Factors Influencing Retrograde Conduction
Study of 30 Patients During Cardiac Catheterization*

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Retrograde conduction from ventricle to atrium is a well-recognized occurrence in the human heart. The phenomenon has been observed repeatedly during ventricular tachycardia (Kistin, 1961), with ventricular premature and interpolated beats (Kistin, 1959, 1963; Kistin and Landowne, 1951), in patients with ventricular pacemakers (Kastor and De Sanctis, 1967; Barold, Linhart, and Samet, 1968), and occasionally in the presence of high-grade atrioventricular (AV) block (Cohn and Fraser, 1914; Winternitz and Langendorf, 1944; Scherf and Cohen, 1964; Castillo and Samet, 1967; Kastor et al., 1967; Fletcher and Morton, 1968). Study of such events has been useful in elucidating the functional characteristics of the cardiac conduction system, and in explaining the electrophysiology of arrhythmias with bidirectional conduction.

The incidence of retrograde conduction, however, as reported, has shown considerable variation. This paper reports the result of a study designed to examine those factors which potentially might influence the frequency of ventriculo-atrial conduction in man. Particular attention was paid to the relation of the incidence and velocity of retrograde conduction to heart rate, the status of antegrade conduction, the nature of the underlying heart disease, and the presence of cardiac drugs.

METHODS

Attempts were made to induce ventriculo-atrial conduction in 30 patients admitted for diagnostic right and retrograde left heart catheterization. Bipolar electrode catheters were passed perversely into the right atrium, and simultaneous bipolar intra-atrial and single limb lead electrocardiograms were then recorded during mechanical stimulation of the left ventricle.

Retrograde conduction was identified by the finding in the intra-atrial recording of P waves that were different in form and premature in timing with respect to the sinus P waves. Bipolar intra-atrial leads were always used, since unipolar leads did not show as faithfully the changes in form with different impulse sources. Kistin (1963) has made similar observations in his studies with oesophageal leads.

RESULTS

Specific information about each of the patients is listed in Tables I and II. Retrograde conduction was observed in 14 of the 30 patients studied. The P-R intervals of conducted sinus beats were significantly shorter (p < 0.01) in those patients who showed retrograde conduction than in those who did not (Fig. 1). The time intervals from the beginning of QRS to the start of the retrograde atrial activation (R-P' intervals) were less than P-R intervals in 7 patients, equal in 1, and longer in 6 (Table I). The incidence of ventriculo-atrial conduction was not clearly related to the sinus rate (Fig. 2), to use of digitalis or other cardiac medication, or to the type of cardiac lesion present (Tables I and II).

Examples of the observed rhythms are displayed in Fig. 3–7. The tracings were prepared by direct copy of the original strips in order to increase clarity. Ladder diagrams have been constructed to illustrate our interpretations of the electrocardiographic events.

DISCUSSION

The results obtained in this study demonstrate the high frequency with which ventriculo-atrial conduction can occur under a variety of circumstances. Despite the use of random stimulation of
Retrograde Conduction

