Tachycardia and Bradycardia-dependent Bundle Branch Block Alternans

Clinical Observations

HOWARD C. COHEN, M.D., IVAN D’CRUZ, M.D., EMANUEL R. ARBEL, M.D., RICHARD LANGENDORF, M.D., AND ALFRED PICK, M.D.

SUMMARY Eleven patients with tachycardia-dependent, bradycardia-dependent, or "pseudobradycardia-dependent" bundle branch block (BBB) alternans were studied. This classification is based on the following criteria: 1) When alternans is initiated by a sudden acceleration in ventricular rate, or it appears with aberration of the second beat after a pause, the alternans is tachycardia-dependent and results from a 2:1 bidirectional block in the affected bundle branch. 2) When alternans begins with the aberrant complex terminating a pause it is bradycardia-dependent; such an alternans results from alternating bundle branch cycle lengths and refractoriness, possibly produced by alternating transeptal retrograde penetration of the affected bundle branch. 3) In cases referred to as "pseudobradycardia-dependent BBB" alternans, a change from alternans to persistent BBB occurs as the cycle lengths; however, the disappearance of BBB with further increase of the cycle length proves the tachycardia-dependence of the conduction defect.

APPLICATION OF A UNIFYING CONCEPT of tachycardia and bradycardia-dependent bundle branch block (BBB) to the phenomenon of electrical alternans has not been reported previously. We have studied the electrocardiograms (ECG) of nine patients with alternating BBB. The proposed mechanisms by which tachycardia and bradycardia-dependent conduction abnormalities produce BBB alternans differ and the characteristics of each type of rate-dependent alternans can be recognized in the standard ECG.

I. Clinical Material

Fourteen ECGs and rhythm strips were obtained in eleven patients, each of whom had a rate-dependent intraventricular conduction defect. Six had tachycardia-dependent BBB, right sided in five and of indeterminant location in one. Examples are presented as cases 1 and 2. In two of these six patients, the effects of changes in heart rate mimicked transiently bradycardia-dependent alternating BBB, and were termed by us "pseudobradycardia-dependent alternans." An example is described as case 4.

Five patients had true bradycardia-dependent BBB. The BBB was left sided in four and right sided in one. An example of this group is presented as case 3.

Tachycardia-dependent Alternans

QRS complexes have the contour of a minimal degree of incomplete right BBB in the first strip of figure 1A (case 1) during sinus rhythm at a rate of 88/min. Sinus tachycardia at a rate of 112/min in the second strip, recorded several minutes later, is associated with development of right bundle branch block (RBBB) alternating with incomplete RBBB. The proposed mechanism of this type of alternans is shown diagrammatically in figure 1B, an enlargement of the rhythm strip during alternans. In all of the diagrams the dashed vertical lines represent conduction in the unimpaired bundle branch, and the solid vertical lines represent the extent of conduction in the impaired bundle branch. Arrows show the direction of conduction, single horizontal bars denote interruption of conduction, and double horizontal bars represent transseptal conduction. The brackets below, labelled A and B in figure 3B and 1 through 4 in figure 4, denote the varying intervals between consecutive successful or unsuccessful bundle branch depolarizations. We attribute the alternating aberrant QRS contour in figure 1 to a tachycardia-dependent 2:1 bidirectional block in the right bundle branch, that is to say, no retrograde concealed invasion of the blocked bundle branch occurred during the aberrantly conducted beats.

In figure 2 (patient 2), the first four QRS complexes represent a 2:1 ventricular response to a paroxysmal atrial tachycardia at a rate of 200 beats/min. The pause (1.12 sec) after spontaneous cessation of the tachycardia is terminated by escape of a single sinus or ectopic atrial beat (arrow) whereupon, after a ventricular cycle of 0.94 sec, the tachycardia resumes at a slower rate of 142, with a 1:1 A-V response. The first QRS complex of this second run of tachycardia is of normal contour; the next is aberrant attributable to its prematurity following the long preceding cycle. Alternans of QRS size persists for at least nine complexes, with a gradual transition of the aberrant beats to normal QRS configuration. The alternating aberrancy begins in the second beat after the pause, rather than in the beat terminating the pause, and is therefore an example of tachycardia-dependent aberration.

Bradycardia-dependent Alternans

Patient 3 was an 81-year-old man with acute anterolateral myocardial infarction and slight sinus arrhythmia with cycles varying between 0.84 and 0.96 sec, corresponding to rates of 71 and 63/min. In lead V1 of his ECG (fig. 3A), the first four beats in the upper panel show incomplete left bundle branch block (QRS = 0.10 sec). A blocked premature P wave (P') most likely is hidden within the T wave of the fourth beat (causing a taller T wave). The long

From the Cardiovascular Institute, Department of Medicine, Michael Reese Hospital and Medical Center, and the University of Chicago Pritzker School of Medicine.

