Alternate Patterns of Premature Ventricular Excitation During Induced Atrial Bigeminy

By Stafford I. Cohen, M.D., Sun H. Lau, M.D.,
Benjamin J. Scherlag, Ph.D., and Anthony N. Damato, M.D.

SUMMARY
Alternate patterns of premature ventricular excitation have been observed during induced atrial bigeminy in 18 subjects, including five normal volunteers. Each study was performed in the cardiac catheterization suite where a transvenous catheter electrode was positioned in the right atrium. Coupled or paired stimuli were delivered to the atrium by an isolated battery-powered source at an adjusted interval which resulted in alternate patterns of ventricular excitation from alternate premature beats ("alternating premature ventricular excitation").

In most instances "alternating premature ventricular excitation" occurred when parameters of preceding cycle length, premature coupling interval, and atroioventricular conduction time were constant.

In man also some observations were made of His bundle excitation during premature atrial stimulation, and in the intact dog heart some observations were made of alternating premature ventricular excitation during His bundle stimulation.

A tenable explanation for alternating premature ventricular excitation is advanced which rests on three postulates: (1) A long cycle length is followed by a long refractory period. (2) The refractory period of each branch of the specialized conduction system is dependent on its preceding cycle length or recovery period; and (3) the diastolic recovery period of a blocked segment of the specialized conduction system is shorter than the recovery period when blockade does not occur.

Additional Indexing Words:
Aberrant ventricular conduction
Left bundle-branch block
Specialized conduction system
His bundle stimulation (dog)

AN UNUSUAL form of alternating ventricular conduction has been observed in 18 patients during right atrial pacing studies. The purpose of this report is to describe the phenomenon and to propose an electrophysiological mechanism of alternating patterns of premature ventricular excitation during induced atrial bigeminy.

From the Cardiopulmonary Laboratory, U. S. Public Health Service Hospital, Staten Island, New York.

This work was supported in part by the Federal Health Program Service, U. S. Public Health Service Project Py 69-1, National Institutes of Health Grants HE-11829 and HE-12536 and NASA Contract T-22416.

Circulation, Volume XXXIX, June 1969

819

Methods
Observations were made on 18 subjects, including five normal volunteers by methods of investigation which have been previously described in detail.1,2 In brief, each study was performed in the cardiac catheterization suite with the subject in the nonsedated postabsorptive state and supine position. A bipolar or tripolar catheter electrode was introduced into an antecubital vein, utilizing sterile percutaneous technic and local anesthesia. The catheter was positioned against the lateral wall high in the right atrium under fluoroscopic and electrocardiographic control. Coupled or paired stimuli were delivered to the atrium by an isolated battery-power source (Medtronics R-wave, coupled pulse generator) at an adjusted milliamperage which would assure atrial capture. The atrial coupling interval, or the interval between paired pulses,
Method of producing alternating ventricular excitation: (A) Coupled atrial premature beats (R1-S coupling interval, 318 msec) results in normal ventricular excitation (R2). (B) All premature beats with a shorter coupling interval (276 msec) result in incomplete right bundle-branch block pattern of ventricular excitation. An S wave has appeared in lead I and an rsR' in lead V1. (C) Further reduction of the coupling interval (250 msec) results in an alternating pattern of ventricular excitation with alternate coupled atrial premature beats in which ventricular excitation alternates between complete right bundle-branch block and incomplete right bundle-branch block. (D) All premature beats with a further decrease in coupling interval (233 msec) now result in a complete right bundle-branch block pattern.

was gradually decreased until an alternating pattern of ventricular excitation occurred from alternate premature atrial beats (fig. 1). The atrial coupled or paired pace interval was maintained above 300 msec to avoid the atrial vulnerable period.

In the remainder of this report, an alternating pattern of ventricular excitation from alternate coupled or paired atrial premature beats will be referred to as “alternating premature ventricular excitation.”

Direct brachial artery pressure was recorded during alternating premature ventricular excitation in one patient.

