Rate Dependent Aberrancy

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SUMMARY
Forty patients with "rate dependent aberrancy" (RDA) were studied. This large group of patients permitted a clear definition of the syndrome and recognition of a number of features not previously described. These proved to have a significant bearing on the recognition and differential diagnosis of RDA and non-rate dependent aberrancy.

It was found that a small change in cycle length, perhaps too small to be recognized in the surface ECG, can result in RDA. Consequently, if a critical shortening of cycle length is to be recognized, it is necessary to record not only the onset of aberrant rhythm but also sufficiently long strips with normal intraventricular conduction preceding and following the RDA. In some patients there was no recognizable sudden change in cycle length and the onset of aberrancy was a function of the duration of the accelerated rate. In others only the first cycle of the rhythm with RDA was shortened and the remaining R-R intervals were paradoxically longer than the R-R cycle which initiated the RDA.

The aberrancy in RDA occurred at relatively slow heart rates (in 26 of the 40 patients the rate was below 80), and was frequently independent of any significant changes in the duration of the immediately preceding cycle length. There was a striking prevalence of left bundle branch block, and in 35 of the 40 patients obvious organic heart disease was documented.

Additional Indexing Words: Aberrancy QRS aberrancy Bundle branch block Left anterior hemiblock

IN 1913 Sir Thomas Lewis1 reported the first instance of intermittent left bundle branch block (LBBB) and although the normal QRS and the bundle branch block (BBB) patterns were not recorded in the same tracing, the heart rate was slower when the normal tracing was inscribed. This may well have been the first observed instance of rate dependent BBB. The phenomenon of intraventricular block at a certain critical heart rate has since been described in scattered reports under a variety of names, of which "rate dependent aberrancy" (RDA) appears most descriptive and will be used in this paper.2-9

Review of the available literature suggests that some important electrocardiographic (ECG), electrophysiologic and clinical features of RDA have not been clearly defined or have totally escaped detection. This is in large measure due to the retrospective nature of most of the studies, the small numbers in each study and the inability to recognize critical interval changes in the conventional surface ECG. Advent of intra-atrial pacing coupled with sophisticated instrumentation circumvented this last obstacle.

The purpose of this report, based on a study of 40 patients with RDA is to detail electrophysiological and clinical characteristics of rate dependent aberrancy.

Material and Method
Forty patients exhibiting normal and rate dependent aberrant intraventricular conduction in the same tracing, in whom the onset of aberrancy was recorded, were included in this study. The clinical records of 36 of the 40 patients were available and were reviewed. Heart disease was assumed to be present only when cardiomegaly, organic valvular disease with or without evidence of heart failure, angina pectoris supported by ECG abnormalities, or myocardial infarction were documented.

The ECG was examined as to (1) the type of intraventricular conduction delay, (2) whether the delay appeared abruptly or gradually, (3) the shortest R-R interval (fastest heart rate) at which normal conduction was present, (4) the R-R interval at which...
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aberrancy became evident, (5) the longest R-R interval (slowest rate) at which aberrancy persisted, once initiated, (6) the control, basic rhythm and (7) the cause of prolongation of the R-R interval which was in turn responsible for normalization of intraventricular conduction.

Three patients were studied with right atrial pacing to determine the magnitude of change in cycle length necessary to induce aberration. The atrium was paced using a pulse generator which delivered rectangular pulses of 2 msec duration and 2–4 volts intensity, through an isolation transformer (Digipulser Model DS-

Table 1

Clinical and Electrocadiographic Data

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Abbreviations: BBB = bundle branch block, RDA = rate dependent aberrancy, L = left, R = right, LAH = left anterior hemiblock, IR = incomplete RBBB, ASHD = arterioesclerotic heart disease, RHD = rheumatic heart disease, HHD = hypertensive heart disease, AS = aortic stenosis, S-A = sinus, APC = atrial premature contraction, PAT = paroxysmal atrial tachycardia, AF = atrial fibrillation, AFL = atrial flutter, A-VD = ativoventricular dissociation, VPC = ventricular premature contraction.
1, Isopuler Model PC-3, WP Instruments, Inc.) with the driving cycles shortened by intervals of 10 msec. The tracings were recorded on an Electronics for Medicine DR8 at a paper speed of 100 mm/sec.

