Paradoxic Phenomenon of Premature Beats with Narrow QRS in the Presence of Bundle-Branch Block

By Rashid A. Massumi, M.D., General Hiliard, M.D., Anthony DeMaria, M.D., Ramon Fabregas, M.D., Alan E. Lindsay, M.D., Ezra Amsterdam, M.D., and Dean T. Mason, M.D.

SUMMARY
In contrast to the functional bundle-branch block (BBB) which is frequently rate-related and disappears when heart rate slows, we have encountered seven cases of a paradoxic disappearance of an existing, ischemia-induced BBB in premature beats. Supernormal conduction within the blocked bundle branch was not present in any of the cases. His bundle electrography in three of the cases demonstrated His spikes before each of the narrow-QRS premature beats. The His-Q intervals of the premature beats were considerably shorter than those of the control sinus beats. This finding clearly indicated an ectopic origin midway between the ventricular myocardium and the His bundle, i.e., within the ischemic fascicles of the left bundle, or the ischemic right bundle. From their points of origin such fascicular impulses propagate antegradely toward the respective area of the ventricular myocardium, and retrogradely toward the main His bundle, and at the same time down the remaining two fascicles. The antegrade conduction time in the short pathway down the ischemic fascicle is equaled by the faster conduction through the longer pathways of the remaining, uninjured fascicles, thereby accounting for the almost simultaneous activation of the myocardium of the two ventricles and the resultant narrow-QRS complexes.

Additional Indexing Words: Supernormality Paradoxic bundle-branch block Fascicular beats

Functional bundle-branch block

In patients with bundle-branch block (BBB), the asynchronous activation of the two ventricles does not necessarily reflect complete cessation of conduction, but may result merely from the slowing of transit through the involved bundle. Termed functional BBB, this type of conduction abnormality is apt to disappear when cardiac cycles are lengthened and the involved bundle is given sufficient time for full recovery of conductivity. This common rate-related BBB contrasts with the less common and paradoxic type of "normalization" of intraventricular conduction in premature beats indicating a transient disappearance of the asynchrony of activation of the two ventricles. This latter condition is produced by a variety of mechanisms of which two are most acknowledged. In the first, a late-diastolic premature ventricular beat arises from the blocked ventricle, activating it at about the same time the unblocked ventricle is undergoing depolarization following the arrival of the antegrade-conducted impulse. Such "normalized" fusion beats are preceded by the normal sinus P wave, and the P-R interval is only slightly shorter than in the control sinus beats. They are premature by only a few hundredths of a second.

The second mechanism for narrow-QRS premature beats in the presence of BBB is supernormal conduction of premature atrial or junctional impulses through the blocked bundle branch. This usually occurs when the impulse reaches the blocked bundle during its supernormal phase corresponding roughly with the end of the T wave.

From the Section of Cardiovascular Medicine, University of California School of Medicine, University of California at Davis, and the Intermountain Clinic, Salt Lake City, Utah.
Supported in part by research grant 71-1072 from the American Heart Association and by research fellowship award HL-52408, training grant HL-5901, and research program project grant HL-14780 from the National Heart and Lung Institute, National Institutes of Health, Bethesda, Maryland.
Address for reprints: Rashid A. Massumi, M.D., University of California School of Medicine, Section of Cardiovascular Medicine, Davis, California 95616.
Received October 28, 1972; revision accepted for publication November 11, 1972.
of the preceding beat. It should be emphasized, however, that despite its wide recognition, true supernormality is a rare phenomenon and that it can be simulated by a number of unrelated mechanisms.\(^5\)\(^6\)

The purpose of this communication is to report four cases of a group of seven in which a rarely recognized mechanism for the apparent normalization or narrowing of the QRS deflection in premature beats in the presence of BBB has been documented. There were five men and two women, with ages ranging between 42 and 70 years, and all had ischemic heart disease. In their control tracings, four had left BBB (LBBB) and three had right BBB (RBBB). Acute myocardial infarction was present in two patients and acute coronary insufficiency in the remaining five. It will be demonstrated that in such premature beats, impulses arise from the bundle or fascicle possessing impaired conductivity, and propagate simultaneously toward the related territory of the ventricular myocardium and, retrogradely, toward the main His bundle, and at the same time down the remaining two fascicles. The slow antegrade conduction through the involved bundle is equaled by the longer distance traveled by the impulse in its advance toward the contralateral ventricle, thus accounting for the almost synchronous depolarization of the two ventricles and the narrow QRS deflections.

