initial Q wave and a small increase in the voltage of the R wave in the left precordial leads. When the degree of incomplete left bundle branch block increases, slurring of the initial portion of the R wave appears and the intrinsicoid deflection becomes prolonged. In advanced incomplete left bundle branch block, the duration and amplitude of the initial slurring increases. When complete left bundle branch block supervenes, QRS prolongation over 0.12 second ensues and a notched plateau after the upstroke of the R wave becomes manifest in the left precordial leads.

According to Sodi-Pallares et al. (24), the duration and amplitude of the initial slurring of the ascending limb of the R wave in the left precordial leads are the most useful diagnostic features of incomplete left bundle branch block. These investigators experimentally demonstrated that when the degree of block increases, reversal of the direction of septal activation occurs. When incomplete left bundle branch block is generated, the initial forces of the vectorcardiogram change direction and are written anteriorly and slightly to the left, while the major part of the QRS loop is inscribed posteriorly. Further degrees of incomplete left bundle branch block cause clockwise rotation of the loop in the transverse plane as is seen in complete left bundle branch block.

Criteria for Left Anterior Fascicular Block

With Rosenbaum's introduction of the "hemiblocks" (17,48), a new concept was introduced, now commonly referred to as fascicular block. According to Rosenbaum and his coworkers (17,48), a delay or interruption of impulse conduction in one of the divisions of the left bundle branch will result in asynchronous activation of the left ventricle.

Ventricular activation. In left anterior fascicular block, the impulse first spreads inferiorly through the posterior division. The excitation of the anterior and lateral wall is delayed and depends mainly on the impulse arriving from the posterior division. Therefore, the QRS vectors are displaced leftward and in a superior direction. The late QRS

forces become prominent because they are mostly unopposed. The initial 0.02 second forces from the septum and inferior wall are directed in most cases rightward and inferiorly (17,48).

Diagnostic criteria. The criteria for uncomplicated left anterior fascicular block are still not firmly established. The original criteria for left anterior hemiblock as proposed by Rosenbaum et al. (17) were: 1) frontal plane QRS axis −45 to −80°; 2) QRS duration of 0.11 second or less; and 3) small Q wave of 0.02 second or less in leads I and aVL.

Diagnostic role of degree of left axis deviation. Milliken (49) recently reviewed these criteria and the amendments added by several investigators since 1970 (18,50–57). The use of left-axis deviation alone is inadequate to recognize left anterior fascicular block, and most authors agree that left-axis deviation and left anterior fascicular block are not synonymous (49,55,56). There are many causes other than an interruption or delay in the left anterior fascicle that may produce a shift of electrical forces to the left and superiorly in the frontal plane (8,9,11,13). A discrete cut-off point of −45°, as proposed by Rosenbaum et al. (17,48), eliminates many of these causes, but may result in the missing of left anterior fascicular block of a lesser degree. This has led some investigators to categorize all left-axis deviation beyond −30° as left anterior fascicular block (13,54,57) or intraventricular conduction delay (53,55). This limit has since then been used in the majority of clinical and pathologic studies related to left anterior fascicular block (19). According to Milliken (49), it might be possible to accept a more liberal definition of the degree of left-axis deviation (that is, < −30°), but this can only be done if there is some way of measuring the regional conduction delay in the left anterosuperior and basal portion of the left ventricle. Because left ventricular conduction in left anterior fascicular block initially spreads from the left posteroinferior fascicle's termination toward the delayed anterosuperior area, it should be stressed that the axis shift should be evident within the first 60 ms of the QRS complex. This criterion should especially be followed when the QRS duration is prolonged to over 0.11 second, for example due to associated complete right bundle branch block, which might cause a change in QRS axis of the terminal QRS vectors. The depolarization delay due to uncomplicated left anterior fascicular block may cause the QRS duration to increase by a maximum of 20 ms (48,53).

