The Dominant Pacemaker of the Human Heart

Antegrade and Retrograde Activation of the Heart

By John W. Lister, M.D., Abner J. Delman, M.D., Emanuel Stein, M.D., Ronald Grunwald, M.D., and George Robinson, M.D.

SUMMARY

In 25 cases at open heart surgery, atrial and ventricular close bipolar electrograms, an atrial unipolar electrogram, and a lead-II electrocardiogram were simultaneously monitored throughout each procedure. In six cases the ventricles or atria, or both, were paced by electrical stimulation. Although atrial activity could frequently not be clearly delineated on the lead II electrocardiogram, the time relationship between atrial and ventricular depolarization could at all times be determined from the electrograms.

During ventricular premature beats and ventricular rhythms, retrograde A-V conduction and retrograde activation of the atria were common, and unidirectional retrograde A-V block was infrequent. In spontaneous and induced cardiac arrhythmias the fastest pacemaker of the heart, atrial, His bundle, or ventricular, usually activated the entire heart.

ADDITIONAL INDEXING WORDS:
Electrograms, atrial and ventricular
Ventricular rhythms
Atrioventricular conduction
Cardiac arrhythmias

THAT the fastest pacemaker of the heart will activate the entire heart is an axiom in cardiac physiology. Yet, in the literature, there is a controversy as to the incidence of ventriculo-atrial conduction during ventricular rhythms in man.1-11

Most observations on cardiac arrhythmias have been made from the standard lead electrocardiogram. During arrhythmias, it is often difficult or impossible to delineate the relationship between atrial and ventricular depolarization from standard lead electrocardiographic tracings. In order to depict clearly atrial and ventricular depolarization during spontaneous and induced arrhythmias, electrograms from the atria and ventricles of the human heart were simultaneously recorded. The purpose of this report is to demonstrate the incidence of retrograde activation of the heart and to reaffirm the concept, that, in man, the fastest pacemaker of the heart will usually activate the entire heart.

Methods

Simultaneous close bipolar atrial and ventricular electrograms, an atrial unipolar electrogram, and a lead II electrocardiogram were monitored during open heart surgery in 25 cases. All patients were in sinus rhythm. The method used in this study for recording epicardial electrograms has been previously described in detail.12-17

Acrylic plaque electrodes containing five silver contacts 2 to 5 mm apart were sutured to the epicardial surface of the right atrium near the sinus node, to the free wall of the left atrium, and to the epicardial surface of the right ventricle. In four cases with an interatrial septal defect, the right atrial electrode plaque was positioned just above the A-V (atrioventricular) groove, and the left atrial electrode plaque was positioned superiority in the area bounded by the right pulmonary artery superiorly, the ascending aorta medially, and the superior vena cava.
cava laterally. The leads from the epicardial electrodes were connected to a distribution box, so that recordings could be obtained from any combination of electrodes. In six cases the epicardial electrodes were connected to an external pacemaker and the heart was paced from the ventricles or atria, or both.

The P-R and R-P intervals were measured from the earliest atrial and ventricular deflection recorded either in the electrograms or the electrocardiogram. All records were taken on an Electronics for Medicine oscilloscopic photographic cardiogram. The epicardial electrograms were obtained with the filters set at a frequency range of 40 to 200 cps. Records were taken at paper speeds of 50 and 200 mm/sec.

Results

Atrial activity frequently could not be clearly delineated from the lead II electrocardiogram, while the time relationship of atrial to ventricular activation was always clearly depicted by the electrograms (figs. 1 to 7). Retrograde A-V conduction and retrograde activation of the atria were easily recognized on the electrograms (figs. 1 to 4 and 6). Retrograde activation of the heart was present: (1) when there was an abrupt change in the atrial rate which related to the ventricular rate (figs. 1 to 4 and 6); (2) when there was a change in configuration of the bipolar and unipolar atrial electrograms (figs. 1 to 4 and 6); and (3) when there was a change in the time interval between the left and right atrial electrograms (figs. 2 to 4).

Ventricular premature beats usually resulted in retrograde conduction to the atria and suppression of the normal atrial impulse (fig. 1). When the atria and a part or all of the

---

**Figure 1**

Retrograde A-V conduction of ventricular beats. Simultaneous atrial and ventricular close bipolar electrograms (A and V), a close bipolar ventricular electrogram recorded with a roving electrode (R), and a lead 2 electrocardiogram. (Strip 1) The first three beats are sinus beats with a normal P-R interval. The fourth beat is a premature ventricular beat with retrograde A-V conduction to the atria. (Strip 2) The first beat is a sinus beat with normal A-V conduction. The next three beats are ventricular in origin. The first ventricular beat is not conducted to the atria, while the next two beats show ventriculo-atrial activation. The third ventricular beat is followed by a ventricular standstill of 1.16 sec and a sinus beat. The R-P interval is longer than the P-R interval. The shortening of the P-P interval and the change in contour of the close bipolar atrial electrogram demonstrates the presence of ventriculo-atrial activation. During the ventricular beats the retrograde atrial activation cannot be seen on the lead 2 electrocardiogram. The P-P, P-R, R-P, and R-R intervals are measured in seconds. Paper speed, 200 mm/sec; time lines at 0.04 sec.

