Tachycardia-Dependent Left Bundle Branch Block
Associated with Bradycardia-Dependent Variable Left Bundle Branch Block

A Case Report

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SUMMARY
This report describes tachycardia- and bradycardia-dependent left bundle branch block in a patient who exhibited normal intraventricular conduction with impulses intermediate in timing. During bradycardia, longer cycles were associated with increasing degrees of incomplete left bundle branch block. These clinical observations support the existence of slow Phase 4 depolarization of latent pacemaker cells as one of the mechanisms for abnormalities of conduction.

Additional Indexing Words:
Aberrant conduction  Refractory period  Bradycardia  Tachycardia

Refractoriness of conducting tissue is intimately related to recovery time. When a pathological process causes unequal refractoriness of the bundle branches (or fascicles) relatively early impulses will be conducted along one branch only, resulting in the well-known entity of tachycardia-dependent intermittent bundle branch block.1-6 Recently, a rarer form of aberration has received emphasis. In these cases, relatively late impulses are conducted abnormally but normal intraventricular conduction occurs with earlier impulses (bradycardia-dependent bundle branch block).7-21 Rarely both tachycardia- and bradycardia-dependent bundle branch block (or hemiblock) occur in the same patient.11, 20, 21

This report describes tachycardia- and bradycardia-dependent bundle branch block (or hemiblock) a patient who exhibited normal intraventricular conduction with impulses intermediate in timing. During bradycardia progressive prolongation of the cycle lengths induced increasing degrees of incomplete LBBB22-24 which eventually culminated in complete (or near complete) LBBB.

Report of a Case
The electrocardiograms presented in this report were recorded from a 72-year-old woman with aortic stenosis and congestive cardiac failure. The abnormalities were consistently present during spontaneous rhythm over a three week period and were unchanged five months later. The morphology of the QRS complexes in leads I, aVL, V5, and V6 (left ventricular leads) were essentially similar during normal and abnormal ventricular conduction. Lead V6 was selected for illustrative purposes. All the conduction abnormalities were present during the spontaneous sinus arrhythmia but carotid sinus pressure was used to induce gradual slowing in the rate to better delineate the various conduction abnormalities. No ventricular escape beats were seen after long periods of bradycardia. There was no change in drug therapy over the five month period except for diuretics.

Figure 1 shows lead V6 recorded at double sensitivity. The upper two strips are continuous and show tachycardia-dependent LBBB. When the cycle length shortens to 0.80 sec LBBB appears abruptly. Longer cycle lengths are associated with relatively normal intraventricular conduction. The lower strips show both tachycardia- and bradycardia-dependent LBBB. Very early impulses with R-R intervals of 0.70, 0.78, and 0.80 sec are conducted with LBBB. The fourth beat in the lower strip is either an atrial or junctional extrasystole. Late impulses with preceding R-R intervals of 1.32 and 1.42 sec are also conducted.

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with LBBB. Intermediate intervals are associated with normal intraventricular conduction. Carotid sinus compression was applied momentarily at the arrows.

Figure 2 shows lead V6 recorded on another day. The upper two strips are continuous. Sinus arrhythmia causes varying cycle lengths. The fourth QRS complex in the bottom strip is either an atrial or junctional extrasystole conducted with a high degree of LBBB (R-R = 0.70 sec). Relatively late sinus impulses are conducted with LBBB, the later the impulse the higher degree of LBBB. The earliest manifestation of incomplete LBBB manifested by the disappearance of the tiny initial Q wave occurs at an R-R interval of 1.15 sec (second last beat in second strip). Higher grades of incomplete LBBB terminate longer R-R intervals of 1.16 and 1.19 sec. This is well seen in the third and last complexes in the second strip, and the third complex in the bottom strip. Note the prominent slurring of the initial upstroke of the QRS complex, delayed intrinsicoid deflection and slight prolongation of the QRS complex. Still higher grades of incomplete LBBB follow R-R intervals of 1.20, 1.30, and 1.36 sec. Normal intraventricular conduction occurs with R-R intervals less than 1.5 sec and more than 0.70 sec.

Figure 3 consists of lead V6 recorded on a different day at a paper speed of 50 mm per second. It shows with greater clarity the varying degrees of LBBB seen with bradycardia. Carotid sinus pressure was applied in the interval indicated by the arrows. All beats are sinus conducted. QRS complexes terminating R-R intervals of 1.38 sec or less are normally conducted. The QRS complex terminating an R-R interval of 1.52 sec
Figure 3

Lead V6 recorded at double speed showing bradycardia-dependent LBBB with increasing impairment of conduction as a function of time. Carotid sinus pressure was applied in the interval indicated by the arrows.

displays the earliest recognizable sign of incomplete left bundle branch block, i.e., disappearance of the tiny initial Q wave (third complex in third row). A QRS complex terminating an R-R interval of 1.53 sec (second complex of the fourth row) or an interval of 1.58 sec (third complex of the fifth row) reflects a higher degree of LBBB. Note the prominent slurring of the initial part of the QRS complex in these beats. Complexes terminating still longer intervals of 1.72 sec (second complex of the third row) and 1.74 sec (first complex of the bottom row) are conducted with the highest degree of LBBB seen in this patient.