Small Q wave in leads I and aVL. The last criterion originally proposed by Rosenbaum, specifying that a small Q wave should be evident in leads I and aVL, has caused much controversy (49,50). Kulbertus et al. (50) found that the initial 10 ms QRS vectors are nearly always directed inferiorly, but in 55% they were directed to the right and in 45% to the left. In 3 of 40 cases, the 10 ms vector was less than +20° in the XY plane (in two it was located close to zero and in one at −60°). Jacobsen et al. (58) and Burchell and Tuna (55) concluded that a Q wave in leads I and aVL is not a requirement for left anterior fascicular block.

Additional criteria. Since one cannot rely on left-axis deviation alone to diagnose left anterior fascicular block, some investigators (49) have searched for additional criteria to provide a measure of delayed conduction in the region of the affected fascicle. According to Medrano et al. (59), there should be slurring of the downstroke of the R wave and a delayed R peak time of 45 ms or more in lead aVL. If these signs are absent in this lead, a late slurred terminal R wave should be present in lead aVR or slurred S waves in leads V_5 and V_6. Horwitz et al. (52) found these additional criteria in lead aVL in 62% of 400 records with left-axis deviation beyond −30°, whereas Fisher et al. (60) reported a late slurred terminal R wave in lead aVR in 44 of their 47 patients.

Recommended criteria. According to the present Task Force, the generally accepted criteria for the diagnosis of uncomplicated left anterior fascicular block are: 1) left-axis deviation of −45 to −90°; 2) a qR pattern in lead aVL; 3) an R peak time in lead aVL of 45 ms or more; and 4) QRS duration less than 0.12 second. The diagnosis of possible left anterior fascicular block can be made when there is left-axis deviation of −30° or more and the preceding listed criteria are present.

It should be emphasized that in an uncomplicated case of left anterior fascicular block, an rS complex is usually recorded in the inferior leads. It may further be noted that the transitional zone in the precordial leads is often displaced to the left, with a decrease in the amplitude of the R wave and an increase in the amplitude and width of the S wave in the left precordial leads. These changes are related to the superior displacement of the QRS forces. The lead axes of V_5 and V_6 are directed not only leftward, but also slightly downward. The late QRS forces in left anterior fascicular block often project on the negative side of these lead axes.

A less common but clinically important finding in left anterior fascicular block is the appearance in some cases of a small q wave in the right precordial leads. This is due to a change in orientation of the initial QRS forces (19). When precordial leads are recorded one intercostal space below their routine locations, the small q waves often disappear. As just described, an r′ wave may also be recorded in the right precordial leads in some patients.

Rotation of the QRS loops in the frontal plane is counterclockwise. This counterclockwise rotation is helpful in diagnosing the condition in the presence of inferior wall infarction. The initial 10 to 20 ms QRS vectors are directed inferiorly in uncomplicated left anterior fascicular block, and the maximal QRS deflection vectors are displaced superiorly. Most of the QRS loop area in the frontal plane in

JACC Vol. 5, No. 6
June 1985:1261–75

left anterior fascicular block is located in the left superior quadrant (50).

Criteria for Left Posterior Fascicular Block

Anatomic factors. Left posterior fascicular block occurs much less frequently than left anterior fascicular block. Rosenbaum et al. (17,48) attributed this to the following factors: 1) the anterior fascicle has a single blood supply, derived from the perforating septal branches of the left anterior descending artery, whereas the posterior fascicle has a dual blood supply from septal branches of both the anterior and posterior descending coronary arteries; 2) the anterior fascicle is long and much thinner as compared with the posterior fascicle; 3) the anterior fascicle is located in the hemodynamically turbulent outflow tract, whereas the posterior fascicle lies in the more quiet inflow tract of the left ventricle; and 4) the posterior fascicle is the first to leave the main left bundle and spread out in wide ramifications.

Ventricular activation. According to Rosenbaum et al. (17,48), block in the posterior fascicle causes the excitation wave to travel first through the area of the anterior fascicle and then to spread out inferiorly. As a result, the initial 10 to 20 ms QRS vector will be directed leftward to about $-45°$ and slightly superiorly as well as posteriorly. In the 12 lead electrocardiogram, this will be reflected by Q waves in the inferior leads. A Q wave should always be present in lead III, but may occasionally be absent or very small in leads II and aVF according to some investigators (61–64). The middle and terminal QRS vectors will be directed inferiorly and rightward. The QRS loop in the frontal plane will rotate clockwise (65). Similarly, as in left anterior fascicular block, block in the posterior fascicle will cause slight (usually not more than 0.02 second) prolongation of the QRS complex.

