The Early Signs of Right Bundle Branch Block


Complete right bundle branch block manifests with a rSR' configuration in right orientated leads and a duration of 0.12 sec or longer. Incomplete right bundle branch block is perceived as this classic rSR' configuration and a duration of less than 0.12 sec. This presentation reflects the early development of right bundle branch block which first manifests with a hitherto undescribed sign, namely: a diminution of the S wave amplitude in lead V1. Further progression of the right bundle branch block leads to slurring or notching of the upstroke of the S wave in lead V1, followed by the development of a r' deflection. With further progression, the r' deflection becomes increasingly taller until the advent of complete right bundle branch block which is characterized by a widening of a very tall R' deflection with an apical notch or plateau.

Right bundle branch block results in a large and abnormal terminal QRS vector which is directed anteriorly, to the right, and either up or down (Fig 1). This causes a terminal R' deflection in right-orientated leads, leads V1 and V3, and a terminal wide and deep S wave in left-orientated leads, leads V5, V6, standard lead I and lead aVL. The resulting triphasic configuration of rSR' in the right-orientated leads, and QRS configuration in left-orientated leads constitutes the hallmark of right bundle branch block, the terminal deflection being its characteristic feature. Complete right bundle branch block is perceived as this classic configuration, with a duration of 0.12 sec or longer. Incomplete right bundle branch block is perceived as the classic rSR' and QRS configurations, with a duration of less than 0.12 sec. As will be shown, however, the early development of incomplete right bundle branch block is not necessarily manifest with the typical triphasic QRS configuration. The changes can be subtle. In particular, attention is drawn to a hitherto undescribed early, and indeed the first, sign of right bundle branch block: diminution of the S wave amplitude in lead V2.

Case Reports

Case 1

The ECG (standard lead 2 and a continuous strip of lead V1, Fig 2) shows atrial flutter with variable second-degree AV block. This includes regular 4:1 ratios (as seen in standard lead 2) and alternating 2:1 and 4:1 AV conduction ratios (sections of lead V1). The latter results in bigeminal rhythm, which may be associated with: (a) normal intraventricular conduction, as exemplified by the second half of the bottom strip, or (b) aberrant ventricular conducting, reflecting right bundle branch block, as shown in the second half of the middle strip of lead V1. This results in alternate QRS complexes with the classic triphasic rSR' configuration of right bundle branch block. Alternating 2:1 and 4:1 AV conduction ratios during atrial flutter form the ideal substrate for aberration. Thus, the conducted impulse terminating the 2:1 AV conduction is relatively premature (when compared with the impulse terminating the 4:1 AV conduction ratio) and is therefore more likely to encounter refractory conducting tissue (Fig 3). Furthermore, the impulse terminating the 2:1 AV conduction ratio is preceded by a long cycle, occasioned by the 4:1 AV conduction ratios (as illustrated in Fig 3). Such a long cycle, by virtue of the Ashman phenomenon, increases the duration of the following refractory period, so that ensuing aberration is more likely. The fourth and sixth complexes of the middle strip of lead V1 are of particular significance in this context. They show the development or beginning of the right bundle branch block aberration, which merely is manifest as a diminution in the size of the S wave.

Case 2

The ECG (Fig 4, a continuous strip of lead V1) was recorded from a 46-year-old man with hypertension. It shows sinus rhythm with minimal sinus arrhythmia: the P-P intervals range from 0.80 to 0.90 sec. The P waves reflect left atrial enlargement as evidenced by the predominantly negative P waves. The first nine beats reflect left ventricular hypertrophy due to the left ventricular systolic overload as evidenced by the deep S waves, the elevated ST segment, and the upright T wave. The last beat of the top strip shows a marked

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Manuscript received and accepted October 4.
FIGURE 2. Electrocardiogram (standard lead 2 and continuous strip of lead V₂) of case 1.

FIGURE 3. Diagram illustrating atrial flutter with alternating 2:1 and 4:1 AV conduction ratios.

FIGURE 4. Electrocardiogram (a continuous strip of lead V₁) of case 2.

Shortening of the S wave with inversion of the T wave. This is also evident in the first two beats of the second strip. The remaining beats of the second strip, as well as the first four beats of the third strip, show incomplete right bundle branch block, as evidenced by a small terminal r' deflection. The r' deflection varies in amplitude depending on the degree of incomplete right bundle branch block, and is more marked when associated with shorter cycles, e.g. 0.80 sec. The long pause following the ventricular extrasystole in the third strip is
followed by three beats which, as before, have a deep S wave. The last beat of the third strip, as well as the first three beats of the bottom strip, are once again associated with short S waves. In addition, there is some slurring of the ascending limb of the S wave as it approaches the baseline in these beats. The last eight beats are yet again associated with deep S waves. The various QRS manifestations are dependent on critical rate. Thus, at a sinus cycle of 0.83 sec or less, the beats have a relatively short S wave. With a sinus cycle greater than 0.83 sec, the beats have a deep S wave. The manifestations are thus an expression of aberrant ventricular conduction which is dependent on critical rate.