Results

The clinical and electrocardiographic findings for all patients are presented in Table 1.

The age of the patients varied from 26 to 90 with an average of 70 years for the entire group. Heart disease, as defined above, was documented in all but five. In one patient a careful evaluation failed to disclose symptoms or signs of heart disease, while in four others clinical records other than the ECG were not available. Of the remaining 35, 24 had arteriosclerotic heart disease with acute myocardial infarction in four; three patients had aortic stenosis; one had aortic stenosis and myocardial infarction; four patients had hypertensive cardiovascular disease; one had rheumatic valvular disease; and in two cardiomegaly was of obscure etiology.

Sinus rhythm was recorded in 26, atrial fibrillation in ten, atrial flutter in one, sinus rhythm and paroxysmal atrial tachycardia (PAT) in two, one of these also exhibiting atrial premature contractions, atrioventricular (A-V) dissociation due to accelerated junctional rhythm in one.

The heart rate at which QRS aberrancy became manifest varied from 48 to 130 beats/min in 39 patients; in one patient aberrancy became manifest at a rate of 176 beats/min. The average heart rate for the group was 89 beats/min. In 26 of the 40 patients, the rate at which BBB became manifest was below 80 beats/min.

The types of aberrancy were LBBB in 32 patients (Fig. 1), right bundle branch block (RBBB) in four (Fig. 2), left anterior hemiblock (LAH) in three, and in one patient incomplete RBBB was present. In three patients the transition from normal QRS complex to a LBBB complex was gradual, with incomplete LBBB preceding the LBBB (Fig. 3, rows 1,3). In 17 patients the three standard limb leads were available for analysis. Ten showed a shift of the axis from normal to greater than −30 degrees (LAH) with appearance of LBBB, suggesting that LAH cannot be recognized in the presence of complete LBBB.

The presence or absence of aberrancy was frequently dependent on very small, critical changes in cycle length. In fact it was at times difficult, if not impossible, to detect a difference between the cycles terminated by the last normal QRS and the first aberrant QRS. It was not until a comparison was made with earlier cycles having a normal QRS that the rate dependent nature of the QRS

Figure 1

Parts of a continuous tracing (lead I) recorded from a patient with sinus rhythm. Acceleration of the heart rate with amyl nitrate results in RDA with LBBB configuration. Two interesting features of RDA are demonstrated in this tracing. A comparison in the duration of the last cycle terminated by a normal QRS (cycle 2) with the first cycle terminated by aberrant QRS (cycle 3) fails to reveal any shortening (R-R 700 msec). However, a comparison of cycle 3 with an earlier cycle, e.g., cycle 1, clearly discloses the RDA. The second interesting feature is the persistence of aberrancy even when the heart rate slowed below the rate which initiated the RDA (cycle 4). The difference between cycles 3 and 4 is 140 msec.

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aberrancy became obvious (fig. 1). In figure 4 a different phenomenon also demonstrates the critical nature of the RDA. In this ECG the presence or absence of aberration of the QRS following the ventricular premature contraction (VPC) depends on the morphology and, thus, the site of origin of the VPC and is probably due to variation in activation time of the bundle branch by the two different VPCs. The VPC (B) which arrives in the LBB later is followed by a shorter left bundle-to-left bundle interval and the compensatory pause is, therefore, terminated by an aberrant QRS. On the other hand, the VPC (A) which activates the LBB earlier, is followed by a longer bundle branch recovery time and the compensatory pause is terminated by a normal QRS complex. In one patient with PAT no change in cycle length was demonstrable preceding the onset of aberrancy (fig. 3, rows 1,4).

The critical nature of cycle length change which may result in RDA was confirmed in three patients during right atrial pacing. In all three, shortening of the cycle length by 10 msec resulted in QRS aberrancy (fig. 5). In one patient the RDA appeared without any change in rate of atrial pacing. Occasionally, only the first cycle of RDA was shorter than the cycles terminated by normal QRS complexes (fig. 6). In such cases a failure to record the onset of aberrancy would lead to an erroneous diagnosis of non-rate dependent BBB or even bradycardia dependent aberrancy.