Report of Cases

Case 1

This 56-year-old man with a history of arterial hypertension and angina pectoris was admitted to the hospital on July 29, 1972 because of a protracted attack of angina pectoris and strong suspicion of an acute subendocardial myocardial infarction. However, serial serum enzymes did not show significant elevations. An admission electrocardiogram showed complete LBBB

---

**Figure 1**

Case 1. Two strips of \(V_1\) taken on July 30, 1972, and two strips taken on July 31, 1972 showing LBBB in all the beats in \(A\) and \(B\) except the premature ectopic ones. In strips \(C\) and \(D\), LBBB is absent not only in premature beats but also when cardiac cycles become longer than 0.76 sec, either after premature beats or as a result of carotid sinus stimulation (CSS).
with QRS complexes of 125-msec duration and left-axis deviation of $-45^\circ$. This combination was attributed to ischemia of both fascicles, greater in the anterior-superior fascicle than the posterior-inferior. Subsequent observations demonstrated the LBBB to be intermittent and rate-related, present for 8 days, and generally when the heart rate exceeded 86 beats/min (fig. 1). In addition to the conducted sinus beats with LBBB and those with normal QRS complexes seen with slowing of the heart, premature beats with normal QRS of 95-msec duration were noted (beats marked in fig. 1). The early premature beats were clearly of normal configuration, but those occurring at the end of diastole fused with the conducted beats, giving rise to QRS complexes of intermediate or fusion morphology (beats marked $F_2$). The normally conducted beats and the narrow-QRS premature beats showed a 2-mm depression of the S-T segment and T-wave inversion in leads $V_2-V_6$, suggesting severe anterior wall ischemia or subendocardial infarction.

A His-bundle electrogram obtained on the second hospital day is shown in figure 2. Beats 1 and 2 are conducted sinus beats with LBBB. The third beat is a narrow-QRS ectopic premature beat. The fourth beat is also an antegrade-conducted beat but with normal QRS due to the pause following the preceding extrasystole. The His-Q interval remains at the normal value of 45 msec in beats 1, 2, and 4, but is significantly shorter, only 25 msec, in the ectopic beat 3. This finding localizes its origin at a point within the fascicles of the left bundle-branch system from which the impulse propagates both antegradely toward the myocardium and simultaneously retrogradely toward the main His bundle, reaching both destinations within a period of time which is necessarily shorter than the His-Q interval of the conducted beats. As was demonstrated in a previous study, impulses arising from the fascicles of the left bundle depolarize the right ventricle last and, therefore, display complete or incomplete RBBB and also axis deviation—left if they originate from the

![Figure 2](https://circ.ahajournals.org/)

**Figure 2**

*Case 1. His bundle recording with simultaneous leads I, II, III, and $V_1$. Frontal-plane mean QRS vectors are depicted both for sinus beats (left) and ectopic beats with narrow QRS (right). His spikes are marked by arrows. Below this tracing, simplified anatomic diagrams depict the main His bundle and the three fascicles (AS, PI, and RB). Dotted areas represent ischemia and density of dots the magnitude of ischemia. Open circle is the presumed origin of the ectopic impulse and arrows through the anatomic diagram depict directions in which the ectopic impulse propagates from its origin to all the three fascicles and also the main His bundle. The zigzag portions of conduction lines are purported to show slowing of conduction (same conventions apply to figures 3, 5, and 6). Mean frontal QRS axis for control sinus (C) and fascicular (F) beats are indicated at bottom.*

*Circulation, Volume XLVII, March 1973*
MASSUMI ET AL.