Diagnostic criteria. The following criteria have been proposed to diagnose uncomplicated left posterior fascicular block: 1) a frontal plane QRS axis of $+90$ to $+180°$; 2) rS configuration in leads I and aVL, associated with a qR pattern in the inferior leads and obligatory Q waves in leads III and aVF. The Q waves in the inferior leads should be 0.04 second or less. Given criterion 1, it follows that the voltage of the R wave in lead III should equal or exceed that in lead II (all these findings result in a so-called S1-Q3 pattern); and 3) the QRS duration is less than 0.12 second.

Left posterior fascicular block is an electrocardiographic diagnosis that needs to be supported by clinical findings. First, it is necessary to exclude other causes of abnormal right-axis deviation such as right ventricular hypertrophy, chronic obstructive pulmonary disease, emphysema, vertical heart and extensive lateral wall myocardial infarction. The diagnosis should be made with caution in those younger than 30 years of age. When an abnormal right-axis deviation is observed in the appropriate clinical setting (that is, in adults with medium or heavy body build with left ventricular disease and no evidence of right heart involvement), the diagnosis of left posterior fascicular block can strongly be suggested in the opinion of most authorities (13,17–19, 48,61–66).

Right-axis deviation as a criterion. Rosenbaum et al. (17,48) first suggested a QRS axis of $+120°$ as the criterion for diagnosis of left posterior fascicular block, but they later accepted $+90°$ or even less. These workers suggested that there might be incomplete forms of left posterior fascicular block. Experimental work by Watt and Pruitt (67) in primate hearts also suggested that left posterior fascicular block need not be accompanied by an extreme right-axis deviation. Their studies as well as those by Pryor (62) emphasized the importance of a shift in the clockwise direction of the late (major) QRS vectors in the frontal plane. Also Chou (19) described a patient with intermittent left posterior fascicular block with a QRS axis in the frontal plane of 80° during the block. According to Watt and Pruitt (67), in the proper clinical setting, a reliable diagnosis of left posterior fascicular block requires not only the presence of the criteria just listed, but also serial comparison with electrocardiograms made before development of the conduction defect.

Precordial lead changes. Although the cardinal findings of left posterior fascicular block are observed in the limb leads, significant changes may also occur in the precordial leads. The transitional zone is often displaced leftward (66). This leads to an RS complex in the left precordial leads, which might cause further difficulties in the differentiation with right ventricular hypertrophy (19). If a Q wave is present in the left precordial leads before the block develops, the leftward shift of the initial QRS forces may cause it to disappear (68).

Bilateral, Bifascicular, Trifascicular and Nonspecific (unspecified) Intraventricular Block

Bilateral bundle branch block. The term bilateral bundle branch block implies a conduction disturbance in the right and left bundle branches. This may occur alternately or intermittently or it may be permanent (1,2). If block is complete in both bundle branches, it results in third degree AV block. The term bilateral bundle branch block has also been used to indicate the combination of first and second degree AV block with complete block in either the right or left bundle branch. However, since incomplete AV block may be located in either the AV node, the bundle of His or the contralateral bundle, the use of the term bilateral bundle branch block in this setting is discouraged.

Bifascicular and trifascicular block. Confusion has also occurred with use of the terms bifascicular and trifascicular block. In its common usage, the term bifascicular block is

applied to the combination of complete right bundle branch block with either left anterior fascicular block or left posterior fascicular block. The former combination is fairly common. The term trifascicular block has been proposed to represent eight possible combinations of complete or incomplete block in the right bundle branch and the two main divisions of the left bundle branch (17,48,69). This frequently results in first or second degree AV block. To avoid semantic errors, it has been recommended that each conduction defect be described specifically in terms of the structure or structures involved instead of by the terms bifascicular and trifascicular block (1,2,13) (see Appendix).

