Experimental Production of Aberrant Ventricular Conduction in Man

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SUMMARY
Aberrant ventricular conduction (AbC) was produced in 17 of 18 subjects, including six normal persons, by the introduction of electric stimuli via a transvenous catheter situated in the right atrium.

Analysis of electrophysiological determinants indicated that AbC was favored by a long cycle preceding the short cycle which terminated in the aberrant complex, a short R to stimulus coupling interval, and fast atrioventricular conduction time.

Electrocardiographic analysis of AbC revealed that all patients had right bundle-branch block (RBBB). In addition, incomplete right bundle-branch block was present in eight patients and was transformed to RBBB in all cases by shortening the coupling interval. Ten patients had slight but distinct variations of their RBBB pattern. There were two instances each of left bundle-branch block and intraventricular conduction disturbance. Hence, any patient may have the potential to produce a variety of AbC patterns. AbC was produced in six normal subjects and must be considered a physiological event which does not require covert pathology of the specialized conduction system.

Additional Indexing Words:
Coupled atrial pacing    Paired atrial pacing
Right bundle-branch block Intraventricular block

Aberrant ventricular conduction (AbC) is an alteration in the electrocardiographic complex which results from the failure of a supraventricular impulse to complete normal ventricular excitation. Impulse propagation is altered because of a refractory state in part of the usual conduction pathway. The clinical recognition of AbC is of critical importance in the management of patients displaying this phenomenon, for it may be mistaken for ventricular premature beats or ventricular tachycardia when occurring in successive beats.

AbC has traditionally been studied from electrocardiograms of spontaneous arrhythmias. The probability that AbC will occur is increased proportionally to the brevity of the cycle following a beat and to the length of the cycle preceding the same beat. The condition of a long cycle preceding a short cycle which terminates in an aberrant complex was produced in this study by the introduction of a coupled atrial premature beat at an appropriate position in the cardiac cycle. The method permitted a controlled analysis of the electrophysiological determinants required to produce AbC, as well as the resultant electrocardiographic patterns. A description of the method and of the analysis constitutes the substance of this report.

Methods
Eighteen subjects in normal sinus rhythm were studied. They composed four groups which included six normal volunteers, six hypertensive patients, all untreated with the exception of G.A. who was receiving reserpine, four patients with
Figure 1

Transformation of incomplete right bundle-branch block to complete right bundle-branch block. Progressively shorter coupling intervals of isolated premature stimuli (R-S msec) transform incomplete right bundle-branch block (B) to complete right bundle-branch block (C). Note that the lengths of the preceding long cycles are constant.
arteriosclerotic heart disease, and two patients with pulmonary disease.

Each study was performed in the cardiopulmonary laboratory with the patient supine and in the postabsorptive state. Utilizing percutaneous sterile technique and local procaine anesthesia, a bipolar or tripolar electrode-pacing catheter was introduced via an antecubital vein under fluoroscopic and electrocardiographic control and was positioned against the wall of the right atrium. A battery-powered pacemaker (Medtronic “R” wave coupled pulse generator) delivered an impulse of 2-msec duration at an adjusted milliamperage which would result in reliable atrial “capture.” The condition of a long cycle preceding a short cycle which terminated in an aberrant beat could be produced by several modalities of atrial pacing. Method 1 involved the introduction of an isolated premature atrial stimulus coupled to the P wave of the preceding beat (fig. 1B and C). The isolated stimulus was introduced after the sixth beat of a series of normal beats. The stimulus was delivered at a variety of controlled coupling intervals. Method 2 utilized the coupling of atrial premature stimuli to the P wave of normally conducted beats in a manner that produced a rhythm in which every other beat was premature (fig. 2). Method 3 utilized paired pacing of the atrium and produced a rhythm similar to that of method 2 with the important exception that all beats were resultant from catheter stimuli (fig. 3).