*Circulation, Volume XXXV, January 1967*
Retrograde A-V conduction and A-V dissociation. Simultaneous right atrial (RA), left atrial (LA), and right ventricular close bipolar electrograms (RV), right atrial unipolar electrogram (RAU), and a lead 2 electrocardiogram (L2). (Strip 1) Normal sinus rhythm with a prolonged P-R interval. The right atrial electrogram precedes the left atrial electrogram. (Strip 2) Ventricular rhythm with ventriculo-atrial activation. The first four, the sixth through eighth, and tenth ventricular beats are conducted to the atria. After the fourth and eighth beats, there is a pause in the ventricular rhythm, the atria escape, and the fifth and ninth beats show A-V dissociation. During retrograde activation of the atria the configuration of the atrial electrograms and the time interval between the right and left atrial electrograms are altered. Antegrade and retrograde atrial activation cannot be clearly distinguished from the lead 2 electrocardiogram. (Strip 3) A-V dissociation. The time interval between atrial and ventricular depolarization is constantly changing. The interval between the right and left atrial electrograms is the same as in strip 1 and the antegrade atrial beats in strip 2. The atrial electrograms are of identical configuration to those of the antegrade atrial beats in strip 2. Atrial activity can be seen only in the first complex of the lead 2 electrocardiogram. P-P, P-R, R-P, and R-R intervals are measured in seconds. Paper speed, 50 mm/sec.
Figure 3

Varying ventriculo-atrial conduction. (Strip 1) Sinus arrhythmia with a prolonged P-R interval. The seventh beat is a premature atrial beat; note the change in the atrial electrograms of this beat. (Strip 2) The first through third and the sixth and seventh ventricular beats are conducted to the atria with a varying R-P interval suggestive of a retrograde Wenckebach's phenomenon. The remainder of the record, except the last beat, shows A-V dissociation. The last beat demonstrates normal A-V conduction. The occurrence of A-V dissociation while the ventricular rate is appreciably faster than the atrial rate, followed by a normal antegrade conducted beat suggests the presence of transient complete or incomplete unidirectional retrograde A-V block. The ventricular electrograms all have the same contour except for the beat with normal antegrade conduction. The lead 2 electrocardiogram does not clearly delineate the retrograde atrial activation. (Strip 3) Multifocal ventricular rhythm with ventriculo-atrial conduction. There are two different R-P intervals, 0.18 sec and 0.30 sec. After the first beat there is a prolonged pause in the ventricular rhythm of 1.26 sec followed by one beat of A-V dissociation. Same abbreviations as in figure 2. Paper speed, 50 mm/sec.

A-V node had been activated from above and was in a refractory state at the time a retrograde impulse reached the A-V junctional tissue, A-V dissociation resulted (figs. 1 to 4 and 6). Independent atrial and ventricular activation at about equal rates resulted in A-V
Figure 4
Antegrade and retrograde A-V conduction. (Strip 1) Sinus arrhythmia. The fourth beat is a premature atrial beat with a prolonged P-R interval. (Strip 2) Idioventricular rhythm with a rate of 41 beats per minute and 1:1 ventriculo-atrial conduction. The R-P interval during the idioventricular rhythm is shorter than the P-R interval during sinus rhythm. The retrograde P waves on the lead 2 electrocardiogram cannot be clearly separated from the QRS complexes. (Strip 3) A ventricular rhythm with 1:1 ventriculo-atrial activation followed by a slowing of the ventricular rhythm and a return to an atrial rhythm with normal antegrade A-V conduction. The second beat after the return of antegrade atrial activation is a ventricular escape beat with A-V dissociation. The R-P interval is greater than the P-R interval. Paper speed, 50 mm/sec.

dissociation (fig. 2). During A-V dissociation when there was a slight disparity between the atrial and ventricular rates and an atrial or ventricular impulse arrived at the A-V junctional tissue while it was in a nonrefractory state, there was antegrade or retrograde conduction (fig. 3). In figure 5, the first atrial beat which captured the ventricles, following atrial standstill and His bundle rhythm or ventricular rhythm, has a prolonged P-R interval.