Intraventricular block. When a prolonged QRS duration beyond 0.11 second exists, but does not satisfy the criteria for either left or right bundle branch block pattern, the diagnosis of nonspecific (unspecified) intraventricular block or conduction delay is preferred (1,2).

Pre-excitation: Wolff-Parkinson-White and Related Patterns

Anatomic basis. According to Durrer et al. (70), pre-excitation exists if in relation to atrial events, the whole or some part of the ventricular muscle is activated earlier by the impulse originating from the atrium than would be expected if the impulse reached the ventricles by way of the normal conduction system only. The possible anatomic pathways between atria and ventricles in the human heart that may play a role in the pre-excitation syndromes include the classical Kent bundles, the atrionodal bypass tract of James and the septal fibers of Mahaim (70–76). To avoid controversy about the eponyms, it has recently been proposed (77,78) to use the terms accessory AV connections (instead of bundle of Kent), nodoventricular and fasciculoventricular connections (instead of Mahaim fibers) and atriofascicular (atrio-His) connections as well as intranodal bypass (short circuit) tracts (instead of James fibers). Atrial tracts that skirt the AV node and approach the tricuspid valve are present in most normal hearts. These generally penetrate the lowest part of the AV node and, as such, constitute a partial bypass of the AV node. In contrast, fibers running from the atrium directly to the bundle of His and completely bypassing the AV node are rare (77,78).

The term Wolff-Parkinson-White pattern has been recommended to describe the salient electrocardiographic findings of shortening of the PR interval, a delta wave and widening of the QRS complex (2). The term Wolff-Parkinson-White syndrome implies the occurrence of tachycardias in association with the Wolff-Parkinson-White pattern (2).

Wolff-Parkinson-White Electrocardiogram

In its classic form, pre-excitation occurs through an accessory AV bundle, which allows the atrial impulse to partially or completely bypass the AV node to activate prematurely the ipsilateral ventricle (70,71). The classic electrocardiographic findings described by Wolff, Parkinson and White (72) consist of: 1) A short PR interval of less than 0.12 second during sinus rhythm; 2) the presence of an initial slurring in the QRS complex (the delta wave); 3) an abnormally wide QRS complex equal to 0.12 second or more; 4) secondary ST and T wave changes; and 5) the frequent association with paroxysmal tachycardia.

Factors determining the degree of pre-excitation. Programmed electrical stimulation of the heart has shown, however, that the electrocardiographic pattern of ventricular activation depends on the contribution to ventricular activation over each AV pathway (73). The Wolff-Parkinson-White electrocardiogram may range from the classic electrocardiogram (short PR, delta wave, QRS width $\geq$0.12 second) to one showing little contribution to ventricular activation by way of the accessory AV pathway. Figure 2 illustrates that one should not rigidly adhere to the rule that a P-delta interval of 0.12 second or less, a clear delta wave and a QRS width of 0.12 second or more are required to make the diagnosis of a Wolff-Parkinson-White pattern. The location of the accessory pathway, the intraatrial conduction time and the times required to traverse the AV node-His bundle branch pathway and the accessory pathway determine the configuration of the electrocardiogram. Conduction over the AV node is especially affected by changes in autonomic tone and a number of drugs. Pre-excitation, therefore, becomes more clear when AV nodal conduction is slowed by carotid sinus massage and may diminish when AV nodal conduction is accelerated by exercise. In many patients the Wolff-Parkinson-White pattern may appear only intermittently. Thus no rigid criteria can be formulated for the diagnosis of the Wolff-Parkinson-White pattern.

Embryology. It has been postulated that the pre-excitation pattern is the result of embryologic faulty development of the AV ring (71). Normally, the AV ring (anulus fibrosus) is a continuous sheet of fibrous tissue separating the atria from the ventricles. In patients with pre-excitation, congenital clefts have been found in the fibrous ring that are occupied by muscular bridges serving as accessory pathways. These bridges may be situated anywhere in the right (tricuspid) or left (mitral) side of the AV ring as well as in the interventricular septal area (71,74,75). Also, multiple accessory pathways may be present (76), the functional characteristics of which may be different. Becker et al. (77) found a well-formed fibrous anulus in four patients with a left-sided accessory bundle and, therefore, they doubt the embryologic fault theory.

