management of the interaction between amiodarone on warfarin, and adjustment of the warfarin dose is a better solution. We recommend that the maintenance dose of warfarin be halved when amiodarone therapy is begun, irrespective of the dose of amiodarone prescribed.

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References

Deceleration-dependent Left Bundle Branch Block: A Spectrum of Bundle Branch Conduction Delay

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SUMMARY Two cases of deceleration (bradycardia)-dependent left bundle branch block showing varying degrees of bundle branch block are described. The degree of bundle branch delay related directly to the duration of preceding cycle lengths. These observations support spontaneous diastolic depolarization as the mechanism of deceleration-dependent aberrancy.

INTRAVENTRICULAR ABERRATION associated with acceleration of the heart rate is a well-recognized phenomenon that appears to be related to an inappropriate response of the refractory period to a change in heart rate. Dressler reported the first case of a bundle branch block occurring with deceleration of the heart rate.1 Deceleration-dependent aberrancy (DDA) is much less common than aberrancy due to acceleration of the heart rate, and its mechanism less clear. Massumi2 reported four additional cases and suggested criteria for diagnosis of DDA. In 1973, Rosenbaum and associates3 added 14 cases. Enhanced phase 4 depolarization was proposed by Singer et al.4 as the mechanism of DDA. They suggested that with a longer cycle, the cell membrane depolarizes and activation is initiated from a reduced resting membrane potential resulting in slowing or block of conduction. This phenomenon has been referred to as diastolic or phase 4 block. If diastolic depolarization is indeed operative, one can assume that patients with incomplete bundle branch block at rates between those that cause complete bundle branch block and those with normal intraventricular conduction would be encountered. To our knowledge, no such cases have been reported. In this paper, we describe two patients who had DDA associated with varying degrees of bundle branch block.

Case Reports

Case 1
The tracings were recorded in a 20-year-old woman after surgery for congenital aortic stenosis, and represent DDA. The basic rhythm in figure 1 is a sinus arrhythmia with PP intervals of 680–1080 msec. The PR interval is constant at approximately 130 msec. In each lead the QRS complexes vary from 80–130 msec. The shortest RR cycles are followed by QRS complexes of normal duration, intermediate RR cycles by QRS complexes of intermediate duration (incomplete
left bundle branch block (LBBB), and the longest RR cycles by complexes with complete LBBB. Although the ST-T segments are slightly abnormal when intraventricular conduction is normal, these changes gradually with prolongation of the QRS complex. Subtle changes in QRS duration with deceleration of the sinus rate are thus further confirmed by appearance of the secondary ST-T changes.

The first five QRS complexes in lead I illustrate the direct relation of QRS duration to sinus rate. The first RR interval measures 740 msec and is terminated by a QRS of 90 msec. The third QRS follows an RR interval of 870 msec, lasts 120 msec, and has complete LBBB morphology. The fourth QRS follows an RR interval of 840 msec, intermediate between the first two RR intervals, lasts 110 msec and has incomplete LBBB morphology.

Similar relationships are noted in the remainder of figure 1. For example, the third through sixth QRS complexes in lead aVL reveal transition from incomplete LBBB to complete LBBB with lengthening of the RR interval from 720 to 1040 msec.

In figure 2, the QRS duration is related to the preceding RR interval. Six QRS complexes from lead I and four complexes from lead V5 are displayed in order of cycle length, which increased from 720 to 1080 msec. In lead I, the QRS prolongs from a normal QRS after RR cycles of 720–740 msec, to incomplete LBBB terminating RR cycles of 840 and 870 msec, and finally to complete LBBB after RR cycles of 920 and 960 msec. The changing QRS morphology is seen especially well in lead V5, where an increase in cycle length from 720 to 760 msec is associated with an initial slur on the upstroke of the QRS, prolongation of the QRS and a decrease in the R-wave amplitude.

The relation of QRS duration to the preceding RR cycle is illustrated in figure 3. All QRS complexes after RR cycles of 760 msec or shorter are normal. Complete LBBB follows RR cycles greater than 930 msec, while incomplete LBBB terminates RR cycles of 760–920 msec. As expected, there was some overlap between the normal QRS and incomplete LBBB at cycle lengths of approximately 770 msec and between incomplete LBBB and complete LBBB at a cycle length of 920 msec.

Case 2

Figures 4, 5 and 6 were recorded in a patient with atherosclerotic cardiovascular disease. Figure 4 represents a continuous lead II. The basic rhythm is sinus with a PP interval of approximately 600 msec with 3:2 and 2:1 atrioventricular block. Longer RR cycles are terminated by LBBB and shorter cycles by a normal QRS. Thus, a deceleration-dependent LBBB is present. The third and eighth QRS complexes in the middle strip of figure 4 follow RR cycles of 1080 msec, are of intermediate duration and represent incomplete LBBB. The cycles terminated by incomplete LBBB are shorter than cycles preceding complete
LBBB, but longer than cycles terminated by normal QRS complexes.