posterior-inferior fascicle, and right if the origin is in the anterior-superior fascicle. The reason for the absence of RBBB in this case is the existence of slow conduction through the fascicles of the left bundle, serving to equalize the conduction times in the anatomically shorter but slower path to the left ventricular myocardium and the longer but faster path to right ventricular myocardium. In a similar manner, the disappearance of the left-axis deviation present in the control beat can be explained on the basis of an equalization of conduction times taken by the ectopic fascicular impulse to travel the short but slow path to the territory of the anterior-superior fascicle and the longer but faster path to the myocardium of the inferior ventricular wall via the less involved posterior-inferior fascicle (see diagrams beneath His bundle tracing in fig. 2). That this explanation is probably correct is supported by subsequent observations on the ninth hospital day when active myocardial ischemia and the ischemic pain were no longer present (fig. 3). Ectopic beats arising from the same focus, recorded at this time, did exhibit a 30° rightward rotation of the spatial QRS axis and an incomplete RBBB manifested by decreased amplitude of the QRS axis, indicating that an anterior-superior fascicular origin is consistent with the electrocardiographic finding of ischemic changes in the anterolateral left ventricular wall.7

Comment
In this case of rate-related LBBB secondary to transient, severe coronary insufficiency ectopic beats with narrow QRS complexes were observed. His bundle electrophysiology showed these impulses to arise from the anterior-superior fascicle of the left bundle corresponding anatomically to the area of ischemia. It is postulated

Figure 3
Case 1. Leads I, II, V₃, and V₆ taken 8 days apart. Each lead contains one premature fascicular beat marked by X. The frontal-plane QRS axis of +55° and the complete absence of RBBB in fascicular beats on July 30 contrast with the axis of +85° and the incomplete RBBB on August 7. The explanation, suggested by the diagrams, is the presence of greater ischemic injury on July 30, accounting for slower conduction from the site of impulse formation open in both directions in the anterior-superior fascicle, and somewhat lesser slowing in the posterior-inferior fascicle. The net result is an equalization of conduction times from the ectopic focus to the three main regions of the myocardium corresponding to the three fascicles. In the tracing of August 7, the ischemic injury has regressed considerably. Impulses arising from the anterior-superior fascicle now propagate with almost normal velocity, reach the myocardium of the anterior left ventricle wall next and the right ventricle last, thus accounting for the axis deviation and the incomplete RBBB.
that the "normalization" of the ectopic QRS complexes reflected synchronized activation of the right and left ventricles following discharge from the fascicular focus.

Case 2

A 70-year-old woman with history of ischemic heart disease was admitted because of a severe chest pain and intense dyspnea, pulmonary congestion, and a mild shock. Admission electrocardiograms taken during the first 2 days showed persistent, complete LBBB which made further interpretation difficult. Figure 4 shows selected leads from the electrocardiogram taken shortly after admission, and in each lead one or more beats marked by x are premature and ectopic, not preceded by P waves. The conducted beats display complete LBBB with QRS duration of 130 msec. The ectopic beats on the other hand, have a QRS complex of 105 msec. The ectopic beats appear with varying coupling intervals in relation to the preceding sinus beats and have features of a parasystolic rhythm at the rate of approximately 60 beats/min. Study of the morphologic characteristics of the ectopic beats reveals a shift of the spatial QRS axis toward the right, from +30° to +90°, and also features of incomplete RBBB best appreciated in lead V1. These relatively narrow beats showed Q waves in midprecordial and left precordial leads, together with ST-T changes quite consistent with a recent anteroseptal infarction.