Supplementary observations are presented which relate to the mechanism of alternating premature ventricular excitation. These data include His bundle electrograms which were recorded in man during the induction of atrial premature beats. The details of the method have been described elsewhere. In addition, alternating premature ventricular excitation was produced in the intact hearts of four mongrel dogs by electrical stimulation of the His bundle through two Teflon-coated stainless steel wires which had been inserted by needle placement directly into the area of the His bundle. The sinus node was crushed after it was ascertained that the pattern of ventricular excitation appeared constant when activated by the normal sinus mechanism or by His bundle stimulation. Atrioventricular nodal rhythm usually followed the crushing of the sinus node. A His bundle stimulus was coupled to each spontaneous control beat at an adjusted interval which produced alternating premature ventricular excitation (fig. 2).

All electrocardiograms were displayed on a multichannel oscilloscopic photographic recorder (Electronics for Medicine or Sanborn 4560 series) and records taken at paper speeds of 25 to 100 mm/sec. His bundle recordings were taken at 200 mm/sec.

Nomenclature

Incomplete right bundle-branch block pattern is defined as a right bundle-branch block pattern of 0.08 to 0.11-sec duration.

Incomplete left bundle-branch block pattern is defined by criteria which include absence of q waves (with or without slurring of the R wave) in leads “facing” the left ventricle (leads I, aV5, V5, and V6), small or absent R waves in lead
V1, and QRS duration of 0.08 to 0.11 sec.7, 8 Left axis deviation was determined by the criterion of a mean frontal axis of −30° or beyond.

Results

The results are presented in Table 1. There were 22 instances of alternating ventricular excitation in the 18 patients. In order of frequency, the alternating patterns were as follows: Right bundle-branch block alternating with control excitation, 10 examples (figs. 3 and 4); left axis deviation alternating with right bundle-branch block with left axis deviation, four examples (fig. 5); right bundle-branch block alternating with right bundle-branch block with left axis deviation, two examples (fig. 6); right bundle-branch block alternating with incomplete right bundle-branch block, two examples (fig. 1); right bundle-branch block alternating with left bundle-branch block, two examples (fig. 7); incomplete left bundle-branch block alternating with control excitation, one example; and right bundle-branch block with left axis deviation alternating with control excitation, one example.

In four patients, it was possible to record two distinct types of alternating ventricular excitation at different coupling intervals. Alternating premature ventricular excitation occurred in 14 of 18 cases when the preceding cycle length, premature coupling interval, and conduction time were constant (figs. 2, 3, 5, and 8).

Alternating durations of the cycle (R2-R1) immediately preceding the cycle which terminated in the premature atrial beat (R1-R2) occurred in four of 18 cases (figs. 4 and 6). In each case, the shorter preceding cycle length was always associated with one form of premature ventricular excitation, and the
Table 1