P to stimuli coupling intervals were maintained at or above 300 msec when premature stimuli were introduced by any of the three methods. No further decrease of the coupling interval was attempted because of the risk of producing atrial fibrillation by stimulating the atrium during its vulnerable period. Simultaneous standard electrocardiographic leads I, II, V1, and V6 were recorded. In addition an intra-atrial electrogram was recorded when a tripolar pacing catheter was utilized. Recordings were made by a multichannel oscillographic photographic recorder (Electronics for Medicine) at paper speeds of 25 and 50 mm/sec.

In the interest of uniformity, the stimulus to R interval (S-R) was assumed to be the same as the P-R interval. This assumption was based on the fact that atrial depolarization immediately follows the stimulus artifact in the standard electrocardiographic leads and in those intra-atrial electrograms which were recorded.

**Results**

The results are summarized in table 1.

![Figure 2](image)

*Figure 2*

Production of aberrant ventricular conduction by alternate premature beats. An illustration of the importance of preceding cycle length in producing aberration. The first stimulus results in a premature beat which is predominantly conducted through the normal pathway and has a prolonged diastolic period before the next sinus beat. The induced premature beat following this prolonged cycle results in aberrant ventricular conduction (Ab). The preceding cycle length increases from 960 to 1,060 msec. Note that coupling intervals (R-S) and conduction time (S-R) are constant.
Table 1

Results of Study

<table>
<thead>
<tr>
<th>Pt.</th>
<th>Age (yr)</th>
<th>ECG</th>
<th>Required method 2</th>
<th>% Stimulus prematurity of preced. cycle</th>
<th>Identical RBBB 0.04 sec. init. force</th>
<th>Aberrant pattern</th>
<th>Normalization with longer A-V conduction</th>
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Abbreviations: IRBBB = incomplete right bundle-branch block; RBBB = right bundle-branch block; LBBB = left bundle-branch block; IVCD = intraventricular conduction disturbance; A-V = atrioventricular; N = normal; LAD = left axis deviation; LVH = left ventricular hypertrophy. Inf. MI = inferior myocardial infarction.
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Electrocardiographic Patterns

Aberrant ventricular conduction manifesting as a clear alteration in QRS configuration was produced in all subjects with one exception. The electrocardiographic patterns included complete right bundle-branch block (RBBB), incomplete right bundle-branch block (IRBBB), left bundle-branch block (LBBB), and intraventricular conduction disturbance (IVCD).

RBBB was present in 100% of patients demonstrating AbC, of which 10 had slight variations of this pattern. In the RBBB pattern, the initial 0.04-sec scalar vector was found to be identical to the comparable force of the normally conducted beat in 47% of the cases. Eight cases of IRBBB were noted (fig. 1).

Two cases of LBBB were recorded (fig. 3). Intraventricular conduction disturbances were found in two patients and were designated

Figure 3
Left bundle-branch block aberration. (A) Pacing method 3. Stimulus 1 (S₁) is the basic driving stimulus, and stimulus 2 (S₂) is the premature stimulus. Left bundle-branch block aberration follows the second labelled S₂. Note the varieties of right bundle-branch block. (B) Left bundle-branch block follows the second labelled stimulus (S).
as such because of QRS intervals of at least 0.12 sec, and patterns which did not satisfy a clear-cut definition (fig. 4).

**Electrophysiological Determinants**

AbC first became evident when the R to atrial premature stimulus interval (R-S) was less than 44% (range, 43 to 21%) of the preceding R-R interval. Once present, the aberrant pattern could continue to be elicited with decreasing coupling intervals until either the premature atrial impulse was blocked during atrioventricular propagation, or the coupling interval had been decreased to the 300-msec limit of safety. Rare exceptions were noted, as described below.

In four patients, isolated atrial stimulation (method 1) either produced a normally conducted complex or a blocked atrioventricular

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**Figure 4**

Effect of decreasing coupling intervals. As the R to stimulus coupling intercal decreases, the S-R' interval generally increases and the aberrant pathway changes. Preceding long cycle R-R intervals are similar with a maximum variation of 120 msec. (A) Normal intraventricular conduction of premature stimulus. (B) Incomplete right bundle-branch block. (C) Complete right bundle-branch block. (D) Intraventricular conduction disturbance. (E) Intraventricular conduction disturbance with deeper S wave in lead II. (F) Atrioventricular block.
impulse. AbC was subsequently produced in each by method 2, in which an atrial premature stimulus was coupled to consecutive normal sinus beats (fig. 2).