A common phenomenon when the idioventricular rate was greater than the spontaneous atrial rate was 1:1 ventriculo-atrial conduction (table 1, figs. 2 to 4 and 6). In one instance an idioventricular rate of 41
beats per minute resulted in 1:1 ventriculo-
atrial conduction (fig. 4). During unidirec-
tional retrograde A-V block, though the idio-
ventricular rate was greater than the atrial rate, the ventricles did not capture the atria; however, when the atrial rate was greater than the idioventricular rate, there was normal antegrade A-V conduction and the supraventi-
cular pacemaker activated the entire heart (figs. 3, 5, and 7). Figure 5 shows a tracing

**Table 1**

<table>
<thead>
<tr>
<th>Instances of ventricular rhythm</th>
<th>1:1 Retrograde A-V conduction</th>
<th>Incomplete unidirectional retrograde A-V block</th>
<th>Complete unidirectional retrograde A-V block</th>
</tr>
</thead>
<tbody>
<tr>
<td>17-spontaneous</td>
<td>13</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>6-paced</td>
<td>5</td>
<td>0</td>
<td>1</td>
</tr>
</tbody>
</table>

**Figure 5**

Unidirectional retrograde block. (Strip 1) Sinus tachycardia with a normal P-R interval. (Strips 2 and 3) These are a continuous record. (Strip 2) High ventricular or His bundle rhythm with unidirectional retrograde A-V block and standstill of the atria. (Strip 3) The second and fourth atrial beats are conducted to the ventricles with prolonged P-R intervals. The prolongation of the P-R intervals is probably the result of retrograde concealed conduction in the A-V node of the previous beat. The last three atrial beats demonstrate normal antegrade A-V conduction. C = concealed retrograde A-V conduction. Paper speed, 50 mm/sec.
Figure 6

Activation of the entire heart by the fastest pacemaker. (Strip 1) Sinus tachycardia and left atrial pacing. The atria are paced at a faster rate than the sinus rate. The arrow points to the first beat captured by the pacemaker. Five beats later there is an escape sinus beat. The P-R interval is increased by the atrial pacing. (Strip 2) Ventricular pacing during sinus tachycardia. The ventricles are captured by the first pacemaker impulse. During the first five paced beats there is A-V dissociation. After the fifth paced ventricular beat, there is 1:1 ventriculo-atrial activation. The configuration of the atrial electrograms is altered during retrograde activation, but the interval between the left and right atrial close bipolar electrograms is about the same. The R-P interval is longer than the P-R interval. (Strip 3) Atrial pacing during a His bundle rhythm. The R-P interval of His bundle rhythm is 0.06 sec. The first pacemaker impulse captures the atria. This beat is followed by a premature beat (small arrow) which is conducted to the ventricles; the next four atrial paced beats suppress the His bundle pacemaker and are conducted to the ventricles. The interval between pacemaker impulses was increased from 0.72 to 0.92 sec between the last two beats; the pacemaker still captured the atria but the His bundle pacemaker escaped. The left atrial electrogram precedes the right atrial electrogram during sinus rhythm because the right atrial electrode plaque is positioned just above the A-V groove, and the left atrial electrode plaque is high in the left atrium. P.I. = pacemaker impulse. The P-R and R-P intervals were measured from the pacemaker impulse to the first atrial or ventricular deflection recorded. Paper speed, 50 mm/sec.
of a His bundle rhythm or high ventricular rhythm with unidirectional retrograde block and standstill of the atria. In this study there were three instances of complete unidirectional retrograde A-V block, and one instance of incomplete unidirectional A-V block (fig. 3, table I).

In five or six patients, pacing the ventricles at a faster rate than the spontaneous atrial rate resulted in 1:1 ventriculo-atrial conduction (fig. 6). In one case there was complete unidirectional retrograde A-V block (fig. 7). In figure 6, strip 2, the ventricles were paced at a faster rate than the spontaneous atrial rate. Although the ventricles were immediately captured by the pacemaker, there was A-V dissociation for five beats before the ventricles captured the atria.

Figure 6, strip 3, shows atrial pacing during spontaneous His bundle rhythm. The pacemaker was set at a rate about equal to that of the His bundle rhythm. The pacemaker captured the atria and the second through fifth paced atrial beats captured the ventricles and suppressed the His bundle pacemaker. The P-P interval between the fifth and sixth paced beats was lengthened and the His bundle pacemaker escaped.

In this study the fastest pacemaker of the heart whether atrial, His bundle, or ventricular, usually activated the entire heart.