Localization of the accessory pathway. With recent advances in surgical treatment of refractory tachycardias in the Wolff-Parkinson-White syndrome, precise localization of the site of the accessory pathway becomes very important. To this end, special electrophysiologic studies, such as pac-

JACC Vol. 5, No. 6
June 1985:1261–75

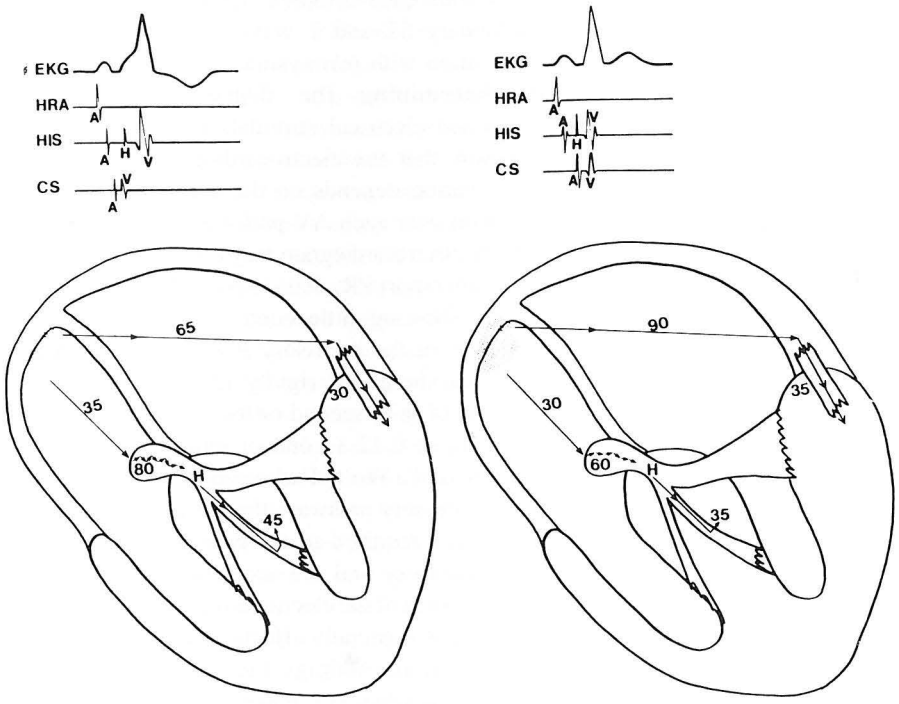

Figure 2. Illustration of the factors determining the degree of ventricular pre-excitation in the Wolff-Parkinson-White syndrome during sinus rhythm. The corresponding electrocardiogram (EKG) and the intracavitary recordings from the right atrium (HRA), His bundle region (His) and coronary sinus (CS) are shown in the **upper panels**. **Left**, The atrioventricular (AV) conduction time from the sinus node over the normal AV pathway measures 160 ms (the sum of the time required to travel from the sinus node to the AV node [35 ms], the trans AV nodal conduction time [AH interval of 80 ms] and the time needed to travel through the bundle of His and the bundle branches to the ventricular myocardium [HV interval of 45 ms]). The time required to travel from the sinus node to the atrial insertion of the accessory pathway measures 65 ms, and the conduction time over the accessory pathway is 30 ms. The total time of AV conduction from the sinus node to the ventricle using the accessory pathway is 95 ms. The corresponding electrocardiogram shows a P-delta wave interval of 95 ms and a wide QRS complex with ventricular activation starting 65 ms earlier than expected (160 minus 95 ms). **Right**, Compared with the **left panel**, there is: 1) a longer conduction time from the sinus node to atrial insertion of the accessory pathway (90 ms), 2) a longer conduction time over the accessory pathway (35 ms), and 3) a shorter conduction time over the AV node (60 ms). As a result of these differences, the AV conduction times over the normal and the accessory pathway are identical (both 125 ms). Now the electrocardiogram shows a PR interval of 125 ms and a QRS complex which is not widened.