The changes resulting in prolongation of the R-R cycle length and normalization of the QRS varied from patient to patient. Slowing of ventricular response in atrial fibrillation was observed in ten patients (fig. 7) and in ten slowing of the sinus rate resulted in normal intraventricular conduction (fig. 1). A compensatory pause following APC and VPC was the mechanism of normalization in six and seven patients (figs. 2, 4) respectively. In four patients normalization of the QRS followed A-V block; in one it followed sino-atrial (S-A) block. In two patients each, two separate mechanisms were responsible for normalization, namely, sinus slowing and a compensatory pause following either an APC or VPC.

In nine patients the BBB, once initiated, persisted despite slowing of the rate to below that which precipitated aberrant conduction (figs. 1, 5, 6, 8). The difference between the cycle length resulting in aberrancy and the paradoxically longer cycle once the aberrancy was initiated varied from patient to patient. In five patients with atrial fibrillation (fig. 8) the difference was 50, 100, 120, 210 and 280 msec, respectively. In the four patients with sinus rhythm (figs. 1, 6) the difference was 120, 82, 80

Figure 2
Sinus rhythm at a rate of 75 beats/min (R-R 800 msec) with QRS of RBBB morphology. APC followed by a compensatory pause of 920 msec permits recovery of the right bundle and normalization of the QRS. This tracing (V1) stresses the slow rates at which RDA may appear.

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and 120, while in one with A-V nodal rhythm the difference was 210 msec. In one patient the "overlap" with right atrial pacing was 110 msec (fig. 5).

In a single patient both RDA and Ashman phenomenon were present (fig. 7). The aberrancy due to the Ashman phenomenon was of the RBBB morphology, exhibited a coupling interval and followed a cycle, both of which were considerably longer than those recorded with RDA. The coupling interval of the QRS initiating the RDA was at times longer (†), equal to or only slightly shorter than the immediately preceding cycle, suggesting that RDA is much less dependent on duration of the immediately preceding cycle than is the Ashman phenomenon.

**Discussion**

In the normal heart, premature excitation is the most common cause of aberrancy and is due in large measure to excitation before repolarization is complete.10, 11 The duration of the cycle immediately preceding the cycle terminated by aberrant QRS, which henceforth will be referred to as the

**Figure 3**

This tracing, recorded during repetitive episodes of PAT, demonstrates (1) that the degree of the BBB may be progressive, i.e. the RDA may be manifest by incomplete and then complete BBB, (2) the independence of RDA from the duration of the "preceding cycle" length and (3) the difference between the Ashman phenomenon and RDA.

Rows 1 and 2 are portions of a continuous tracing of lead II (L2) demonstrating the onset and termination of PAT. The degree of aberrancy increased gradually over a number of cycles progressing from incomplete to complete LBBB despite the absence of any demonstrable change in heart rate. In row 2 the sinus node is suppressed and the PAT is followed by junctional escapes with ventricular echoes due to reciprocation. Only the first of the three reciprocating complexes is aberrant and this may be due to the "fatigue" phenomenon.

In row 3 the first QRS of the PAT is aberrant (†). It is followed by normal QRS and a gradual secondary appearance of aberrancy. The first aberrant QRS is due to a preceding long R-R interval and represents the Ashman phenomenon. The aberrancy recurs without any demonstrable change in cycle length and is an example of RDA. The RDA is most likely dependent on both the rate and "fatigue" with persistence of the tachycardia.

Row 4 (lead V5) similarly demonstrates onset of aberrancy without change in cycle length. In fact, the R-R interval terminated by the first aberrant QRS († †) appears to be 20 msec longer than the first cycle (†) of the PAT. Ordinarily, one would expect the QRS (†) following a long cycle to exhibit aberrancy.
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This figure was obtained shortly after open heart surgery and demonstrates A-V dissociation with an atrial rate of 130 beats/min and junctional rate of 75 beats/min with LBBB due to RDA.

The dominant rhythm is interrupted by VPC of two different configurations. The VPC with RBBB morphology (A) is followed by a junctional complex with a normal QRS, while the VPC with LBBB morphology (B) is followed by junctional complex with aberrant QRS. The most likely explanation is that the QRS of RBBB type originating in the left ventricle arrives at and discharges the LBB earlier than the QRS of LBBB type which originates in the right ventricle. The former VPC (A) is followed by a longer left bundle-to-left bundle interval and normalized QRS while the latter VPC (B) is followed by a shorter (critical) left bundle-to-left bundle interval and an aberrant QRS complex.