**CASE 3**

The ECG (Fig 5, a continuous strip of lead V1) reflects very long P-P intervals, which range from 1.98 to 2.14 sec. This represents either (a) a marked sinus bradyarrhythmia of 28 to 32 beats/minute, or (b) a sinus rate of 56 to 64/minute complicated by 2:1 sinoatrial block. The first conducted sinus beat is the second beat of the top strip. This is followed by an AV nodal escape beat. The ensuing P wave occurs 0.38 sec after the escape beat and is conducted with right bundle branch block aberration. The sequence of escape and capture constitutes a form of escape-capture bigeminy. The next escape beat (the last beat in the top strip) occurs synchronously with the P wave (concealed within the QRS complex), whose impulse is consequently interfered with and not conducted. These conduction sequences occur throughout the tracing. The capture beats are associated with the following forms of intraventricular conduction:

1. **Normal intraventricular conduction**: This occurs with the second beat of each strip and the last beat of the bottom strip. These are associated with R-P intervals of 0.66, 0.62, 0.60, 0.64, and 0.52 sec, respectively.

2. **Right bundle branch block**: This occurs with the fourth beats of the first and second strips, which are associated with R-P intervals of 0.35 sec.

3. **Diminished S wave**: This is reflected by the fourth beat of the third strip, and is associated with an R-P interval of 0.38 sec.

The form of the intraventricular conduction is thus dependent on the R-P interval. The late impulses of relatively long R-P intervals are associated with normal intraventricular conduction. The early impulses of relatively short R-P intervals are associated with right bundle branch block conduction. An intermediate R-P interval is associated with a QRS complex that merely shows a diminished S wave.

**CASE 4**

The ECG (Fig 6, a continuous strip of lead MCL1) shows a basic fast sinus rhythm in the upper strip. The P-P intervals measure 0.66 sec, representing a rate of 90/minute. The second beat in the top strip is a ventricular extrasystole. The rhythm is further complicated by atrial extrasystoles, which are conducted with right bundle branch block aberration. These are represented by the 9th and 14th beats in the top strip, and the 5th and 10th beats in the bottom strip. The P' wave of each atrial extrasystole is superimposed upon and deforms the ST segment of the preceding beat. The second atrial extrasystole of the bottom strip falls during the vulnerable period of the atria and precipitates atrial fibrillation, as is evidenced by the ensuing fast irregular rhythm. Some of the beats during the atrial fibrillation also reflect the rsR' configuration of right bundle branch block aberration. The fourth beat of the top strip is of particular interest and merely reflects a diminution of the S wave as the only change. This beat is preceded by a long cycle occasioned by the pause of the ventricular extrasystole. A long pause, by virtue of the Ashman phenomenon, will increase the ensuing refractoriness and therefore favor aberration. The diminished amplitude of the S wave thus represents a degree of aberration which, in view of the classic right bundle branch block aberration associated with the atrial extrasystoles, represents an early phase of right bundle branch block. The diminution of the S wave in the last beat of the top strip and the last

**FIGURE 5.** Electrocardiogram (a continuous strip of lead V1) of case 3.

**FIGURE 6.** Electrocardiogram (a continuous strip of lead MCL1) of case 4.
two beats of the bottom strips probably represent the same phenomenon.

**Comment**

The rhythm strips of all of the cases depicted above show intermittent right bundle branch block aberration, during which a diminished S wave in right-oriented leads appears as the earliest manifestation of incomplete right bundle branch block. This stimulated the search for this manifestation in serial 12-lead ECGs.

**Cases 5 to 8**

An analysis of 11,000 ECGs of healthy aviation personnel yielded 25 cases of right bundle branch block. Four of these cases had two preceding ECGs with normal (narrow) QRS configurations, presented here as cases 5 to 8. These ECGs preceding the complete right bundle branch block were recorded on two different dates, the intervening times ranging from one to seven years. An example is shown in Figure 7. The amplitudes of the S waves in leads V1 and V2 as well as the R waves in lead V6 of all four cases are shown in Figure 8. All four cases showed a diminution of the S wave in lead V2. Two cases had a diminution of the S wave in lead V1, one showed no change, and one had a minimal increase of 1 mm. The R wave amplitude in lead V6 was diminished in two cases and remained the same in the other two. Right bundle branch block was observed in the tracings of all four cases recorded one to five years later.