In the His bundle recording shown in figure 5 the first and the last beats are normally conducted sinus beats with LBBB. His deflections are clearly seen, and His-Q intervals are 50 msec. Beat 2 is a premature atrial beat with negative P waves in leads II and III. This beat also is conducted with LBBB and a His-Q interval of 50 msec. Beat 3 is an example of the narrow-QRS ectopic beats shown in the previous figure and has features of incomplete RBBB and right-axis deviation. The His-Q interval of this beat is only 10 msec. The explanation for the morphologic features of the ectopic beats is shown in the diagrams below the His bundle tracing which may be interpreted in a manner similar to the diagram of figure 2. It should be noted, however, that in contrast to case 1, in which the QRS complexes of the ectopic beats were entirely normal, in the present case the ectopic beats have all the features expected of fascicular beats originating from the anterior-superior fascicle of the left bundle, i.e., RBBB and right-axis deviation. This suggests that activation of the myocardium of the left and right ventricles during the ectopic
fascicular beat maintained the abnormal sequence known to exist in such beats. For this reason, the ischemic damage to the left bundle is assumed to be in a restricted area above the level at which the fascicular impulse deflects and descends down the right bundle. Alternatively, a unidirectional block (antegrade block, preserved retrograde conduction) involving the main portion of the left bundle branch, with a fascicular pacemaker situated distal to the region of block, can explain the phenomenon observed in this case.

**Comment**

In this case of LBBB secondary to an acute anteroseptal wall myocardial infarction, ectopic beats arising from the ischemic anterior-superior fascicle of the left bundle displayed incomplete RBBB and relatively narrow QRS complexes. The area of major ischemia responsible for the LBBB was assumed to be high, above the origin of the three fascicles. Therefore, the ectopic impulses originating from the anterior-superior fascicle of the left bundle could reach the myocardium of both ventricles without passing through the ischemic area with decreased conduction velocity. Accordingly, the ectopic beats possessed the characteristics of regular, uncomplicated fascicular extrasystoles. The theoretic localization of ischemia in the upper regions of the left bundle is consistent with the pure pattern of LBBB unassociated with any axis deviation in the conducted beats. This feature contrasts with the left-axis deviation seen in case 1 in which ischemia was believed to involve primarily the fascicles of the left bundle.

**Case 3**

In this 56-year-old man with acute inferior wall myocardial infarction, RBBB developed on the second day of infarction. This is shown in the left panel of figure 6 where deep Q waves, S-T-segment elevation and T-wave inversion can be seen in leads II and III, while features of RBBB are best seen in lead V₁. The

---

**Figure 5**

*Case 2. His bundle recording together with leads I, II, III, and V₁.* The accompanying diagrams serve to show the short His-Q interval of 10 msec for the ectopic beat numbered 3 as compared with the value of 50 msec for the control beats. Note the origin of the ectopic beat in the anterior-superior fascicle (open circle in AS) and the manner in which the ectopic impulse is believed to bypass the area of ischemic injury in the left bundle, thereby propagating to the other fascicles without local delay. C and f = frontal QRS vectors in control and fascicular beats, respectively.
Case 3. Three conducted sinus beats with long P-R and RBBB (QRS 140 msec) in A and three beats from an episode of complete heart block with an escape rhythm possessing features of LBBB and QRS duration of 110 msec in panel B. QRS complexes during heart block are preceded by His spikes. However, the His-Q intervals are only 35 msec as compared with 50 msec in the conducted beats. Diagrams beneath A and B, pertaining to the two types of beats indicate the propagation of sinus and ectopic escape impulses. In A, descending sinus impulses are slowed in the A-V node because of the acute inferior wall infarction (dotted area proximal to His bundle). This explains the long P-R interval. Furthermore, the ischemic injury has slowed conduction through the proximal portion of the right bundle branch (blood supply from the A-V node artery), thus producing RBBB in the conducted beats. Implicit in the normal His-Q interval of 50 msec is the persistence of normal conduction through the left bundle. In B, the beats are clearly ectopic, unrelated to the P waves. They are believed to arise from the injured right bundle branch (open circle inside dotted area), and travel antegrade toward the right ventricular myocardium and simultaneously retrograde toward the main His bundle and down the unimpeled left bundle-branch fascicles. The slow conduction down the right bundle is matched by the more rapid conduction to the myocardium of the right ventricle, using the much longer path. The activations of the two ventricles are thus “synchronized” despite the existence of RBBB, and the resultant QRS is narrower than those of conducted beats.