In each patient with AbC of an IRBBB pattern, a shorter coupling interval produced RBBB (fig. 1).

In two patients demonstrating more than one type of conduction pattern (excluding IRBBB), it was possible to produce a consistent type of AbC within a narrow range of coupling intervals. Atrioventricular (A-V) conduction time generally increased with decreasing coupling intervals (figs. 1 and 4). Occasional unexpected pattern deviations were found in two patients with otherwise predictable aberration configurations within a limited range of coupling intervals. These unexpected patterns (minimal aberration and normal intraventricular conduction time) were accompanied by longer than usual atrioventricular (S-R) conduction times (figs. 5 and 6). A-V conduction was also prolonged in the sole patient in whom induced AbC failed (fig. 7).

**Discussion**

Aberrant ventricular conduction was produced easily and without complication in man. The method, as outlined, did not result in any untoward effects. Attentive handling of the distal electrode terminals and utilization of an isolated battery-driven power source minimized theoretical complications from improper electrical grounding. The vulnerable period of the atrium was avoided by introducing electrical stimuli at an interval of at least 300 msec after the onset of atrial depolarization. With this precaution atrial fibrillation or flutter did not occur.5

Experimentally produced AbC was considered to render a valid account of the aberrant pathway(s) of each subject, for, in two patients, spontaneously occurring AbC was similar to that artificially induced (fig. 8).

**Electrophysiological Determinants**

This study indicates that several factors are of importance in the production of AbC. They are (1) the length of the long cycle which precedes the short cycle terminating in
Normalization of aberration with increasing atrioventricular conduction. Series A and B are of the same patient and were separated in an attempt to minimize variation in the preceding cycle length (R-R interval). Progressive shortening of coupling intervals shows normal conduction, two examples of right bundle-branch block aberration, and an unexpected return to normal intraventricular conduction. Atrioventricular conduction time of the normalized beat reveals an increase of 223 msec in series A, and an increase of 130 msec in series B when compared to the previous conduction times resulting in aberration. IAE = intra-atrial electrogram.

Each favors production of AbC because each enhances the possibility of the propagating impulse arriving on refractory or partially refractory conducting tissue. The impulse will either fail to progress in the former case and become blocked, or continue to progress via nonrefractory parts of the usual route and result in AbC.
Figure 7

Prolonged atrioventricular conduction time in the patient in whom aberrant ventricular conduction did not occur. With approximately the same preceding cycle length and coupling intervals, the patient in A has approximately twice the atrioventricular conduction time (S-R) as the patient in B or C. Patient C did not have any aberration at the coupling interval shown here, but did have aberrations with shorter coupling intervals as shown in figure 3A.

It is well established in man that the refractory period of the A-V node shortens with naturally occurring abbreviations of preceding cycle length. The refractory period of the specialized conduction system (SCS), which includes His bundle, right and left bundle branches, and peripheral Purkinje system, is also thought to shorten with abbreviation of preceding cycle length in man. This has been elegantly demonstrated by direct methods in the SCS of dogs but must be assumed in man through less direct evidence. Conversely, a prolonged refractory period of the A-V conducting system results from an increasing preceding cycle length. It follows that AbC should be favored by an increase in refractory period of the SCS which results from a slowing of heart rate. All patients in this study had normal sinus rhythm with minor fluctuations in cycle length. Therefore, it was not
Experimentally induced aberration versus spontaneous aberration. Experimentally induced aberration shown in panel A (Ab) is similar to that which occurred following a spontaneous atrial premature contraction (APC) which is shown in panel B. Both records are of the same patient.