Summary of Results

<table>
<thead>
<tr>
<th>Pt.</th>
<th>Age (yr)</th>
<th>ECG diagnosis</th>
<th>Alternating patterns</th>
</tr>
</thead>
<tbody>
<tr>
<td>J.G.*</td>
<td>43</td>
<td>N</td>
<td>RBBB C</td>
</tr>
<tr>
<td>R.H.*</td>
<td>31</td>
<td>N</td>
<td>RBBB C</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>RBBB LAD RBBB</td>
</tr>
<tr>
<td>B.A.</td>
<td>58 Inf. MI</td>
<td>RBBB</td>
<td>C</td>
</tr>
<tr>
<td>V.C.</td>
<td>48 LAD</td>
<td>RBBB</td>
<td>C</td>
</tr>
<tr>
<td>B.B.*</td>
<td>35 N</td>
<td>LAD</td>
<td>RBBB LAD</td>
</tr>
<tr>
<td>P.I.*</td>
<td>30 N</td>
<td>LAD</td>
<td>RBBB LAD</td>
</tr>
<tr>
<td>R.N.</td>
<td>51 S1S2S3</td>
<td>RBBB</td>
<td>C</td>
</tr>
<tr>
<td>F.S.</td>
<td>62 LAD</td>
<td>RBBB LAD LAD</td>
<td>LAD</td>
</tr>
<tr>
<td>F.F.*</td>
<td>32 N</td>
<td>RBBB</td>
<td>C</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>RBBB IRBBB</td>
</tr>
<tr>
<td>J.S.</td>
<td>54 NS ST &amp; T</td>
<td>RBBB LAD</td>
<td>C</td>
</tr>
<tr>
<td>A.D.</td>
<td>70 LAD</td>
<td>RBBB</td>
<td>C</td>
</tr>
<tr>
<td>J.A.</td>
<td>51 LVH</td>
<td>RBBB</td>
<td>C</td>
</tr>
<tr>
<td>H.L.</td>
<td>36 Inf. MI</td>
<td>RBBB</td>
<td>IRBBB</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>RBBB LBBB</td>
</tr>
<tr>
<td>G.R.</td>
<td>19 N</td>
<td>LAD</td>
<td>RBBB LAD</td>
</tr>
<tr>
<td>G.G.</td>
<td>50 R/S V1 &gt; 1</td>
<td>RBBB</td>
<td>C</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>RBBB LAD RBBB</td>
</tr>
<tr>
<td>G.A.</td>
<td>51 LAD</td>
<td>LAD</td>
<td>RBBB LAD</td>
</tr>
<tr>
<td>F.H.</td>
<td>70 N</td>
<td>RBBB</td>
<td>LBBB</td>
</tr>
<tr>
<td>T.F.</td>
<td>55 LAD Inf. MI</td>
<td>RBBB</td>
<td>C</td>
</tr>
</tbody>
</table>

* Normal volunteers.

Abbreviations: N = normal; LAD = left axis deviation; Inf. MI = inferior myocardial infarction; NS ST & T = nonspecific ST & T-wave abnormality; LVH = left ventricular hypertrophy; R/S V1 > 1 = R/S ratio in lead V1 is greater than 1; RBBB = right bundle-branch block; RBBB LAD = right bundle-branch block and left axis deviation; C = control QRS; LAD = left axis deviation; IRBBB = incomplete right bundle-branch block.

longer preceding cycle length was always associated with the alternate form of premature ventricular excitation.

The peak arterial pressures generated by alternate pathways which were not necessarily reflected in measurements of atrioventricular conduction time, preceding cycle length, and premature coupling interval.

When a premature atrial beat results in a pattern of aberrant ventricular conduction, there is either a primary delay in excitation through a branch of the specialized conduction system or functional block of a branch of the specialized conduction system. In the event of a functional block of conduction, the ventricular myocardium supplied by the blocked branch is ultimately activated by an indirect route. The blocked branch has a shorter diastolic recovery period than the unblocked branches by virtue of its late activation. It follows that the next coupled atrial premature beat should find the previously blocked segment less refractory than alternate pathways. This "unblocking" permits either normal or less delayed conduction. Thereafter the recovery time of the segment would once again be relatively longer because of a longer diastolic recovery period and the coupled atrial premature beat which follows the "unblocking" may once again find the branch in an increased refractory state which does not permit normal passage. This view is presented in schematic form in figures 3, 5, 6, and 7. The alternating patterns of ventricular excitation indicate that the right bundle branch, anterior division of the left bundle branch, and common left bundle branch may have blocked or delayed conduction and that the refractory period of each branch of ventricular excitation varied with the pattern type in the one case in which it was recorded (fig. 4).

Selected records of His bundle electrograms are presented which demonstrate aberrant ventricular conduction with retrograde His bundle excitation (fig. 9), and alternating ventricular excitation with unchanged atrium to His bundle and prolonged intraventricular conduction times during aberrant beats (fig. 8).

Discussion

This report demonstrates that alternate coupled or paired atrial premature beats may excite the ventricles through alternate pathways. Katz and Pick illustrate a case of atrial bigeminy which is similar to the type of "alternating premature ventricular excitation" described herein. However, these authors ascribed alternating pathways of excitation to slight differences in atrial coupling intervals.