14 PATIENTS WITH RETROGRADE CONDUCTION STUDIED WITH INTRA-ATRIAL ELECTROCARDIOGRAPHS

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age and sex</th>
<th>P-R interval (sec.)</th>
<th>R-P' interval (sec.)</th>
<th>Sinus rate</th>
<th>Digitalis</th>
<th>Cardiac lesion(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>58 F</td>
<td>0.12</td>
<td>0.10</td>
<td>100</td>
<td>Yes</td>
<td>Mitral regurgitation; ruptured chordae tendineae</td>
</tr>
<tr>
<td>2</td>
<td>28 F</td>
<td>0.14</td>
<td>0.14</td>
<td>75</td>
<td>Yes</td>
<td>Aortic and mitral stenosis; aortic regurgitation</td>
</tr>
<tr>
<td>3</td>
<td>47 F</td>
<td>0.14</td>
<td>0.10</td>
<td>85</td>
<td>No</td>
<td>Idiopathic hypertrophic subaortic stenosis</td>
</tr>
<tr>
<td>4</td>
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<td>0.20</td>
<td>88</td>
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</tr>
<tr>
<td>5</td>
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<td>0.22</td>
<td>115</td>
<td>Yes</td>
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</tr>
<tr>
<td>6</td>
<td>45 F</td>
<td>0.14</td>
<td>0.12</td>
<td>85</td>
<td>Yes</td>
<td>Arteriosclerotic heart disease</td>
</tr>
<tr>
<td>7</td>
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<td>0.14</td>
<td>0.16</td>
<td>88</td>
<td>Yes</td>
<td>Arteriosclerotic heart disease</td>
</tr>
<tr>
<td>8</td>
<td>36 M</td>
<td>0.16</td>
<td>0.14</td>
<td>115</td>
<td>—</td>
<td>Primum atrial septal defect</td>
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<tr>
<td>9</td>
<td>2 M</td>
<td>0.16</td>
<td>0.20</td>
<td>115</td>
<td>Yes</td>
<td>Mitral regurgitation; ruptured chordae tendineae</td>
</tr>
<tr>
<td>10</td>
<td>54 F</td>
<td>0.16</td>
<td>0.12</td>
<td>85</td>
<td>No</td>
<td>No heart disease</td>
</tr>
<tr>
<td>11</td>
<td>54 F</td>
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<td>0.20</td>
<td>115</td>
<td>Yes</td>
<td>Mitral regurgitation; ruptured chordae tendineae</td>
</tr>
<tr>
<td>12</td>
<td>55 M</td>
<td>0.16</td>
<td>0.28</td>
<td>95</td>
<td>Yes</td>
<td>Aortic stenosis</td>
</tr>
<tr>
<td>13</td>
<td>42 M</td>
<td>0.18</td>
<td>0.16</td>
<td>90</td>
<td>No</td>
<td>Idiopathic hypertrophic subaortic stenosis</td>
</tr>
<tr>
<td>14</td>
<td>67 M</td>
<td>0.22</td>
<td>0.16</td>
<td>55</td>
<td>Yes</td>
<td>Mitral regurgitation</td>
</tr>
</tbody>
</table>

16 PATIENTS WITHOUT DEMONSTRABLE RETROGRADE CONDUCTION

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age and sex</th>
<th>P-R interval (sec.)</th>
<th>Sinus rate</th>
<th>Digitalis</th>
<th>Cardiac lesion(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>15</td>
<td>59 M</td>
<td>0.14</td>
<td>95</td>
<td>No</td>
<td>Aortic stenosis and regurgitation</td>
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<tr>
<td>16</td>
<td>57 M</td>
<td>0.16</td>
<td>90</td>
<td>Yes</td>
<td>Aortic stenosis and regurgitation</td>
</tr>
<tr>
<td>17</td>
<td>43 F</td>
<td>0.16</td>
<td>88</td>
<td>Yes</td>
<td>Aortic stenosis; arteriosclerotic heart disease</td>
</tr>
<tr>
<td>18</td>
<td>57 M</td>
<td>0.18</td>
<td>110</td>
<td>—</td>
<td>Mitral regurgitation</td>
</tr>
<tr>
<td>19</td>
<td>65 M</td>
<td>0.20</td>
<td>70</td>
<td>No</td>
<td>Aortic stenosis; arteriosclerotic heart disease</td>
</tr>
<tr>
<td>20</td>
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<td>78</td>
<td>Yes</td>
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</tr>
<tr>
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<td>45 M</td>
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<td>80</td>
<td>No</td>
<td>Aortic regurgitation</td>
</tr>
<tr>
<td>22</td>
<td>50 F</td>
<td>0.20</td>
<td>90</td>
<td>No</td>
<td>Mitral stenosis</td>
</tr>
<tr>
<td>23</td>
<td>62 M</td>
<td>0.20</td>
<td>70</td>
<td>No</td>
<td>Aortic stenosis</td>
</tr>
<tr>
<td>24</td>
<td>70 M</td>
<td>0.20</td>
<td>88</td>
<td>Yes</td>
<td>Aortic regurgitation and stenosis</td>
</tr>
<tr>
<td>25</td>
<td>53 F</td>
<td>0.20</td>
<td>75</td>
<td>Yes</td>
<td>Mitral stenosis</td>
</tr>
<tr>
<td>26</td>
<td>47 M</td>
<td>0.20</td>
<td>85</td>
<td>Yes</td>
<td>Aortic regurgitation</td>
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<tr>
<td>27</td>
<td>58 M</td>
<td>0.20</td>
<td>75</td>
<td>Yes</td>
<td>Mitral regurgitation; arteriosclerotic heart disease</td>
</tr>
<tr>
<td>28</td>
<td>57 F</td>
<td>0.20</td>
<td>95</td>
<td>Yes</td>
<td>Aortic stenosis and regurgitation</td>
</tr>
<tr>
<td>29</td>
<td>54 M</td>
<td>0.22</td>
<td>90</td>
<td>Yes</td>
<td>Aortic regurgitation</td>
</tr>
<tr>
<td>30</td>
<td>67 M</td>
<td>0.48</td>
<td>92</td>
<td>Yes</td>
<td>Aortic regurgitation</td>
</tr>
</tbody>
</table>