possible to put strictly to the test the general observation that "Of 2 premature systoles with identical coupling but with varying duration of the preceding cycle, the one which follows the longer cycle is more prone to exhibit aberrant conduction. . . ." Yet, it was possible partially to test this thesis in the four patients who did not respond with AbC after isolated stimuli, but who had AbC induced by method 2. In this method, the first premature beat was conducted through the normal pathway and resulted in a slightly prolonged diastolic period before the next sinus beat. The induced premature beat following this slightly prolonged cycle resulted in AbC. In the example shown in figure 2, the prolongation of preceding cycle length was the only variable among those concluded in this study to be of importance for the production of AbC. Another factor must be mentioned, however, which prevents an absolute incrimination of cycle length as the sole determinant responsible for the induction of AbC in the four patients cited. Alteration in cycle length results in altered cellular action potential. It may take several beats at a new rate before a constant action potential is achieved for that rate. Consequently, AbC produced by coupling a premature beat to the first cycle of an abruptly slowed heart rate may be partially the result of an excessively prolonged action potential and may not be reproducible after subsequent beats at the same cycle length and coupling interval. Because of this consideration, an isolated atrial stimulus was coupled to at least the sixth of a series of normal sinus beats in method 1.

Short coupling interval, the second factor, was found to enhance the possibility of producing AbC. The atrioventricular node (A-V node) or atrial A-V node junctional tissue is generally the area where most instances of A-V block occur. Yet, animal experimentation has shown that the refractory period...
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increases progressively and distally in the SCS.11

A mandatory requirement for the production of AbC is that impulse propagation proceeds through the A-V node to meet a partially refractory specialized conduction system (SCS). Therefore, the A-V node must be in a nonrefractory state while the SCS is in a partially refractory state. If the premature stimulus is too early, the impulse will find the junctional A-V nodal tissue refractory and will progress no further. If the premature stimulus is too late, it will progress through a completely repolarized pathway and induce a normal-appearing complex. The point is emphasized by the difficulties encountered in producing experimental aberration in the in situ canine heart. The introduction of an atrial premature stimulus usually results in normal conduction or blocking of the impulse. The challenge of delivering a stimulus to the SCS during its relative refractory period is met by directly stimulating the His bundle via an implanted electrode. AbC is readily produced in this manner.8

The more premature a stimulus, the more likely the chance of producing AbC, up to the point of block.3 Because of clinical electrocardiographic emphasis on preceding cycle length as it relates to AbC, it was decided to express stimulus prematurity as a function of this interval. The influence of cycle length on refractory period has been discussed previously in this paper. When stimulus prematurity was expressed as a percentage of preceding cycle length, the broad limits required to produce AbC became apparent. The coupling conditions for producing AbC were met when the R-S interval ranged from less than 44% of the preceding cycle length to greater than the point of producing A-V block.

The third factor of importance in producing AbC was fast A-V conduction time. Rapid conduction time of premature stimuli enhanced aberration, and delayed conduction inhibited AbC by permitting a longer time for distal repolarization. Several examples of inhibition were noted in this study. Delays may result in a less marked degree of AbC (fig. 5) or, if of great enough magnitude, may result in normal conduction in a setting which is usually conducive to AbC (fig. 6). In the sole patient who did not manifest AbC, excessive A-V conduction time was apparent when the records of this patient were compared with those of others in the study who had the same or approximately the same preceding cycle length and coupling intervals (fig. 7).

It was not possible to identify the location of A-V delay by the methods used in this study. Animal investigation has demonstrated delays at the A-V nodal area as well as within the SCS.8,10

Electrocardiographic Patterns

Clinical aberration has demonstrated an 85% likelihood of RBBB configuration when resulting from atrial premature beat,12 and an 80% likelihood of such a configuration when recorded during atrial fibrillation.12 It is not generally appreciated that Ashman and Byer12 were in fact the first to describe the prevalence of RBBB aberration. They said: “It was of particular interest to us to discover that in nearly 200 hearts, the greater delay was in the right branch in 85% and, sometimes more doubtfully, in the left branch in 15%. . . .” In many of the cases of left bundle delay, the left ventricle was hypertrophied. These authors concluded: “It is possible that if aberration could be studied in a sufficiently large series of healthy hearts, the right branch would prove to show the greater delay in much more than 85% of the cases.”

