Simulated 2:1 Left Bundle-Branch Block
Normalization of the Left Bundle-Branch Block Pattern by Ventricular Extrasystoles

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VENTRICULAR extrasystoles can occur anywhere in the cardiac cycle. When
they occur toward the end of diastole they may be synchronous with the next conducted
impulse resulting in a fusion complex. We report a case of left bundle-branch block as-
associated with end-diastolic ventricular extrasystoles that occur in bigeminal rhythm; this
results in fusion complexes that apparently normalize the bundle-branch block pattern.
The resulting arrhythmia simulates 2:1 left bundle-branch block.

Case Report

The electrocardiograms are those of a 35-year-old African woman who died of hypertensive
heart disease with complicating pleural empyema. Figure 1 (continuous strip of standard lead II)
shows a basic pattern of left bundle-branch block with alternate complexes becoming progressively
more normal, giving the appearance of 2:1 left bundle-branch block. The P-R intervals of all
the beats are constant. The rhythm is interrupted by a short period of paroxysmal ventricular
tachycardia with retrograde conduction to the atria. This sequence of events is again shown in
figure 2 (continuous strip of lead V1 in the same recording). The beginning of this strip shows
left bundle-branch block only in the first three complexes; this is followed by gradual, progres-
sive, normalization of alternate complexes, so that eventually a pattern of 2:1 left bundle-
branch block appears.

The mechanism of this arrhythmia becomes apparent from another section of the same re-
recording (fig. 3) which shows the same left bundle-branch block pattern with alternate ventricular extrasystoles having the same basic pattern as the complexes of the ventricular tachycardia. However, their occurrence in end-diastole syn-
chronously or near synchronously with the supraventricular impulses results in various fusion
complexes, some of which normalize the bundle-branch block pattern.

Discussion

When impulses from two origins (e.g., supraventricular and ventricular or ventric-
ular from two foci), discharge synchronously or near synchronously, simultaneous invasion
of the ventricles may occur, each impulse stimulating part of the ventricular muscula-
ture. These impulses may modify the QRS complex so that its contour is intermediate
between that of the unmodified individual or principal complexes. As the two impulses
usually discharge with varying relationship to each other, e.g., parasystole, fusion com-
plexes of various types may result, depending on the amount of ventricular muscle con-
trolled by each pacemaker.

Bisten and his associates have shown experimentally that, in the presence of left bun-
dle-branch block, the occurrence of a ven-
tricular extrasystole in end-diastole—between
the peak of the P wave and the beginning of
the QRS complex—from a focus in the ven-
tricle of the blocked side may result in a
fusion beat that normalizes the bundle-branch
block pattern.

In left bundle-branch block, left ventricular
activation occurs late. This may, however, be
counterbalanced by a ventricular extrasystole
originating in the left ventricle more or less
synchronously with supraventricular activation.
Thus, left and right ventricular activation
occur together, and normalization of the
complex results.

Various types of fusion complexes may
occur depending on the timing of the ectopic
and ventricular discharges. When the ectopic
focus discharges near the P wave of the ensu-
ing sinus beat, the ventricles are activated
almost entirely by the ectopic stimulus and
the QRS complex is bizarre. The later the
ectopic stimulus occurs, the greater is the op-
portunity for synchronism with the supraventricular impulse and a consequent fusion complex that normalizes the pattern of bundle-branch block. This is demonstrated in figure 3. The true ectopic ventricular complex may be seen in the first two beats of the ventricular tachycardia (complexes 25 and 26). The true supraventricular complex—the left bundle-branch block pattern—can be seen in complexes 7, 9, 11, 13, 15, 17, 19, 20, 21, and 23. The other complexes represent varying degrees of fusion that depend on the timing and relationship of the supraventricular and ectopic impulses. For example, complex four occurs just after the P wave, well before the arrival of the next supraventricular impulse, and ventricular activation is therefore entirely by the ectopic stimulus; the complex thus resembles that of the ventricular tachycardia. Complex 22 resembles the true bundle-branch block pattern. Activation of this complex is therefore predominantly supraventricular with the extrasystolic impulse occurring late and thus influencing only the terminal deflection of the bundle-branch block pattern. The resulting pattern resembles a normally conducted supraventricular complex. Note that the P-R interval of this fusion complex is the same as that of the pure conducted beat. Other extrasystolic complexes represent varying degrees of fusion between these two extremes. For example, complex six probably represents equal activation by supraventricular and extrasystolic impulses; normalization is again evident. The pattern of complexes 2, 4, 8, 10, 12, 14, 16, and 18
bear a greater resemblance to the true ectopic complex and are therefore a result of dominant ectopic activation.

Since the coupling interval of the extrasystoles approaches the sinus cycle length, the different degrees of ventricular fusion may be caused by sinus arrhythmia or variation in the coupling intervals or both. The variation in the fusion complexes in our case was due predominantly to the presence of slight sinus arrhythmia (fig. 3, mean sinus cycle length 0.62 second; range 0.60 to 0.64 second). The coupling intervals (fig. 3) are constant at 0.60 second except for the intervals between complexes 19 and 20 and 21 and 22; these intervals are at least 0.62 and 0.63 second respectively (the modification of the terminal vector only, makes accurate measurement difficult).

The mechanism of apparent 2:1 left bundle-branch block seen in figures 1 and 2 now becomes explicable. The extrasystoles occur toward the end of supraventricular activation, affecting only the terminal deflection and gradually encroaching further on the bundle-branch block complex until normalization results. This is well seen in the beginning of each recording; in figure 1 the ter-

Figure 3
Electrocardiogram (continuous strip of lead V1). Full description in text. A, atrial level; A-V, nodal level; V, ventricular level.
minal deflection of alternate bundle-branch block complexes is progressively more affected until normalization results; in figure 2 this commences with the fourth complex.

True 2:1 bundle-branch block is a rare arrhythmia. The reason for this is that the bundle branch below the blocked area is activated by the impulse from the contralateral branch and is thus refractory at the time of arrival of the next supraventricular impulse.

A 2:1 bundle-branch block may be simulated by two different mechanisms: 1. Extrasystolic ventricular bigeminal rhythm, during sinus rhythm with normal intraventricular conduction, when the extrasystoles occur late in diastole. In such cases it is the ectopic impulse that simulates the bundle-branch block. Careful inspection, however, will reveal that the P-R interval of the so-called bundle-branch block complex is shorter than that of the normal complex. 2. The mechanism of the arrhythmia described here. In this case it is the apparent normal complex that is due to a fusion between the bizarre pattern of the ventricular extrasystole and the bundle-branch block pattern of the supraventricular impulse. As the terminal deflection is chiefly affected, the P-R interval of the fusion complex is the same as that of the conducted impulse.

Four conditions are necessary for the occurrence of this arrhythmia. 1. The supraventricular impulses must have a bundle-branch block pattern. 2. There must be a discharging ectopic focus in the ventricle of the blocked side. 3. The focus must discharge in bigeminal rhythm. 4. The ectopic impulse must discharge in end-diastole synchronously with the supraventricular impulse.

This arrhythmia should be suspected when in apparent 2:1 bundle-branch block, the so-called normal complexes show slight variation in shape affecting particularly the terminal deflection.

Summary

A case is presented of end-diastolic ventricular extrasystoles occurring in bigeminal rhythm during left bundle-branch block; this results in fusion complexes that normalize the bundle-branch block pattern and simulate 2:1 left bundle-branch block.

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References

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