The incidence of retrograde conduction in our patients approached 50 per cent. Samet, Castillo, and Bernstein (1967), by selectively inducing the ventricular ectopic beat at a time when the ventricles and atria were least likely to be refractory, showed retrograde conduction in 29 of 31 subjects, some with normal and others with abnormal hearts. Thus, it is likely that the incidence of ventriculo-atrial conduction in the present series could have been increased if such specialized pacing techniques had been employed.

The existence of a relation between the speed of forward conduction and the incidence of retrograde conduction found in our patients has not, to our knowledge, been previously documented in man.

Though there was some overlap, the P-R intervals in those patients showing retrograde conduction were significantly shorter than in those who did not. Such a relation might be anticipated, since with the

![Retrograde Conduction](http://heart.bmj.com/)

Fig. 1.—Relation of P-R interval to occurrence of retrograde (VA) conduction shown by intra-atrial leads during ventricular catheter stimulation. The likelihood of finding VA conduction is directly related to the speed of AV conduction.
FIG. 2.—Relation of sinus rate to occurrence of retrograde conduction. The likelihood of finding VA conduction does not appear to be related to the sinus rates observed in this study.

FIG. 3.—Left ventricular stimulation without retrograde conduction (Case 28, P–R = 0.20 sec., sinus rate 95 beats/min.). Abbreviations in this and subsequent figures: P = sinus beat; P' = retrograde atrial beat; QRS = supraventricular depolarization of ventricle; QRS' = ventricular beats induced by catheter. Time lines 0.20 sec. Ladder diagram: AM = atrial musculature; SP = sinus pacemaker; A–VJ = atrioventricular junction (node); VP = ventricular pacemaker; VM = ventricular musculature. Retrograde conduction is shown with broken lines.

The atria are continuously discharged by the sinus pacemaker which stimulates the ventricles for three beats (A2, A4, A6). Most of the ventricular beats, however, are produced by mechanical stimulation of the left ventricle and none are conducted in retrograde fashion to the atria (V3–V6, V7–V11). The retrograde pathways are never able to conduct the ventricular beats to the atrium; the exact locus of the VA block is not known.
Intra-atrial lead

Lead I

Fig. 4.—Left ventricular stimulation with retrograde conduction (Case 4, P–R = 0·14 sec.; sinus rate = 88 beats/min.). Beats \( A_1 \) and \( A_2 \) are normal sinus beats. \( A_3 \) does not reach the ventricles because of the discharge of ventricular beat \( V_2 \) at about the same instant. Then ventricular beats \( V_4-V_6 \) capture the atria with 1:1 retrograde conduction; the sinus pacemaker is suppressed (Pick, Langendorf, and Katz, 1951).

With cessation of ventricular stimulation, the sinus impulse reappears after a pause \( A_n \) and is conducted in usual fashion to the ventricles \( V_7 \).

longer P–R intervals the refractory period of the conduction system would probably be longer, thus more often precluding retrograde penetration into the atrium by a randomly induced ventricular ectopic beat.

Similarly, in those patients with ventriculo-atrial conduction the P–R and R–P' intervals usually approximated one another within a few hundredths of a second, suggesting that the factors influencing the speed of antegrade conduction affect retrograde conduction in an equal manner. Kistin, Tawakkol, and Massumi (1967) also noted a similar relation between antegrade and retrograde conduction times. In some instances, however, measurement of the total time for retrograde conduction may be subject to error due to the inability to recognize the isoelectric portion of the QRS complex if single leads are used. This difficulty may be overcome through the simultaneous recording of multiple external leads in addition to the oesophageal or intra-atrial leads (Kistin, 1966).