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Address for reprints: Howard C. Cohen, M.D., Chief, Heart Station, Michael Reese Hospital and Medical Center, 29th Street and Ellis Avenue, Chicago, Illinois 60616.

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ventricular pause of 1.56 sec that follows is terminated by a wide QRS complex of "complete" left bundle branch block (LBBB) configuration (QRS = 0.14 sec). The degree of LBBB in this beat is thus bradycardia-dependent. With shortening of the ventricular cycle to 1.00 sec a transient QRS alternation between incomplete and complete LBBB is observed in the next four beats, which disappears once R-R intervals have shortened from 1.00 sec to 0.96 sec or less. The lower strip was recorded one-half hour later. The first five QRS complexes occur at regular intervals (0.92-0.94 sec) and show alternation in size and QRS duration. A conducted premature atrial beat (P') is most likely hidden within the T wave of the fifth complex. Its QRS complex, though occurring early (R-R' measuring 0.56 sec), is relatively narrow whereas the next QRS complex, occurring after a long pause (1.08 sec), is wide and again initiates alternation during shortening of the ventricular cycle to 0.92 to 0.88 sec. These relationships confirm a bradycardia-dependent intraventricular conduction defect.

Figure 3B shows an enlarged portion of lead I with a diagram of a proposed mechanism of alternans in the same patient. In contrast to figure 1B, it is postulated that the antegrade blocked bundle branch is entered retrogradely following transseptal conduction (concealed intraventricular conduction). Thus, the interval B between the concealed retrograde depolarization and the next antegrade depolarization of the left bundle branch will be relatively shortened. At this reduced interval propagation within the LBB does take place leading to a more narrow QRS complex. On the other hand, the interval A between the antegrade left bundle branch depolarization of the more narrow QRS complex and the next unsuccessful attempt at antegrade left bundle branch traversal will be longer than interval B, resulting in more delay in left bundle branch conduction and a pattern of "complete" LBBB. Thus, here, too, the alternation of QRS duration and contour is the consequence of alternation of cycle lengths measured from the last depolarization of the left bundle branch (whether antegrade or concealed retrograde) to the point of attempted antegrade conduction in the next beat. However, the longer of these cycles is followed by a pattern of "complete" LBBB and hence the conduction defect must be considered as bradycardia-dependent. This phenomenon occurs in the presence of precisely regular R-R intervals.

Pseudobradycardia-dependent Alternans

Patient 4 presented with spontaneous QRS alternans of the RBBB type at a regular rate. This abnormality was detected in a 72-year-old man who had had chest pain one day before. A rhythm strip of lead I showed RBBB on alternate beats at a sinus rate of 115 per minute (fig. 4). Less than one minute later a decrease in rate to 94 beats per minute converted alternans to persistent RBBB, apparently of the bradycardia-dependent type. However, a further slowing to 72 beats per minute produced persistent normal conduction. These observations indicate that the BBB is actually tachycardia-dependent.

The details displayed diagrammatically are as follows: at the initial fastest heart rate of 115/min, a 2:1 antegrade and retrograde block in the right bundle branch (RBB) was present because of bidirectional refractoriness (as in fig. 1B). Persistent aberrancy during initial slowing to 94/min can be accounted for by repetitive concealed transseptal and retrograde depolarization of the blocked bundle in each beat.

In the diagram, at 115/min, interval 1, from successful to attempted antegrade RBB conduction, and interval 2, from antegrade to attempted retrograde conduction, are both shorter than the RBB effective refractory period, and so there is bidirectional RBBB. As the rate slowed, a critical rate was reached, between 115/min and 94/min, at which interval 1 was still shorter than the effective refractory period, but interval 2 became longer, so that retrograde conduction became possible. At 94/min, interval 3, from retrograde to
attempted antegrade conduction stays shorter and interval 4 from retrograde to retrograde conduction remains longer than the RBB effective refractory period so that RBBB persists in every beat. As the rate slowed further, a new critical rate was reached between 94/min and 72/min, at which interval 3 became longer than the RBB effective refractory period, so that antegrade conduction occurred. Antegrade conduction persists because of the new longer interval 1. Of course, effective refractory periods may also be changing. Conduction or lack of it depends upon the relationship between the bundle branch interval and the immediate effective refractory period.

We have called this sequence of events pseudobradycardia-dependent alternans, because its true basis is that of tachycardia-dependent BBB, even though initially, persistent BBB appeared with slowing of the rate. The most sensitive factor depending on changes in cycle length appears to be the facility of concealed retrograde conduction within the RBB.