Figure 4 is a recording of lead 2 (upper two rows are continuous) showing tachycardia- and bradycardia-

Figure 4

Lead V6 showing tachycardia- and bradycardia-dependent LBBB. Note that in the incomplete LBBB phase (arrows) the mean frontal plane axis does not change. Upper two rows are continuous.
VARIABLE LEFT BUNDLE BRANCH BLOCK

dependent LBBB. Note that the complexes labeled by the arrows exhibit incomplete LBBB reflected by alteration of the initial forces but no change in the mean axis.

Discussion

When the basic rate accelerated, the transition from normal to abnormal intraventricular conduction was abrupt. In contrast, late sinus impulses during bradycardia were conducted with varying degrees of incomplete LBBB; the later the impulse the more advanced the degree of LBBB.22-24 Disappearance of the tiny initial Q wave in the left ventricular leads (as exemplified by V6) constituted the earliest recognizable sign of incomplete LBBB. Increasing degrees of LBBB were manifested by progressive slurring of the proximal limb of the QRS complex, delayed intrinsicoid deflection and an increase in QRS duration without the appearance of striking secondary ST-T wave changes. The spectrum of incomplete LBBB during bradycardia in our patient therefore corresponds with the various phases of incomplete LBBB previously reported by Schamroth and Bradlow,5 and Barold et al.6 in tachycardia-dependent, incomplete and complete intermittent LBBB.

The concept of Phase 4 depolarization of latent pacemakers25 stands out as the most rational and physiologically acceptable of the several mechanisms proposed to explain bradycardia-dependent bundle branch block. Conduction velocity in Purkinje fibers is closely related to the amplitude of the action potential and the maximum rate of change of membrane potential (dv/dt) during its upstroke (Phase 0). Both these variables are in turn closely dependent upon the level of membrane potential at the time of excitation.25-27 Thus, a critical reduction of membrane potential at the time of excitation will cause delayed conduction by diminishing the amplitude and dv/dt of the action potential. This holds true when the reduced level of membrane potential originates early in the cycle from incompletely repolarized fibers (Phase 3) or later in the cycle from slow Phase 4 depolarization causing a progressive decrease in diastolic potential. The His-Purkinje system contains a large number of latent pacemaker cells which may acquire enhanced automaticity (gradual lowering of diastolic potential) from ischemia, stretch, and many other factors.25 Spontaneous Phase 4 depolarization of these latent pacemaker cells may therefore result in slowing of conduction by influencing the amplitude and dv/dt of the action potential.

Impairment of conduction due to Phase 4 depolarization should theoretically be more marked with longer cycle lengths as the slow rising slope of diastolic depolarization is allowed to proceed. Yet, a progressive decrease in conduction velocity as a function of time has rarely been observed clinically in bradycardia-dependent intraventricular block. Garcia and Rosenbaum did not mention it in their recent report on this subject.21 The electrocardiograms in Friedberg's report19 and that of Singer and Ten Eick17 do show increasing degrees of aberrancy but only with slight changes in cycle length. The most striking example of this electrophysiological prediction was recently described by Elizari et al.20, who reported tachycardia- and bradycardia-dependent left anterior hemiblock in the same patient. During bradycardia progressive lengthening of the R-R intervals clearly induced increasing degrees of left anterior hemiblock. In that respect the increasing degrees of incomplete LBBB in our case also coincide with the proposed electrophysiological mechanism but the lesion was presumably preidi- sional (main stem) because the mean axis in the frontal plane did not change significantly during the incomplete LBBB phase. The critical cycle length required to produce bradycardia-dependent LBBB in our patient varied somewhat from day to day, presumably because the following variables were changing either singly or in combination: 1) membrane potential at the onset of Phase 417, 2) the slope of Phase 4 depolarization, which is influenced by electrolytes, stretch, anoxia, catecholamines, etc.17, or 3) responsiveness curve: relationship between the maximum rate of depolarization during Phase 4 and the level of membrane potential at the time of excitation.25-27

The association of both tachycardia- and brady- cardia-dependent bundle branch or fascicular block in the same patient is relatively rare. Garcia and Rosenbaum recently reported eight such cases unmasked by carotid sinus pressure (seven cases showed intermittent LBBB and one, intermittent left anterior hemiblock; the latter was subsequently described in detail by Elizari et al.20). Massumi's example11 (his Case 1 showing right bundle branch block) also represents an unquestionable example of this phenomenon. However, other electrocardiograms purporting to show this unusual combination either do not show the association restricted to the same bundle or fascicle or fail to exclude a separate ventricular (or fascicular) rhythm that might account for the bradycardia-dependent bundle branch block.18 Consequently, when carotid sinus
pressure unmasks bradycardia-dependent bundle branch block the diagnosis requires careful analysis to avoid confusion with a ventricular escape rhythm.\textsuperscript{11}

Elizari et al.\textsuperscript{21} postulated the existence of two areas with different cell populations at the site of the block to explain the electrophysiological mechanism of tachycardia- and bradycardia-dependent bundle branch block. The cells responsible for tachycardia-dependent bundle branch block (Phase 3 block) would have a prolonged recovery time (Phase 3) with a normal resting potential. Activation above a certain critical rate would therefore occur in the relative refractory period when repolarization is incomplete. The cells responsible for bradycardia-dependent bundle branch block (Phase 4 block) would have slight reduction in the membrane potential at the onset of Phase 4 together with enhanced diastolic depolarization. Both prolongation of Phase 3 and enhanced Phase 4 depolarization could however conceivably occur in the same cell population. Preliminary electrophysiological observations from isolated specimens of living diseased human hearts tend to support these contentions.\textsuperscript{17}

References

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