"preceding cycle," is important in any discussion of aberrancy. It has been shown that the duration of recovery is directionally related to the length of the cardiac cycle. Thus, with the coupling of the premature complex remaining constant, and a changing duration of the "preceding cycle," aberration may follow the longer "preceding cycles," a relationship referred to frequently as the Ashman phenomenon. In the normal heart, aberrancy may appear during sustained tachycardia but then only rarely and only with extremely rapid rates, and is most likely related to excitation prior to complete repolarization or during the refractory period. The two are not always synonymous. The aberrancy associated with tachycardia may terminate with shortening of the recovery time of the action potential, a normal phenomenon which takes place over a number of cycles after an abrupt onset of a tachycardia. In the absence of heart disease, aberrancy with isolated premature excitation or...

![Figure 4](image)

**Figure 4**

These tracings recorded during right atrial pacing demonstrate (1) the critical nature of the cycle length change which results in aberrancy and (2) the persistence of aberrancy, once begun, at cycle lengths longer than those which initiated the aberrancy.

The records (leads I, II, III, and V6) were obtained during gradual speeding (panel A) and gradual slowing (panel B) of the pacing rate. When the driving cycle length was shortened from 510 to 500 msec (panel A), LBBB appeared. The BBB persisted in spite of the prolongation of the R-R to 600 (panel B). Again the critical nature of the aberrancy is demonstrated by normalization of the QRS with a 10 msec prolongation in the cycle length (from 600 to 610 msec).

*Figure 5*

* Circulation, Volume XLVIII, October 1973
This figure demonstrates that once RDA is initiated, the aberrancy may be perpetuated at a rate slower than during normal intraventricular conduction.

The records are a part of a continuous tracing (lead V1). In each row a critical shortening of the R-R cycle (●) initiates the RDA. However, the RDA continues at cycle lengths which are longer (1) than those recorded with normal intraventricular conduction (C).

Each sequence of RDA is terminated by a short cycle with a paradoxically normal QRS (S). The exact explanation is not clear; however, during a critical portion of diastole, supernormal conduction in the left bundle branch may explain this paradox. Similarly, sudden uniform prolongation of conduction in both bundles would explain the normal QRS. The latter is supported by the appearance of a prolonged P-R interval preceding the unexpected normalization of the QRS complex.

sustained tachycardia is usually of RBBB configuration, and rarely of the LBBB type.15,16

It is clear from the present study that RDA differs in a number of respects from the aberrancy seen in the normal. It (1) occurs at relatively slow rates, (2) is most often of the LBBB type, (3) appears to be much less dependent on changes in duration of the "preceding cycle," (4) may occasionally appear...
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Figure 8

This tracing (leads I, II, III, V₆) demonstrates atrial fibrillation with RDA. The QRS aberrancy persists at R-R intervals longer (●) than those terminated by QRS with normal intraventricular (†) conduction.

Figure 9

This tracing is representative of a number of records (lead I) obtained in a patient with RDA and demonstrates suppression of bundle branch conduction as a possible mechanism for the paradoxical persistence of BBB at rates at which the QRS would be expected to normalize.

In B the sinus rhythm is interrupted by PVC and a ventricular escape. As expected, the longer R-R intervals are terminated by normal QRS (●) and the shorter R-R by QRS with LBBB.

In A ventricular tachycardia is followed by QRS with LBBB. The QRS normalizes as the rate accelerates. The difference between the first posttachycardia cycle terminated by LBBB (1620 msec) and the first cycle terminated by a normal QRS (1120) is 500 msec. The most likely phenomena responsible for the paradoxical behavior of LBB conduction include concealment of the ventricular impulses into the bundle and suppression of conduction.
without any, or with only slight changes in cycle length, and (5) is seen almost exclusively in patients with heart disease.

The frequent independence of the RDA from the influence of the duration of the "preceding cycle," appears to be an important electrophysiological difference separating it from aberrancy seen in the normal. This difference is supported by the following observations: (1) aberrancy may not be evident immediately after a long "preceding cycle" but may appear after a number of shorter cycles which are of the same duration, (2) with the onset of a regular tachycardia the first QRS after a long "preceding cycle" may be aberrant, and may then be followed by a sequence of normal QRS and finally by secondary reappearance of BBB (fig. 3, row 3). The initial aberrancy is a manifestation of the Ashman phenomenon while the recurrence of aberrancy represents RDA.

