Bundle Branch Block After Ventricular Tachycardia: A Manifestation of "Fatigue" or "Overdrive Suppression"

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Three cases of bundle branch block due to ventricular tachycardia are presented. Although the exact mechanism remains obscure, this phenomenon may be related to previously described acceleration-induced atrioventricular and bundle branch block.

Overdrive suppression of primary and secondary pacemakers and atrioventricular (AV) conduction may occur after spontaneous or pacing-induced acceleration of atrial (1-3) and ventricular (4,5) rates. In 1927, Scherf (6) reported suppression of right bundle branch conduction in the dog by rapid ventricular pacing. To the best of my knowledge, this phenomenon has not been described in human beings. This report presents three cases of bundle branch block on resumption of sinus rhythm after ventricular tachycardia.

Case Reports

Case 1. The electrocardiogram illustrated in Figure 1 was recorded in a 30 year old man with severe three vessel atherosclerotic coronary artery disease with complete occlusion of the right coronary artery. Repeated electrocardiograms documented acceleration-dependent left bundle branch block. During normal intraventricular conduction, nonspecific ST-T changes were present. After the cineangiographic study was completed and while the patient was still in the laboratory, spontaneously terminating ventricular tachycardia (Fig. 1) was recorded. The first three sinus impulses after the ventricular tachycardia conduct with left bundle branch block. In parallel with shortening of the sinus interval from approximately 1.28 to 0.92 second, the intraventricular conduction normalizes.

Case 2. The electrocardiograms (lead V1) illustrated in Figure 2 were recorded in a 57 year old man with a history of anterolateral myocardial infarction complicated by congestive heart failure and recurrent and refractory ventricular tachycardia. Panels A and B were inscribed at different times. In panel A, ventricular tachycardia with a right bundle branch block configuration and an RR interval of 0.52 second is followed by sinus rhythm with a PP interval of 0.68 second and a normal PR interval and QRS complex. The record in panel B is continuous and illustrates ventricular tachycardia with a left bundle branch block configuration and an RR interval of 0.30 second that terminates spontaneously. The positive inscription superimposed on the last QRS complex of the tachycardia is an artifact. After resumption of sinus rhythm, the PR interval shortens gradually from 0.20 to 0.12 second. The first five QRS complexes are sinus in origin with a right bundle branch block configuration and are followed by normal intraventricular conduction. The RR interval foreshortens gradually from 0.84 to 0.80 second.

Case 3. The electrocardiograms (lead I) illustrated in Figure 3 were recorded in a 47 year old man with severe atherosclerotic coronary artery disease, manifest by inferior myocardial infarction and an acceleration-dependent left bundle branch block. Panel A was recorded at a paper speed of 25 mm/s, and panels B and C at a paper speed of 10 mm/s.

In panel A, bundle branch block conduction normalizes without any demonstrable foreshortening of the cardiac cycle. However, the acceleration-dependent origin of the left bundle branch block is confirmed in panel B, where the 0.75 second pause that follows the premature ventricular complex is terminated with a normal QRS complex. The configuration of the QRS complex preceding the premature ven-
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Figure 1. Left bundle branch block after termination of ventricular tachycardia.

Discussion

Scherf (6) was able to reinitiate right bundle branch block with bursts of rapid ventricular pacing by first inducing right bundle branch block with mechanical injury and allowing the conduction to recover. His studies indicate that "overdrive suppression" of bundle branch conduction is possible, provided the conducting tissue is in some way disturbed.

Our findings agree with those of Scherf in that all three patients had advanced heart disease, with acceleration-dependent left bundle branch block present in two patients. It has been our experience (7) that acceleration-dependent aberration is nearly always associated with significant heart disease. Interestingly, Scherf's studies (6) also appear to be the earliest experimental demonstration of concealed intraventricular conduction; the concealed conduction of the ventricular impulses into the right bundle branch manifesting as right bundle branch block.

Mechanism of post-tachycardia bundle branch block.

The bundle branch block in our patients may be similar to acceleration-dependent atrioventricular (AV) block (3-5). Such a relation is supported by Case 2, in which the ventricular tachycardia induced delay of both AV and bundle branch conduction. Similarly, assuming that the bundle is depolarized repeatedly by the ventricular impulses, the mechanism of the post-tachycardia bundle branch block may be similar or the same, which is responsible for persistence of bundle branch block in patients with acceleration-dependent bundle branch block at cycles longer than the "critical" cycle that initiated the aberration. This paradox, seen in acceleration-dependent aberration, is usually ascribed to delayed transseptal concealed conduction from the contralateral conducting bundle, resulting in a bundle branch to bundle branch interval shorter than the manifest QRS cycle (8-11). "Fatigue" and "overdrive suppression" (7) have also been suggested as possible mechanisms of the paradoxical delay of normalization of bundle branch conduction.

Relation of heart rate to suppression of conduction.

The duration of the paradoxically suppressed conduction in acceleration-dependent aberration is dependent on the heart rate; the more rapid the rate, the more likely the paradox and the longer the recovery time of the bundle conduction. This relation of the heart rate to presence or absence of the paradox may explain the absence of post-tachycardia bundle branch block in Figure 2A. In this trace, the interectopic interval of the ventricular tachycardia is 0.52 second and in Figure 2B, the interval is 0.30 second. It is also possible

Figure 2. Right bundle branch block after termination of ventricular tachycardia.

Figure 3. Left bundle branch block after ventricular tachycardia in panel C. Paper speed 25 mm/s in panel A and 10 mm/s in panels B and C.
that in Figure 2A, the site of origin of the ventricular tachycardia is left ventricular and, thus, the tachycardia may not reach the right bundle branch. "Fatigue" and "overdrive suppression" of bundle branch conduction in acceleration-dependent aberration and that occurring after ventricular tachycardia suggest that adjustment of conduction to the new heart rate, in this case normalization of conduction, is time-dependent (12). The cellular electrophysiologic determinants of conduction responsible for the post-tachycardia bundle branch block remain purely speculative, but may include, among others, decrease in upstroke of phase 0, reduction in resting membrane potential or shift of the membrane responsiveness to the right.

References