Discussion

We have shown that either of the two possible forms of electrical alternans, the tachycardia or the bradycardia-dependent type, may be initiated by a long R-R interval, frequently a postextrasystolic pause. Hitherto no attempts have been reported to differentiate among varieties of QRS alternans with respect to tachycardia-dependent versus bradycardia-dependent BBB even though alternating configuration of the QRS complex in successive beats was described in experimental animals and in man as long as 65 years ago, and has since been extensively studied and reviewed. An interesting phenomenon related to our study of electrical alternans was described by Cohen et al. under the title "Alternate patterns of premature ventricular excitation during induced atrial bigeminy." Their assumption of shortening of the cycle in a blocked bundle branch by transseptal and retrograde conduction was fully confirmed by microelectrode studies reported by Bandura and Brody. In their studies, the shortening of the bundle branch cycle preceding the cycle terminated by premature beats shortened the refractory period and prevented tachycardia-dependent bundle branch block of alternate premature

![Figure 3](image1)

**Figure 3.** A) Upper tracing: The pause ends with a wide QRS. Alternans disappears as R-R intervals decrease. Lower panel: Alternans is followed by a conducted premature P' with a narrow QRS (see text). The subsequent long interval ends with a wide QRS complex which begins another episode of alternans. LBBB alternans is bradycardia-dependent (patient 3). B) LBBB alternans in lead I of patient 3. In the second beat antegrade block occurs but transseptal conduction and retrograde penetration of the LBB takes place. These events cause alternation of LBB cycle lengths, the shorter cycle (B), ending with successful antegrade conduction in that bundle, whereas the longer cycle (A) ended with unsuccessful attempts at antegrade penetration, indicating bradycardia-dependent antegrade block.

![Figure 4](image2)

**Figure 4.** RBBB is present in alternate beats at a sinus rate of 115/min, in all beats at 94/min and in none at 72/min. "Pseudobradycardia-dependent" BBB. The diagrams, corresponding to those of figure 1B and 3B, are discussed in the text.
beats. In our patients with bradycardia-dependent bundle branch block, transseptal conduction leading to a shortening of the immediate bundle branch cycle prevented development of bradycardia-dependent aberration of intraventricular conduction possibly engendered by phase 4 depolarization.

The classification of conduction disturbances into two types, phase 3 (tachycardia-dependent) and phase 4 (bradycardia-dependent) was made possible by the electrophysiologic findings of Singer et al. Application of these concepts to clinical electrophysiology allows us to postulate these concepts in the area of electrical alternans.

Until recently alternating BBB was regarded as a transient phase in the progression to permanent and complete BBB. It was assumed that a simple 2:1 block existed in the affected bundle branch, and that this block resulted from the fact that the refractory period of the blocked bundle branch was longer than the refractory period of the non-blocked bundle branch. Thus the demonstration of electrical alternans in single myocardial fibers under various experimental conditions, a tendency has arisen to attribute alternation of QRS contour in clinical electrocardiograms, in some instances at least, to en masse alternation of depolarization. In contrast to the well recognized initiation of mechanical alternans by premature systoles, the initiation of electrical alternans by change in heart rate has been pointed out only rarely. Analysis of our cases suggests that a change in rate can determine the beginning or end of alternating BBB, and that the initiation of QRS alternans by a postextrasystolic or other pause can help to explain and distinguish two different types of alternation of BBB.

Tachycardia-dependent Alternans

If the QRS complex terminating a pause is normal and the next one terminating the short cycle is aberrant, the BBB in the latter beat must be tachycardia-dependent. The longer refractory period of a bundle branch after the long preceding cycle (the postextrasystolic pause) could account for such a block. This phenomenon was described by Lewis and Master and explained by Hauswirth et al. in terms of the effect of diastolic interval on repolarization of Purkinje fibers, although in hypopolarized cells, tachycardia-dependent block may be due to a time dependent delay in recovery of excitability after full repolarization. Also, BBB must be of the tachycardia-dependent type if it occurs in all or alternate beats subsequent to an increase in rate.

Patients with tachycardia-dependent BBB alternans may exhibit three types of electrophysiologic behavior depending on three factors: the relationship between the time elapsed between successive activations, the transseptal conduction time, and the duration of the refractory periods of the involved fascicle.

1. Bidirectional Block

When the effective refractory period of the bundle branch (BB) is longer than the sum of its cycle and the transseptal conduction time, but is shorter than the sum of two BB cycles, the result will be QRS alternans due to a 2:1 antegrade block of the involved fascicle without concealed retrograde activation. This is postulated as the mechanism of the alternans in figure 1 and in the first strip of figure 4.