The exact mechanism of RDA remains obscure. The complete BBB seen on the surface ECG may be due to slowing of conduction in the bundle or actually complete block of conduction. There is little doubt that RDA is a manifestation of heart disease and the accompanying cardiac pathology is frequently diffuse, as for example in patients with coronary artery disease. The slow rates at which the BBB appears suggests that excitation prior to full electrical recovery (repolarization) may not be the sole mechanism of the aberrancy. If such were the case, one would have to assume an action potential of very long duration. Other possible mechanisms responsible for depression of conduction and thus aberrancy include a uniform reduction in the resting potential of the bundle branch and/or change in membrane responsiveness.

Obvious heart disease was present in all but one of our patients with RDA. On the other hand, in some instances of established LBBB evidence of significant heart disease may be absent. It is possible that in such patients the BBB is due to a localized area of fibrosis interrupting the bundle branch, with the rest of the myocardium and the coronary vessels normal. However, if rate dependent BBB is a function of altered local perfusion, significant clinical heart disease is most likely to be present.

In some cases, normalization of intraventricular conduction did not take place until the R-R interval was significantly longer than the R-R which initiated the RDA. A number of phenomena may explain this paradox. These include (1) concealment of an impulse arriving from the contralateral bundle into the blocked bundle, (2) "fatigue" of the blocked bundle, (3) concealed invasion as in (1), but with suppression of conduction somewhat analogous to suppression of pacemakers by ectopic beats (fig. 9), and (4) prolongation of recovery time. The difference of 210 msec observed in one patient suggests that in any one case more than one factor may be responsible. For example, in this case concealment could not have been the sole factor responsible for the persistence of the BBB because the difference of 210 msec is too large to be accounted for simply by the time involved in conduction from the RBB to the LBR.

The cause of the unexpected failure of recovery of intraventricular conduction may be different in patients with sinus rhythm and those with atrial fibrillation. In the latter, the discrepancy may be due to simple concealment of fibrillatory impulses into the bundle branch so that the bundle branch-to-bundle branch interval may be much shorter than suggested by the manifest QRS-to-QRS interval of the surface ECG. Although concealment into the bundle branch as such has not been documented, concealment into the A-V junction is clearly a mechanism responsible for the varying ventricular rate in atrial fibrillation.

Differentiation of RDA from aberrancy due to the long-short cycle relationship (Ashman phenomenon) is of some clinical importance. For example, a comparison of cycle lengths is frequently employed to differentiate ventricular from supraventricular rhythms with aberrancy. Aberrancy of the QRS, as a rule, follows a longer "preceding cycle." Thus, when a normal QRS terminates the short cycle in a long-short cycle grouping and is followed by anomalous QRS, a ventricular origin for this anomalous QRS is favored. The present study suggests that this differential criterion is not always reliable because such a sequence may be a feature of RDA (fig. 3, row 4).

Likewise, LBBB aberration cannot be taken as strong evidence of ventricular tachycardia simply because LBBB is a characteristic feature of RDA.

When a diagnosis of RDA is suspected the critically small changes in rate which may initiate the aberrancy must be considered. These changes may be in the order of 10 msec (figure 5), a difference most difficult, if not impossible, to detect on a surface ECG because of the limitations of the conventional direct writing ECG at a paper speed of 25 mm/sec.

Because of the critical duration of the cycle length which determines the presence or absence of
aberration, it may well be that some of the cases of intermittent BBB are a manifestation of RDA with extremely small changes in cycle length. Such an example is shown in figure 10. The presence of normal QRS in leads V4 and V6 clearly suggests that QRS aberrancy recorded in leads I, II, III, V1, V2 and V3 during PAT is an example of RDA. It is reasonable to assume, therefore, that the intermittent normalization of the QRS in leads I, II and III is most likely due to changes in cycle length not recognizable in the surface ECG. It should be pointed out that in this particular tracing only the presence of normal QRS in V4 and V6 makes it possible to recognize RDA in the remaining leads.

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