ing of the atria and coronary sinus as well as endocardial and epicardial mapping are currently performed (74,79). However, study of the QRS configuration from the conventional electrocardiogram and vectorcardiogram, more specifically the direction of the initial QRS forces (the delta wave), can also be very helpful (74,75,80–82). Indeed, since the description of the Wolff-Parkinson-White syndrome in 1930, many attempts have been made to localize the accessory pathway according to the electrocardiogram. For a long time, a classification into type A and B, as proposed by Rosenbaum et al. (81), has been used. However, programmed electrical stimulation and epicardial excitation mapping have resulted in a more refined classification, as described by Tonkin et al. (80) and Gallagher et al. (75). A similar attempt at classification was proposed by Boineau et al. (74) and Frank et al. (82). Becker et al. (77) used these classification criteria and were able to predict the location of the accessory atrioventricular connections in five of seven patients. In the two remaining patients, no connections could be found histologically.

From these studies, it is evident that the variation in electrocardiographic type, including the direction of initial or delta forces, is related to the site of pre-excitation. The direction of the peak QRS vector is determined by the interaction between the site of entry of the anomalous wave front and the relative time of arrival of the anomalous and normal impulses in the ventricles (74).

Other Forms of Pre-excitation

Bundle of James. The bundle of James is a paranodal pathway that bypasses the upper and central AV node, where normal AV conduction delay occurs, to connect with the lower third of the node or directly with the bundle of His (83,84). As a result of conduction over this AV nodal bypass

tract, the PR interval becomes shortened, but the QRS complex remains normal because the ventricular activation pattern is unaffected (70,71).

Mahaim fibers. The bypass fibers of Mahaim are short direct connections between the lower AV node or His bundle and the ventricular septum. Excitation over these fibers will result in pre-excitation of the ventricular septum and, thus, in a delta wave. So far, only connections to the right side of the ventricular septum have been described.

Short PR syndrome. The term Lown-Ganong-Levine syndrome has been used since 1952 to describe the electrocardiographic findings of a short PR interval, a normal QRS complex and the presence of paroxysmal tachycardia (85). These electrocardiographic findings had already been described in 1938 by Clerc et al. (86). The term "short PR syndrome" has therefore been recommended (2). However, it is generally agreed that this term should be restricted to those patients who have a definite history of paroxysmal tachycardia and should not be used for patients who only present a short PR interval and normal QRS complex (1,2). Atrioventricular junctional abnormalities of unknown origin resulting in enhanced AV conduction and abbreviated AV node refractory periods (87,88) may explain this syndrome, but the presence of a paranodal AV bypass tract of James may according to some authors also be the explanation (70,78,89). As in the classic Wolff-Parkinson-White syndrome, the presence of a bypass tract facilitates reciprocal return of an impulse to the atria, which may lead to development of reciprocal tachycardia (79).

Intraatrial Conduction Disturbances

Atrial activation. In dog experiments (91,92), atrial excitation during sinus rhythm has been shown to have a radial spread. Although detailed data on the sequence of atrial activation in human beings are scarce, they are rather consistent. Although different experimental set-ups and techniques of measurements have been used, the results confirm a radial sequence of activation (23,93,94). More or less concentric isochrones may show slight deviations due to faster conduction over areas where the muscle is thicker, and do not necessarily imply conduction over specialized pathways (23,92,94,95). During sinus rhythm, excitation proceeds from the anterior surface of the high right atrium to the low right atrium and over the interatrial band to the high left atrium, the low left atrium being excited latest (96).

Intraatrial conduction delay. The normal P wave is a wave whose direction and configuration are determined among other factors by the timecourse and order of the atrial depolarization process. Its duration increases with age, but should not exceed 0.11 second in the adult (8). A longer P wave is considered a sign of delayed intraatrial conduction, such as may be seen with atrial disease and left or right atrial enlargement. In addition, a P wave configuration similar to that of left atrial enlargement (a wide P wave [>0.11 second] with a terminal negative force in lead V_1 greater than 0.04 second and 0.1 mV or greater [43]) consistently showed delayed activation of the lower left atrium, with a poor correlation with actual left atrial enlargement (93).