Our study yielded a 100% incidence of RBBB aberration in patients demonstrating AbC.

Animal studies have demonstrated that atrial premature stimuli may actually be blocked within a bundle branch. The right and left bundle branch qualitatively respond in the same manner.10,14 The canine right bundle-branch system generally takes slightly longer to repolarize than the left; consequently, it is more likely to slow or prevent passage of a premature impulse.8 Scherf15 has emphasized that a bundle-branch block pattern may result not only from failure of impulse propagation, but also from a 0.04-sec delay in
conduction time in one of the bundle branches. IRBBB may result from a delay of only 0.02 sec. Whatever the actual mechanism, the results of this study have demonstrated a predominance of block or inhibited conduction within the right bundle branch as opposed to the left bundle branch.

IRBBB aberration was transformed to RBBB aberration in eight patients by decreasing the coupling interval of the premature stimulus (figs. 1 and 4). This finding tends to implicate the same mechanism of production in both patterns and supports Dodge and Grant’s observation16 that IRBBB frequently precedes RBBB. Such was the case in 20% of their series of 50 patients with RBBB.

Grant17 has taken the position that RBBB is a result of distal impulse blockade. He cited as partial evidence the “identical” initial 0.04-sec forces in the electrocardiograms of patients displaying both normal and RBBB patterns. Other authorities do not adhere as strictly to the criterion of “identical” initial forces. Massie and Walsh,19 for example, suggested that in the great majority of cases, the conduction disturbance does not significantly alter the instantaneous forces produced in the first 0.04-sec.

In a critical review of experimental canine RBBB produced by surgical interference with the main right bundle, Dodge and Grant16 noted that all cases resulted in altered initial 0.04-sec forces.

In this study, the RBBB aberrant complex had initial forces which were found to be identical to the normally conducted beats in 47% of the patients. Hence the RBBB produced by this experimental method is comparable to that seen in clinical RBBB. The remaining 53% of the patients had initial forces which deviated from normal. Sandler and Marriott13 also noted this disparity in the initial instantaneous forces of RBBB aberration during atrial fibrillation. There is no clear explanation for the phenomenon. It is possible that the identical early scalar vector of the AbC RBBB pattern might result from distal blockade, whereas a nonidentical initial vector might result from proximal block within the right bundle branch.

Two instances of AbC with intraventricular conduction disturbance (IVCD) were noted. One was without normal left-to-right septal activation. A qR was present in lead V1 and the terminal forces were to the right. The initial forces conform to Massie and Walsh’s classification of the variant type of RBBB;19 however, superior axis deviation was not present. The other example of IVCD had a left axis, initial q in lead I, and a qR in V1. The terminal forces were not to the right (fig. 4). Although it is tempting to invoke block or delay in both the right and the left conducting systems, neither pattern conforms to those described as bilateral bundle-branch block.20

Attempts have been made to formulate workable rules for the clinical recognition of AbC by electrocardiographic patterning.21 It remains a difficult diagnosis in atrial fibrillation or in supraventricular arrhythmias with absence of clearly recognizable P waves. The findings of this report confirm the preponderance of RBBB with an rR’ in V1, and similar to normal, but not identical, 0.04-sec forces. A point not emphasized previously is the variety of patterns which may be present in the same patient, and which may include both right and left bundle-branch block.

**Significance of Aberrant Ventricular Conduction**

There is a marked difference of opinion among authorities regarding the significance of aberrant ventricular conduction. Some believe that it implies a pathological conducting system,22–24 while others believe that it may be a normal electrophysiological event.25, 26 For example, Batley28 stated: “Ventricular aberration is frequently observed in premature ectopic beats or an ectopic tachycardia, flutter, or fibrillation, particularly if there is an underlying defect of intraventricular conduction.” The opposite view is expressed by White26: “Such abnormal response has not been found to have any clinical significance as it is a more or less normal, transient, functional condition.”
This study establishes that AbC is a physiological event which does not require covert pathology of the conducting system in man, for AbC was induced in six subjects considered to have normal hearts on the basis of history, physical examination, and electrocardiograms.

References
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