**Discussion**

There is considerable variation in the reported incidence of retrograde A-V conduction and retrograde activation of the atria during ventricular rhythms in man.\(^1\)\(^-\)\(^11\) Though ventriculo-atrial activation during ventricular rhythms in the experimental animal has been a commonly observed phenomenon,\(^18\)\(^-\)\(^25\) most investigators believe that retrograde activation of the human heart is an uncommon occurrence.\(^1\)\(^-\)\(^8\)

Most observations on retrograde activation of the human heart have been made from standard lead electrocardiographic tracings of patients with varying degrees of A-V conduction disturbances.\(^1\)\(^,\)\(^26\) Scherf and Cohen\(^1\) reviewed from the literature 81 cases of ventriculo-atrial conduction and retrograde activation of the atria. All of the patients in the cases which they reviewed had varying degrees of A-V conduction disturbances. Retrograde conduction in patients who had fixed rate ventricular pacemakers because of complete heart block or Adams-Stokes seizures...
or both have been reported and have been observed in this laboratory several times.

During ventricular tachycardia it is often difficult or impossible to delineate atrial activity from the electrocardiogram. The QRS complex is often prolonged and the P wave is obscured because it falls into the latter part of the QRS, S-T segment, or T wave. Nevertheless, ventriculo-atrial activation in the presence of ventricular tachycardia has on occasion been documented by the electrocardiogram.

Kistin and Landowne and Kistin by the use of esophageal electrocardiography were able clearly to delineate atrial activity in the presence of ventricular premature beats and ventricular tachycardia. In their studies, in 15 of 33 cases of ventricular premature beats there was ventriculo-atrial conduction, and in 14 of 21 cases of ventricular tachycardia there was retrograde conduction to the atria. These authors concluded that ventriculo-atrial conduction in ventricular tachycardia is a common phenomenon. The results of our study are in agreement with those of Kistin and Landowne. When a ventricular impulse arrives at the A-V node during the nonrefractory period, ventriculo-atrial conduction will usually occur. The normal His-Purkinje system and A-V node comprise a two-way conducting system.

Schemroth and Friedberg demonstrated that the first ventricular beat captured by the atria after A-V dissociation has a prolonged P-R interval. In this study, the first beat after A-V dissociation frequently had a prolonged P-R interval when the atria captured the ventricles, or a prolonged R-P interval when the ventricles captured the atria. The prolongation of the P-R or R-P interval in the first beat after A-V dissociation probably indicates that the previous ventricular or atrial beat had partially penetrated the A-V node and that retrograde or antegrade concealed conduction in the A-V node had taken place.

Unidirectional A-V block probably only occurs when there is a disturbance in the A-V transmission system. Unidirectional retrograde A-V block appears to be a more common phenomenon than unidirectional antegrade block. In our opinion, unidirectional A-V block is best explained by decremental conduction within the A-V node and the direction of block is determined by the point of maximal delay or decrement within the A-V node. If an action potential of normal amplitude arrives at the area of maximal decrement, it will be conducted, but if an action potential of diminished amplitude arrives at this area, it will partially penetrate the area and die out. Therefore, if the point of maximal delay or decrement is high in the A-V node, unidirectional retrograde block will result, and if the point of maximal delay or decrement is low in the A-V node, unidirectional antegrade block will result. This explanation of unidirectional A-V block would account for the greater frequency of unidirectional retrograde block as compared to unidirectional antegrade block, because in the normal node the major delay in A-V conduction is high in the node at the atrio-nodal junction.

In our study the fastest pacemaker of the heart usually activated the entire heart. Retrograde A-V conduction was the rule when a ventricular premature beat arrived at the A-V node during the nonrefractory period or when the idioventricular rate was faster than the atrial rate. When the atria and ventricles were activated independently of each other at about equal rates, A-V dissociation was present. In five of six cases when the ventricles were paced at a faster rate than the spontaneous atrial rate, the atria were captured by the ventricles and there was ventriculo-atrial conduction. Retrograde unidirectional block was relatively uncommon. In one patient when the atrial rate was increased by pacing above that of the spontaneous His bundle rate, the rhythm was converted from a His bundle to an atrial rhythm.

Acknowledgment

The authors wish to thank Dr. Brian F. Hoffman, Chairman of the Department of Pharmacology, College of Physicians and Surgeons, Columbia University, and Mr. Samuel Ross for supplying the electrodes used in this study and for their technical assistance.
References


The Dominant Pacemaker of the Human Heart: Antegrade and Retrograde Activation of the Heart

JOHN W. LISTER, ABNER J. DELMAN, EMANUEL STEIN, RONALD GRUNWALD and GEORGE ROBINSON

Circulation. 1967;35:22-31
doi: 10.1161/01.CIR.35.1.22

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1967 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/35/1/22

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/