Furthermore, it has been demonstrated that surgical damage to the atrial wall and interatrial septum may produce local block and a change of epicardial and endocardial activation patterns without significantly lengthening atrial activation time (94). It is uncertain whether such local delay or block may result in abnormal notching of the P wave without an increase in duration as a sign of local intraatrial conduction disturbance.

The following criteria indicate possible intraatrial conduction defects: 1) P wave duration greater than 0.12 second, and 2) notching of the P wave.

Comparison of Visual and Computer-Derived Interval Measurements

Factors determining discrepancy in measurements. It is generally known that computer-derived intervals are longer than visually determined time measurements from conventional electrocardiographic recordings obtained at a paper speed of 25 or 50 mm/s. There are three main reasons for this apparent discrepancy.

Single versus three simultaneous leads. First, manual results have mostly been derived from single leads, whereas computer measurements are widely based on at least three simultaneously recorded leads. The shortcomings of electrocardiographic measurements (P and QRS duration, as well as PR and QT interval) from single leads and the need for multichannel data for greater accuracy of electrocardiographic time-phase analysis have been stressed by several investigators (97–99).

Baseline and writing characteristics of the recorder. Second, it has been demonstrated that visual interval measurements are highly dependent not only on the quality of the electrocardiographic recording, but also on the baseline and writing characteristics of the recorder. Rautaharju et al. (3) noted that a round stylus with a uniform width of 0.25 mm may produce a bias of up to 8 ms in Q wave duration measurements (100). Through computer processing, these errors can be avoided by means of signal conditioning and improved measurement techniques.

Paper speed and amplification. Third, paper speed and amplification factor play an important role for accurate interval measurements. When visual measurements are performed on high gain recordings with an amplification factor of 10 or 20, significantly wider intervals, similar to computer-derived results, have been obtained as demonstrated

in the Common Standards for Quantitative Electrocardiography (CSE) Project (5,6) and other studies.

Comparison of electrocardiographic measurements. In general, computer-derived P and QRS duration measurements as well as PR intervals are on the average 8 to 12 ms longer than results derived by visual interpretation of normal speed and gain recordings. Hence past criteria using previously published measurements cannot be used in computer programs unless adjustments are made. For example, instead of using a PR interval of 120 ms as a criterion for pre-excitation, various computer programs apply 128 or 130 ms, and for first degree AV block the limit has similarly been moved up.

Based on simultaneously recorded Frank XYZ leads, Draper et al. (101) found that the QRS duration extended up to 0.112 second (96% range) in 510 normal subjects, which is significantly longer than the normal upper limit of 0.10 second published in many textbooks. Accordingly, the dividing limit for computer diagnosis of ventricular conduction defects has been increased to 0.123 (102) and 0.126 ms (103) in the Pipberger program.

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APPENDIX

Definition of Ventricular Conduction Delays

A. Complete Bundle Branch Blocks

Qualifying statements:
- S1) QRS duration ≥0.120 second (adults)
- S2) Supraventricular rhythm
- S3) Absence of WPW pattern

Criteria for a complete bundle branch block:
- S1 and S2 and S3

1. Complete right bundle branch block (RBBB).

Qualifying statements:
- S1) R′ or r′ in V_1 or V_2
- S2) S duration > R duration in I and V_6
- S3) S duration > 0.040 second in I and V_6
- S4) R peak time >0.050 second in V_1 or V_2

Criteria for RBBB:
- a) S1 and S2 or
- b) S1 and S3 or
- c) S4 and (S2 or S3)

2. Complete left bundle branch block (LBBB).

Qualifying statements:
- S1) Broad and notched or slurred R in I and V_5 or V_6
- S2) Absence of Q wave in I and V_5 and V_6
- S3) R peak times ≥0.060 second in V_5 or V_6

Criteria for LBBB:
- a) S1 and S2 and S3

3. Nonspecific (unspecified) intraventricular block. All cases with QRS duration greater than 0.12 second which do not meet the criteria for LBBB or RBBB