**Discussion**

The right bundle branch block form of aberration in the rhythm strips of cases 1 to 4, and the sequential, time-spaced, 12-lead ECGs of cases 5 to 8 reflect the earliest manifestation of right bundle branch block as a diminution of the S wave amplitude in right-oriented leads, particularly lead V2.

**Mechanism**

Normal ventricular activation begins in the left side
In the case of complete right bundle branch block, conduction through the right bundle branch is no longer possible. The impulse coming from the left side of the interventricular septum therefore must "jump" a physiologic intraseptal barrier and then proceed through ordinary myocardial tissue, in contrast to the specialized conducting system, to activate the right ventricle (Fig I). Since ordinary myocardial tissue is a poor conducting medium, conduction is slow, producing a relatively large, late, unopposed force.

In the case of incomplete right bundle branch block, however, conduction through the right bundle branch is still possible but is delayed. The effect of this is shown in Figure 10. Delay within the right bundle branch causes a delay in the right paraseptal force (vector 3c, Fig 10) which, as a result, now occurs synchronously with the free wall forces (vectors 3a and 3b, Fig 10). And since vector 3c is directed slightly opposite vector 3b, it will diminish the magnitude of the resultant vector 3, which is responsible for the S wave in lead V$_2$. In other words, both vectors 3a and 3c counter vector 3b (Fig 10). A slight delay in conduction through the right bundle branch will thus cause a diminution in the S wave of lead V$_2$. There should also be a diminution of the R wave in lead V$_r$. This has previously been noted.  

Diminution of the R wave in lead V$_r$ was observed in two of the four cases, with no change in the other two. A possible reason for this lack of consistency is that lead V$_r$ is more remote from the heart than lead V$_2$ and thus subject to more variations in amplitude due to such factors as variations in body build and the amount of intervening lung tissue. The same probably applies to lead V$_1$ and requires further study. Since, however, the right paraseptal vector is the most affected, the effect of this would most likely be evident in lead V$_2$, which is directly oriented to this vector (Fig 9 and 10).

**The Forms and Progression of Right Bundle Branch Block**

The earliest manifestation of right bundle branch block, and its progression through the phases of incomplete right bundle branch block to complete right bundle branch block, are illustrated in Figure 11. The first sign of bundle branch block is the diminution of the S wave in lead V$_2$. Further progression of the right bundle branch block leads to slurring or notching of the upstroke of the S wave in lead V$_2$ (case 2 and diagram C, Fig 11). This manifestation previously has been the earliest described manifestation of incomplete right bundle branch block. Further increase in the right bundle branch block results in an r' deflection in lead V$_r$, and the QRS complex assumes a triphasic configuration (case 2, diagram D, Fig 11). With further progression, the r' deflection becomes increasingly taller (diagram E, Fig 11). Complete right bundle branch block is characterized by a widening of the very tall R' deflection with an apical notch or plateau. The duration of the QRS complex is thereby increased to 0.12 sec or longer (diagram F, Fig 11). The S wave is much reduced in amplitude and, with the maximal degree of right bundle branch block, may indeed disappear completely.

The development of right bundle branch block is thus characterized empirically by two major manifestations in lead V$_2$:

1. Progressive diminution of the S wave.
2. Progressive enlargement of the r' or R' wave with
final widening of this deflection. This process begins slightly later than, and overlaps with, the process leading to diminution of the S wave.

ACKNOWLEDGMENT: I am indebted to Dr. Alan Lindsay for the electrocardiogram shown as Figure 6.

REFERENCES

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**Hemodynamic Monitoring/Patient Care and Pulmonary Artery Catheterization**

This continuing education program will be held at the Johns Hopkins Medical Institutions, 725 North Wolfe Street, Baltimore, March 23-24. Sponsor is the Department of Anesthesiology and Critical Care Medicine, The Johns Hopkins Medical Institutions. For information, contact the Program Coordinator, Office of Continuing Education, Turner 22, 720 Rutland Avenue, Baltimore 21205 (301:955-6046).

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**Current Status of Tissue and Mechanical Heart Valve Replacement**

St. Joseph's Hospital Foundation and the Division of Cardiovascular Surgery will sponsor this international seminar in Tampa, Florida, April 27. For further information, please contact Dennis Pupello, M.D., 3001 West Buffalo Avenue, PO Box 4227, Tampa 33677 (813:870-4130 or 875-8988).