Altered ventricular excitation following an atrial premature beat depends upon the duration of the preceding cycle length, the coupling interval, and the atrioventricular conduction time. Each of these factors is of
importance in the production of alternating premature ventricular excitation because one or both of the alternating patterns is in the form of aberrant ventricular conduction.

The refractory period of the conduction system is directly related to the preceding diastolic interval.\textsuperscript{10, 11} It can be inferred from the duration of phase 3 of the myocardial action potential that there is a very sensitive relationship between diastolic cycle length and refractory period in man which may change from beat to beat in the presence of a changing heart rate.\textsuperscript{12} In four of the 18 cases, there was an alternation of the duration of the cycle length (R\textsubscript{2}-R\textsubscript{1}) which preceded alternate fixed-coupled atrial premature beats. The longer preceding cycle lengths resulted in greater prolongation of the refractory period of the specialized conduction system, and the shorter preceding cycle lengths resulted in less prolongation of the refractory period. Therefore, the atrial premature beats which followed the longer preceding cycle lengths had a greater chance of abnormally exciting the ventricle than those which followed the shorter preceding cycle lengths. In the majority of cases (14 of 18) electrophysiological parameters, such as preceding cycle length, atrioventricular conduction time, and premature coupling interval, were constant for each pattern of premature ventricular excitation. It is unlikely that the specialized conduction system would both permit and prevent conduction under the same electrophysiological circumstances. Analysis of the records revealed that alternating premature ventricular excitation resulted from electrophysiological changes within the branches of the specialized conduction system is dependent upon its rate of excitation (figures 3, 5, 6, and 7).

Figure 3
Right bundle-branch block alternating with normal control excitation: Coupled premature atrial beats result in alternating patterns of ventricular excitation. The coupling interval (R\textsubscript{1}, S), preceding cycle length (R\textsubscript{2}-R\textsubscript{1}), and A-V conduction time (S-R\textsubscript{1}) intervals are constant. A schematic representation of the recovery period of the right bundle branch is presented at the bottom of the figure. See text for explanation of why there is a longer diastolic recovery period of the right bundle branch following a normally conducted premature beat and why right bundle-branch block patterns of aberrant conduction follow the longer recovery period.
Figure 4

Alternating ventricular excitation with simultaneous atrial pulse: Coupled atrial premature beats result in alternating excitation of normal and right bundle-branch block patterns. Atrioventricular conduction time (S-R2) is constant for all premature beats. Coupling interval (R1-S) vary 10 msec. The diastolic interval (R2-R1) which follows normally conducted premature beats is consistently longer than the diastolic interval which follows premature beats of right bundle-branch block configuration. The peak systolic pressure in the brachial artery generated by normally conducted premature beats is consistently greater than that generated by the premature beats of the right bundle-branch block pattern.

Figure 5

Alternating left axis deviation with right bundle-branch block with left axis deviation: Paired atrial pacing results in alternating ventricular excitation. Preceding diastolic interval (R2-R1), premature atrial stimulation interval (R1-S2), and atrioventricular conduction time (S2-R2) are all constant. Pairs 1, 3, and 5 terminate in left axis deviation. Pairs 2, 4, and 6 terminate in right bundle-branch block with left axis deviation. The recovery periods of the right bundle branch and anterior division of the left bundle branch (which results in left axis deviation when blocked) are schematically represented at the bottom of the figure.
Consecutive supraventricular impulses may stimulate repetitive ventricular beats (fig. 10) or ventricular tachycardia. This event is believed to occur because the branch of the specialized conduction system which was initially refractory to antegrade conduction remains refractory to subsequent stimuli from above as a result of intermediary late (retrograde) activation. Repetitive functional block can thus occur, provided that the atrial input frequencies fall within required limits. An example is shown of retrograde activation of the His bundle in man after an aberrant beat of right bundle-branch block configuration (fig. 9). In all likelihood retrograde activation of the His bundle occurred through late activation of the right bundle branch. This observation would support the thesis that following the right bundle-branch block pattern of aberrant ventricular conduction, the right bundle branch has a shorter recovery period than that following normal conduction.