B. Incomplete Bundle Branch Blocks

1. Incomplete LBBB.

Qualifying statements:
- S1) QRS duration ≥0.100 second and QRS duration <0.120 second
- S2) Absence of Q waves in I and V_5 and V_6
- S3) R peak time >0.060 second in V_5 or V_6

Criteria for incomplete LBBB:
- a) S1 and S2 and S3

2. Incomplete RBBB.

Qualifying statements:
- S1) QRS duration <0.120 second
- S2) r′ or R′ in V_1 or V_2
- S3) R′ >R in V_1 or V_2
- S4) R peak time >0.050 second in V_1 or V_2

Criteria for incomplete RBBB:
- a) S1 and S2 and S3 or
- b) S1 and S4

C. Fascicular Blocks

1. Left anterior fascicular block (LAFB).

Qualifying statements:
- S1) QRS duration <0.120 second
- S2) QRS axis ≤ −45°
- S3) QRS axis ≤ −30° and QRS axis > −45°
- S4) rS pattern in II and III and aVF
- S5) qR pattern in aVL
- S6) R peak time ≥0.045 second in aVL
- S7) Slurred R downstroke in aVL
- S8) Slurred S in V_5 or V_6

Criteria for uncomplicated LAFB:
- a) S1 and S2 and S4 and S5 and S6 or
- b) S1 and S2 and S4 and S5 and S7 or
- c) S1 and S2 and S4 and S5 and S8

Qualifying statement S4 is usually present with criteria a, b and c above. If there is a QS in lead II, LAFB cannot be differentiated from inferior myocardial infarction.

Criteria for possible uncomplicated LAFB:
- a) S1 and S3 and S4 and S5 and S6 or
- b) S1 and S3 and S4 and S5 and S7 or
- c) S1 and S3 and S4 and S5 and S8

2. Left posterior fascicular block (LPFB).

Qualifying statements:
- S1) QRS duration <0.120 second

S2) QRS axis >90° and
QRS axis <180°
S3) R in III > R in II (note that S3 is a consequence of S2)
S4) qR pattern in III and aVF with Q duration ≤0.040 second
S5) absence of other causes of right-axis deviation

Criteria for LPFB:

a) S1 and S2 and S3 and S4 and S5

Definition of Pre-excitation Patterns

1. Classic Wolff-Parkinson-White (WPW) pattern.*

Condition statements:

S1) PR <0.120* ms
S2) P axis >0° and
P axis ≤90° (in frontal plane)
S3) QRS duration ≥0.120 second*
S4) Presence of delta wave

Criteria for WPW pattern:

a) S1 and S2 and S3 and S4

*Note that these criteria are not absolute but depend on the location and the contribution to ventricular excitation of the accessory AV pathway (see text).

2. Subclassification of pre-excitation patterns (according to primary patterns described in Ref. 74, 75, 79 and 81).

Condition statements:

S1) Delta wave (initial QRS) positive in I
S2) Delta wave positive in V_1
S3) Delta wave negative in V_1
S4) Delta wave isoelectric in V_1
S5) Q or QS waves in I and V_6
S6) Q or QS waves in III and aVF
S7) Main QRS positive in III
S8) Main QRS negative in III
S9) Main QRS positive in V_1
S10) Main QRS negative in V_1
S11) Delta wave positive in II and aVF and positive or isoelectric in III
S12) Delta wave negative in II, III, aVF
S13) Delta wave positive from V_2 until V_6
S14) Delta wave negative or isoelectric in V_6
S15) QRS configuration rS in V_1 and Rs in V_2

Criterion for anterior right ventricular pre-excitation:

a) S1 and S4 and S8 and S10

Criterion for posterior right ventricular pre-excitation:

a) S3 and S6 and S8 and S10

Criterion for posterior left ventricular pre-excitation:

a) S1 and S2 and S6 and S8 and S9 and S14

Criterion for lateral left ventricular pre-excitation:

a) S2 and S5 and S7 and S14

Criterion for anterior paraseptal pre-excitation:

a) S1 and S4 and S11

Criterion for posterior paraseptal pre-excitation:

a) S1 and S12 and S13 and S15

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