2. Unidirectional Block

If the rate slows during tachycardia-dependent alternans the effective refractory period of the involved BB may remain longer than its cycle length but may become shorter than the sum of the latter and the transseptal conduction time. As a result the bundle branch will remain refractory for antegrade propagation, but may become available for concealed transseptal retrograde activation which will maintain refractoriness of the BB and cause repetitive block. Such unidirectional retrograde BB activation in consecutive beats with slowing of the heart rate is postulated as the mechanism in the second strip of figure 4. A transition from a bidirectional 2:1 block in a BB (without concealed retrograde activation) into a sustained unidirectional antegrade block (due to initiation of concealed transseptal activation) with a critical slowing of the rate, may simulate bradycardia-dependent block, and therefore we propose the name "pseudobradycardia-dependent" block for this phenomenon. Perpetuation of aberrant ventricular conduction resulting from a functional bundle branch block with concealed transseptal retrograde activation of the blocked BB was originally described by Lewis. Later, this concept was used to explain repetitive aberrant conduction masquerading as ventricular tachycardia. This type of concealed intraventricular conduction was demonstrated by Moe et al. in the dog heart and was explained in terms of electrophysiology by Edmunds et al. Transseptal retrograde conduction has been shown to be responsible for perpetuation of both BBB and LBBB in the same patient.

3. Transient Alternans

If a transient and diminishing alternans is seen during tachycardia or in the several beats after a long pause, as in figure 2, an increasing extent of antegrade penetration of the involved conduction pathway during the transition of its refractory periods to a new steady state may be postulated.

Bradycardia-dependent Alternans

Bradycardia-dependent alternating BBB might depend on concealed transseptal retrograde penetration of the "blocked" bundle branch in every aberrant beat (i.e., every alternate beat), so that even though the heart rate and R-R intervals remain constant, the activation cycle of the "blocked" bundle branch alternates between a long and a short duration (fig. 3B, interval A vs interval B). When challenged by the atrial impulse, beats ending a short bundle branch cycle (interval B) conduct normally, but beats ending a long bundle branch cycle (interval A) show BBB which is thus bradycardia-dependent. It must be assumed that the site of bradycardia-dependent BBB in every aberrant beat can be depolarized by retrograde transseptal activation, even after it has become refractory to antegrade stimulation, perhaps because of a difference in the geometry of the proximal and distal ends of the bundle branch. Elizari et al. emphasized the unidirectional nature of phase 4 block and showed that after phase 4 antegrade block had occurred in the right bundle branch, retrograde conduction through the area of block was still possible. However, alternative mechanisms must also be considered. Thus, bundle branch block during bradycardia-dependent alternans may be, in
fact, bidirectional. At some point after the beginning of the aberrant beat, there may be spontaneous complete depolarization of automatic cells in or near the area of block. If this depolarization occurs without antegrade or retrograde propagation, it will be concealed. It is also possible that attempted transesophageal retrograde conduction does not actually penetrate the area of block but that the depolarization of the large number of ventricular cells electronically causes depolarization to threshold and firing of the cells in the area of block. The interval between this spontaneous or electronically induced depolarization and the next beat is relatively short so that the subsequent ventricular complex does not display bradycardia-dependent block.

Therefore, the alternating bundle branch cycle lengths postulated in bradycardia-dependent alternans may be produced by concealed spontaneous (phase 4) or electronically induced diastolic depolarization of the cells of the area of block, or by concealed depolarization with retrograde conduction into an area of unidirectional block. Similar assumptions were made to account for the well known phenomenon of resumption of 1:1 A-V conduction following a ventricular or junctional escape in bradycardia-dependent paroxysmal A-V block. The prevalence of RBB in phase 3 block is well known. In contrast, prevalence of LBBB in phase 4 block in our small series is unexpected and will have to be confirmed by a study of a larger series; however, it is in agreement with observations of Lie et al. Finally, additional types of alternans, such as alternation between RBBB and LBBB, may be rate dependent.

In conclusion, our findings suggest that electrical alternans of the BBB type may be categorized according to its adherence to the usual rules of tachycardia or bradycardia-dependence if the presence or absence of concealed depolarization of the area of block is taken into account. Confirmation of the type of BBB alternans may be obtained elsewhere in the ECG record if isolated aberrant beats are observed to terminate unusually short (tachycardia-dependent BBB) or unusually long (bradycardia-dependent BBB) cycles. We postulate that tachycardia-dependent BBB alternans is related to the refractory period of the blocked bundle branch, and that bradycardia-dependent alternans is related to phase 4 depolarization, although these postulations do not exclude the possibility of other mechanisms of rate-dependent BBB.

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References

5. Lewis T: Note upon alternation of the heart. Quart J Med 4: 141, 1910
20. Lewis T, Master AM: Supernormal recovery phase, illustrated by two clinical cases of heart block. Heart 11: 371, 1924
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H C Cohen, I D'Cruz, E R Arbel, R Langendorf and A Pick

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