It is possible for retrograde conduction of a main bundle branch to occur without His bundle activation. Thus, retrograde His bundle activation does not follow all instances of aberrant conduction and is not apparent in figure 8. The fact that alternating ventricular excitation can be produced in dogs by His bundle stimulation precludes implicating pathways other than those of the specialized conduction system (fig. 2).

Several examples of alternating incomplete and complete bundle-branch block were noted (fig. 1). This type of alternation can be explained by the fact that the refractory period of the specialized conduction system is

**Figure 6**

Alternating right bundle-branch block and right bundle-branch block with left axis deviation: Coupled atrial premature beats result in an alternating pattern of excitation consisting of alternating right bundle-branch block and right bundle-branch block with left axis deviation. The coupling intervals (R1-S) and A-V conduction times (S-R2) are identical for all premature beats. The diastolic interval (R2-R1) is 60 msec longer in those cycle lengths preceding right bundle-branch block with left axis deviation. The diastolic recovery period of the right bundle branch and anterior division of the left bundle branch (which results in left axis deviation when blocked) is schematically represented at the bottom of the figure.
Alternating right bundle-branch block and left bundle-branch block: Coupled atrial premature beats result in alternating right bundle-branch block and left bundle-branch block patterns of excitation. Coupling interval (R₁-S) and conduction time (S-R₂) are constant. Diastolic intervals (R₂-R₁) vary but do not appear to influence the alternating pattern. The recovery periods of the right and left bundle branches are schematically represented at the bottom of the figure.

Figure 8
Alternating right bundle-branch block and normal conduction with His bundle recording: Coupled premature atrial stimuli result in alternating ventricular excitation; the preceding cycle length (R₂-R₁) and coupling interval (R₁-S) are constant. A His bundle electrogram (HBE) reveals that A-V conduction time (P-HB) is also constant. Intraventricular conduction time (HB-R₂) is longer for premature beats of right bundle-branch block configuration (190 msec) than premature beats of normal configuration (158 msec).
shortest at the His bundle and progressively increases as distal points are measured. Previous communications have noted the ease of transforming an incomplete right bundle-branch block pattern of aberrant ventricular conduction to a complete right bundle-branch block pattern of aberrant ventricular conduction by shortening the premature coupling interval. Incomplete right bundle-branch block is believed to result from distal block or delay of impulse passage and complete right bundle-branch block from proximal block of impulse passage. Because of the distal location of block in incomplete right bundle-branch block, it is unlikely that antidromic excitation can occur with retrograde activation of the right bundle branch. The premature atrial coupling interval can be adjusted from one which will always result in incomplete right bundle-branch block to one which will result in unsustained complete right bundle-branch block (fig. 1). In all likelihood, complete right bundle-branch block cannot be sustained because proximal block permits antidromic stimulation from the left side with retrograde activation of the right bundle branch. The result is shortening of diastolic recovery time and refractory period which permits the next coupled premature beat to traverse the proximal portion of the right bundle branch. The end result is alternating ventricular excitation of incomplete right bundle-branch block and complete right bundle-branch block patterns.

There is ample evidence that varied sites of direct ventricular stimulation result in varied patterns of ventricular excitation and stroke volume. It appears from the case in which a direct brachial pulse was obtained that each of the alternate patterns of aberrant ventricular conduction resulted in a different contractile force and peak systolic pressure.
Consecutive premature atrial impulses with consecutive aberrant ventricular excitation: The second and third labelled atrial stimuli (S) result in a right bundle-branch block pattern of ventricular excitation indicated by arrows.

References
Alternate Patterns of Premature Ventricular Excitation During Induced Atrial Bigeminy

STAFFORD I. COHEN, SUN H. LAU, BENJAMIN J. SCHERLAG and ANTHONY N. DAMATO

_Circulation_. 1969;39:819-829
doi: 10.1161/01.CIR.39.6.819

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1969 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/39/6/819

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/