QRS complexes are 140 msec in duration and His-Q intervals 50 msec. In the afternoon of the second hospital day, this patient developed short periods of third-degree heart block with an atrial rate of 120 beats/min and an escape rhythm of 72 beats/min in which the QRS complexes had features of incomplete LBBB and were 110 msec in duration as compared with 140 msec in the conducted beats. The His-Q intervals of the escape beats were 35 msec, considerably shorter than the 50 msec observed during sinus rhythm. The escape beats from the subsidiary pacemaker are, therefore, believed to have arisen from the right bundle (see diagrams below His tracing). In this patient also, the narrower QRS complex in the ectopic beats in comparison with the conducted complexes is explained on the basis of a slow conduction through the shorter path, extending from the ectopic focus in the right bundle, to the right ventricular myocardium and faster conduction through the longer path extending between the ectopic focus and the left ventricular myocardium.
In this case, the narrow QRS complexes actually represent an escape mechanism rather than prematurity. Nevertheless, the case is presented because it sheds light on the mechanism responsible for the narrow-QRS ectopic beats in the presence of an already existing BBB.

Case 4

In this patient with angina pectoris and complete LBBB, premature beats with narrow QRS deflections were noted to occur in a parasystolic mode with interectopic intervals on 1.76 sec. In figure 7, in which leads I, II, and V1 are depicted, the ectopic beats are marked by X. The varying coupling intervals are quite evident and account for the appearance of fusion beats (marked F) in leads II and V1. The spatial QRS axis is +30° for conducted beats and indeterminate in the ectopic beats. Even though His bundle tracings are not available, the obvious similarity between this case and case 1 strongly indicates that the ectopic beats arose from the left bundle or its fascicles proximal to the area of major conduction delay, with an explanation similar to that given in the preceding three cases. Premature ventricular beats with RBBB configuration fusing with the conducted beats would be a possibility in the fusion complexes. However, the earlier members of the parasystolic rhythm are also narrow and, therefore, exclude premature ventricular beats with fusion as the explanation.

Discussion

The appearance of narrow-QRS ectopic beats in the face of an existing BBB is an interesting phenomenon which can be explained by any one of several mechanisms. Perhaps the most common is the appearance of a late diastolic ventricular ectopic beat of opposite configuration fusing with the conducted beat. These late ventricular beats are frequently members of a parasystolic ventricular rhythm, and less frequently isolated ectopic beats. In the former situation, correct diagnosis is quite simple because the cardinal features of parasystole are present, while single ventricular beats are more difficult to recognize. In either case, the narrow-QRS complexes are late in appearance and, therefore, are not confused with the "normalized" premature beats.

One of the most readily considered mechanisms for narrow-QRS complexes in premature beats is the phenomenon of supernormality. A premature atrial or junctional beat may arrive at the division of the bundle-branch system at the time corresponding with the end of the T wave of the preceding conducted beat, thus coinciding with the supernormal phase of the blocked bundle, and be

Figure 7

Case 4. Narrow-QRS premature beats in the presence of complete LBBB.
conducted normally to both ventricles. A preexisting BBB in such cases will temporarily disappear in this type of premature beat. Certainly diagnosis of supernormality in these extrasystoles depends on the demonstration of the impulse being conducted from a focus above the point of division of the bundle-branch system and, for this reason, the His-Q interval should be similar to that of the conducted beats. The demonstration of a short His-Q interval in three cases of the present study clearly indicates that the ectopic impulses did not descend through the main His bundle, and that they originated at some point midway between the ventricular myocardium and the main His bundle. Since cases 4, 5, 6, and 7 of the present study did not have His-bundle recordings they should be considered as probable examples of fascicular beats in the presence of BBB. Supernormality could not be invoked in these four cases because ectopic beats appeared late in the cycle, beyond the region of supernormality.

Other important differential diagnoses include incomplete bilateral BBB and echo, or capture beats in ventricular tachycardia associated with retrograde VA conduction. In the former situation, equalization of conduction times in both bundle branches results in normalization of the QRS (fig. 8). In ventricular tachycardia, on the other hand, the presence of a Wenckebach pattern of retrograde VA conduction is usually responsible for echo capture beats with normalization or near normalization of the QRS (fig. 9).