Figure 5 illustrates sinus rhythm with varying atrioventricular block. QRS complexes after RR intervals of 0.72 and 1.00 second are of normal duration. LBBB follows RR intervals of 1.20, 1.24 and 1.28 seconds, and incomplete LBBB follows cycles of 1.08 seconds. This record demonstrates DDA with transition from normal QRS to incomplete and complete LBBB with lengthening of the preceding RR interval.

Figure 6 depicts the relation between QRS duration and the preceding cycle length. RR intervals of 1.040 msec or shorter are followed by normal QRS and RR intervals of 1.110 msec or longer by complete LBBB; cycles of approximately 1.080 msec are terminated by incomplete LBBB.

Discussion

The apparent paradox of delayed intraventricular conduction with deceleration of rate has been of interest to both clinical electrocardiographers and basic electrophysiologists. Although deceleration-dependent complete bundle branch block has been described, the two cases presented here are the only reported examples of intraventricular conduction duration varying directly with preceding cycle length. These observations have a bearing on the mechanism of DDA.

Massumi proposed five criteria for the diagnosis of bradycardia-dependent bundle branch block. Our two cases met all five of these criteria. First, all complexes were conducted from the atria to the ventricles through the normal atrioventricular pathway. Second, atrial fibrillation or flutter was not present, which rules out the possibility of the wide complex being ventricular in origin. Third, the phenomenon was recorded in two or more consecutive QRS complexes. If the bundle branch block pattern occurred as an isolated event, the QRS could represent a ventricular escape coincidentally related to a P wave. Fourth, the QRS complexes followed a constant PR interval, which makes a varying bilateral bundle branch block unlikely. Patients with bilateral bundle branch block can show narrowing of QRS morphology with an increase in heart rate and bilateral, uniform lengthening of conduction that would result in prolongation of the PR. Fifth, supernormal conduction with normalization of the QRS would not apply in our cases, as even the shortest RR cycle is too long for the conducted QRS to decrease during the supernormal period of bundle branch recovery.

There is a remote possibility that concealed depolarization of the left bundle branch would result in a short manifest bundle-to-bundle interval, causing an acceleration-dependent bundle branch block. Such foreshortening of the cycle was evident with fully conducted impulses, and yet these shorter RR cycles were terminated by normal QRS. Thus, absence of bundle branch block with the shorter cycle lengths argues against concealed bundle branch conduction as a cause of QRS aberrancy.

The most widely accepted mechanism for DDA was suggested by Singer et al., who proposed that DDA is related to abnormal spontaneous diastolic (phase 4) depolarization of the conduction system in a diseased heart. Because of the spontaneous depolarization after longer cycles, the cell is activated from a less negative resting membrane potential and conduction delay or block may result. If the conduction is slower or completely blocked in one of the bundles, a deceleration-dependent bundle branch block results.

Gambetta and Childers, while supporting the concept of phase 4 block, raise some arguments against this hypothesis. They point out that block should not necessarily be the all-or-none phenomenon previously reported, but that incomplete bundle branch block should occur at some critical cycle length. Rosenbaum postulated a time during which incomplete bundle branch block would be expected and beyond which complete bundle branch block would be recorded. The findings in our two cases demonstrate progressive QRS prolongation from normal to complete LBBB. The presence of varying degrees of LBBB, depending on the cycle length, is responsive to the issue raised above and supports the concept of
spontaneous phase 4 depolarization as the mechanism of DDA.

If spontaneous diastolic depolarization is allowed to reach the threshold potential, an escape would occur with a bundle branch block configuration opposite that of the conducted QRS complex. Such observation would add additional circumstantial evidence in support of diastolic depolarization as the mechanism for DDA.

Because the full width of the bundle branch must be affected to cause slowing of conduction, the thickness of the bundle branch has been an argument against the diastolic depolarization hypothesis. Similarly, there is experimental evidence that in some circumstances the diastolic depolarization may actually enhance conduction rather than slow it by bringing the resting potential closer to the threshold potential. Jalife et al. suggested that deceleration-dependent bundle branch block results not from phase 4 depolarization but “complex oscillatory changes of membrane properties of depressed bundle branch Purkinje fibers during diastole.” Despite these observations, there is ample electrophysiologic evidence in favor of diastolic depolarization as the mechanism for DDA, and the two cases reported here add strong electrocardiographic evidence.

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