The data further indicate that within the range of sinus rates encountered (65 to 115 beats/min.), there is no correlation between rate and the incidence of retrograde conduction. At faster heart rates, a correlation might have appeared, since less time would be available for a properly timed retrograde impulse to capture the atria, despite the absolute shortening of atrial refractoriness which occurs as heart rate increases (Mendez, Gruhzit, and Moe, 1956).

Similarly, there was no relation between the incidence of ventriculo-atrial conduction and the use of digitalis or the type of heart disease. It is possible, of course, that in a larger series of patients, or in patients with faster heart rates or with selectively (as opposed to randomly) induced ventricular ectopic beats some difference might have emerged. The implication, however, is that whatever influence was exerted by glycosides or myocardial disease similarly affected conduction in both directions.
Animal Studies. In the present work and in the clinical studies mentioned previously, attempts to correlate the presence of retrograde conduction with such factors as speed of forward conduction, drugs, type of heart disease, and coupling intervals have produced variable results. In experimental models such influences have been applied more predictably. Hypothermia has, for example, been shown to speed ventriculo-atrial conduction, which may account for the finding at low temperatures of a reversed form of the usual irregular ventricular response to atrial fibrillation (Francis, Campbell, and Hoff, 1960). When the ventricles fibrillated, an irregular atrial tachycardia developed presumably brought about by retrograde conduction.

The role of vagal stimulation has also been investigated and found to have a greater inhibitory effect on retrograde than on antegrade conduction (Francis and Hoff, 1965). Paradoxically, however, carotid sinus pressure has occasionally facilitated retrograde conduction as observed in human electrocardiograms (Scherf, Cohen, and Rafailzadeh, 1966).

Clinical Significance of Ventriculo-atrial Conduction. The influence of retrograde conduction in clinical situations has been emphasized by several investigators. Suppression of forward conduction by retrograde penetration of the AV tissues is revealed by the presence of “compensatory-like” pauses after ventricular ectopic beats, which occur in patients with atrial fibrillation (Langendorf, Pick, and Katz, 1965b; Langendorf et al., 1965a), and by the prolongation of AV junctional escape intervals after ventricular premature beats (Fig. 7) (Hwang and Langendorf, 1950). The P–R interval can be prolonged through a similar mechanism by an interpolated ventricular beat in sinus rhythm (Katz and Pick, 1956), or by the junctional pacemaker during AV dissociation with ventricular capture beats (Schamroth and Friedberg, 1965; Lister et al., 1967). An inverted P wave need not necessarily be seen in order to infer the presence of retrograde penetration, at least as far as the AV junction (Schamroth and Rosenzweig, 1962; Moe, Abildskov, and Mendez, 1964). Concealed retrograde conduction...
has also been invoked to explain some instances of parasystole with fixed coupling (Langendorf and Pick, 1967).

Conversely, retrograde penetration, it is thought, may facilitate forward conduction through the property of supernormal conductivity. Examples of this phenomenon are found clinically in AV block when relatively normal atrioventricular conduction unexpectedly occurs, apparently produced by effects of the preceding ventricular beat on the AV junction (Lewis and Master, 1924; Pick, Langendorf, and Katz, 1962; Burchell, 1963).

**SUMMARY**

The characteristics of ventriculo-atrial conduction induced by random left ventricular stimulation during cardiac catheterization have been studied by measured intra-atrial electrocardiograms. Retrograde conduction was detected in 14 of 30 patients studied. Ventriculo-atrial conduction was more frequently seen when shorter P–R intervals were present. In those patients with ventriculo-atrial conduction R–P' intervals were less than P–R intervals in 7, equal in 1, and longer in 6 patients. The sinus rate, use of digitalis, or the type of heart pathology did not appear to influence the likelihood of recording ventriculo-atrial transmission.

Ventriculo-atrial conduction in man is clearly not an uncommon event, and techniques are now available for its study. The possible clinical significance of its occurrence has been reviewed.
The authors would like to thank Drs. Charles L. Schulman and Yolando Q. M. Sulit who participated in the collection of the data. The diagrams were prepared by the Medical Art Department of the Massachusetts General Hospital.

REFERENCES
Cohn, A. E., and Fraser, F. R. (1914). The occurrence of auricular contractions in a case of incomplete and complete heart-block due to stimuli received from the contracting ventricles. Heart, 8, 141.


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