Finally, one must consider the bradycardia-dependent type of BBB to be responsible for the presence of BBB at slower, normal heart rates and its disappearance when cardiac cycles are shortened. In this phenomenon, it is presumed that spontaneous phase 4 depolarization in one bundle proceeds more steeply than in the other and causes a greater decline in its transmembrane potential, thus rendering it less conductive. This situation is very rare. Moreover, it cannot explain the occurrence of fusion QRS complexes which indicates collision of two independent wavefronts invading the ventricles from two different directions. The demonstration of abnormally short His-Q intervals, furthermore, documents the ectopic nature of the narrow QRS complexes.

It should be stressed, at this point, that the concept of fascicular rhythm is not entirely new. Actually, origin of ectopic beats from the interventricular septum was invoked by Wilson and Hermann in 1920 as an explanation for the unexpectedly narrow QRS complexes in a case of BBB. One year later, Hewlett offered the same explanation in a case of narrow-QRS extrasystoles in a

![Figure 8](image)

*Figure 8*

Two strips of lead V1 in a patient with bilateral BBB. (Top) A 2:1 conduction with RBBB. (Bottom) Three beats marked X have narrow, normal QRS deflections and are preceded by longer P-R intervals. Conduction along the right and left bundles (R and L) from the beginning of the P waves to the beginning of the subsequent QRS complexes are depicted by arrows when conducted and by double bars if blocked. A glance at the function of each bundle will demonstrate that conduction along the right bundle is completely blocked in the top strip and is 3:1 in the bottom strip, while along the left bundle it is 2:1 in the top strip and 3:2 Wenckebach in the bottom strip. When conduction times in both bundles equalize, the resultant QRS will be normal and narrow.
Leads I, II, and III during ventricular tachycardia (left) and after restoration of normal sinus rhythm (right) taken in a 47-year-old man with recent anterior wall myocardial infarction. Narrow QRS complexes are marked by asterisks. During ventricular tachycardia, retrograde conduction with a Wenckebach pattern is present, and when retrograde conduction becomes very long, as after beats 3 and 9 of lead III, echo or reentry into the ventricle takes place. The resulting QRS deflections are narrower than the ectopic ventricular beats but are not entirely normal because fusion between the conducted QRS and the ectopic ventricular QRS is inevitable.

The findings set forth in this manuscript present yet another application of the concept of fascicular impulse formation which has been found in our laboratory to be extremely useful in explaining many complex and elusive abnormalities in clinical electrophysiology. It has proved useful to recognize that the intraventricular fascicles may in fact produce impulses and function as pacemakers. Their discharges have been found to follow a variety of patterns including single premature beats, escape beats, parasystolic discharges, self-sustained accelerated ventricular rhythms, and occasionally tachycardias. Their morphologic characteristics when the basic QRS complexes are normal have been described previously. The present manuscript serves to indicate that they may in fact give rise to narrow or even normal-appearing QRS complexes in the face of BBB which is merely due to delay of conduction in that bundle. Of course, a complete block in one bundle, capable of preventing propagation of a fascicular impulse, will be inconsistent with the phenomenon described here.

References


2. Lepeschkin E, Rosenbaum MB: Coupling intervals of ventricular extrasystoles in relation to the heart rate: The U wave and the supernormal phase of excitability. Circulation 15: 82, 1957
15. Hewlett AW: A case showing bundle branch block with extrasystoles originating in the ventricular septum. Heart 9: 1, 1921
Paradoxic Phenomenon of Premature Beats with Narrow QRS in the Presence of Bundle-Branch Block
RASHID A. MASSUMI, GENERAL HILLIARD, ANTHONY DEMARIA, RAMON FABREGAS, ALAN E. LINDSAY, EZRA AMSTERDAM and DEAN T. MASON

Circulation. 1973;47:543-553
doi: 10.1161/01.CIR.47.3.543

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1973 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/47/3/543

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/