Retrograde Conduction from Premature Ventricular Contractions, a Common Occurrence in the Human Heart

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On the basis of studies of esophageal electrocardiograms the authors conclude that retrograde conduction to the atria from premature ventricular beats is common and that the theory of normal unidirectional block in the A-V node is not tenable. In most of the cases with retrograde conduction, the latter is not discernible in simultaneously recorded lead II, which explains why the phenomenon has previously been considered rare. Observations on the ventriculoatrial conduction time and the compensatory pause are presented. In two of the cases there are two orders of magnitude of ventriculoatrial conduction time, which, the authors postulate, may be due to two separate pathways of retrograde conduction.

**Retrograde** conduction to the atria from premature ventricular contractions is generally believed to be rare in the human, although Ashman and Hull state that they have observed this phenomenon repeatedly. We studied the esophageal leads of 33 individuals with premature ventricular beats and believe there is evidence of retrograde conduction in 15. These include cases in which there was no evidence of heart disease as well as cases of heart disease and cases in which the premature beats may have been related to the use of digitalis. The esophageal leads were recorded simultaneously with lead II in 12 of the 15 cases and in most of these the retrograde conduction, apparent in the esophageal leads, is difficult or impossible to recognize in lead II. Little attempt has been made to use this technic in the past. Brown showed that retrograde P waves could be discerned in esophageal leads. Kline, Conn and Rosenbaum used esophageal leads to demonstrate ventriculoatrial conduction in a case of complete atrioventricular block; at one time they could demonstrate the retrograde conduction in the esophageal leads but not in the simultaneously recorded standard leads.

**Technic**

For esophageal exploring electrodes a modified Nyboer tube was used consisting of rings of Monel metal about 5 mm. in diameter and 3 to 5 mm. long mounted on a soft rubber tube. The optimum position of the esophageal exploring electrode was determined in each case by trial, or several esophageal leads were recorded. The positions of the exploring electrodes in centimeters from the nares are designated by the numbers following the E in the figures. The esophageal and precordial exploring electrodes were paired with the Wilson indifferent electrode. A standard recording speed of 25 mm. per second was used so that the smallest time intervals on the illustrations represent 0.04 second. All records were taken with amplifier type instruments. A Technicon three channel electrocardiograph was used for the simultaneous tracings. The width of the record has been reduced for some of the illustrations without altering the alignment. For the records of single leads, the Sanborn Cardilette or Viso-Cardiette was used. An electropositive potential at the precordial and esophageal electrodes was recorded as an upward deflection.

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There was no deliberate selection of cases available for study. As we became interested in the problem, we attempted to record esophageal leads in all cases showing numerous premature ventricular contractions. In this sense our material is a random series from a hospital population. In a few cases esophageal leads had already been recorded for other purposes and incidentally showed premature ventricular contractions. In some cases satisfactory esophageal leads could not be obtained.

Criteria of Retrograde Conduction

In esophageal leads from atrial levels there are large characteristic P waves with a rapid negative intrinsic deflection. Differences in direction of the atrial excitation wave may produce change in contour which may be minimal or not discernible in other leads. Furthermore the sharp positive peak of the esophageal P wave furnishes a precise reference point for timing from one P wave to another, even frequently when the latter are superimposed upon QRS or T.

Fig. 1 (Case 11, table 1). Lead II and esophageal leads. X = premature ventricular contraction. The arrows point to the P waves produced by retrograde conduction. These differ in contour from the sinus P waves and occur prematurely with respect to the expected sinus P wave. (Some of the rapid deflections were retouched for illustration.)

Fig. 2. Esophageal leads (A = E40, B = E42.5). X = premature ventricular contraction. The arrows point to retrograde P waves (P'). These differ in contour from the sinus P waves. In 2A (Case 15) the retrograde P is also premature with respect to the expected sinus P. The P'-P interval is longer than the preceding P-P. In 2B (Case 10) the P following the premature ventricular contraction is not premature and is probably due to fusion of retrograde activation and antegrade atrial activation from the S-A node.

The characteristics of P in the esophageal leads which we observed in these cases and which we interpret as due to retrograde conduction are: (1) it may differ from the sinus P waves in contour; (2) it may be premature with relation to the expected P wave of sinus origin, and in these instances it is followed by an interval to the next sinus P wave which is longer than the usual sinus P-P interval and which may or may not be compensatory; (3) it occurs within a limited range of time following the premature QRS. Not all the criteria are demonstrable in each case. Retrograde P waves superimposed on QRS and T may show a definitely premature peak although the superim-
position makes contour analysis difficult or impossible. In cases of fusion beats, that is, combinations of retrograde activation and anterograde activation from the sinus node, the contour of the P following the premature QRS considerably deeper negative deflection below the baseline than the sinus P waves.

In the esophageal leads the retrograde P need not be inverted or opposite in direction to the P of sinus origin, although the latter may be quite distinctive, yet the peak of the P wave may not be premature.

Contour. Retrograde P waves differing in contour from the sinus P waves are illustrated in figures 1 to 6. In figures 1, 4, and 5, the upstroke of the retrograde P is slower than that of the sinus P. In the 40 cm. lead of figure 1 and in figure 6 the retrograde P has a may occur. The exploring electrode of a “unipolar” esophageal lead properly placed is close to the atrium and therefore a semidirect lead. An electropositive deflection will be recorded whenever the mean direction of the excitation wave is approaching the electrode, regardless of the origin of this approach. The steep negative or “intrinsic” deflection starting from the
potential peak of P represents the peak arrival of the excitation wave beneath the electrode in direct leads from the myocardium, and is believed to approximate the same phenomenon in semidirect leads. Deflection of the retrograde P sometimes may appear to be opposite
tion of P and a later intrinsic deflection will occur.

Fusion P waves are reliable evidence of retrograde conduction. These not only differ distinctly in contour from the sinus P waves (figs. 2B, and 5, upper tracings), but may be

![Image](http://circ.ahajournals.org/)

FIG. 4 (Case 4, table 1, as in fig. 3). The arrows point to the retrograde P waves which differ in contour from the sinus P waves. In the lower tracings the retrograde P waves are also premature with respect to the expected sinus P wave. In the upper tracings the first retrograde P is also premature, but the second retrograde P is slightly “late” on the basis of the immediately preceding P-P interval, and this is explained by the sinus arrhythmia. In lead II of the upper tracings retrograde conduction is clearly indicated by the inverted P, but when the retrograde P occurs sufficiently early after the premature ventricular contraction (last premature complex, lower tracings), there is no evidence of retrograde conduction in lead II. There are reciprocal beats in the upper tracings. In this case there were two discontinuous orders of magnitude of ventriculoatrial conduction times, the shorter times being illustrated in the lower tracings, and the longer times in the upper tracings. See text and figure 7.

in direction to that of the sinus P. For example, an electrode opposite a region of early antegrade activation will tend to register a relatively early intrinsic deflection, a small positive deflection of the P and a large negative deflection (figs. 4 and 5). Since this region may be relatively further away from the A-V node, in retrograde activation a larger positive deflec-

recognized also as intermediate in character between P waves produced by retrograde activation and those produced by antegrade activation (fig. 3). For the recording of fusion atrial beats, lead II and other remote leads seem to be particularly unsuitable, while an esophageal lead may be most advantageous. Two simultaneous excitation waves traveling
in approximately opposite directions toward each other, one from the S-A node and one from the A-V node, should tend to neutralize potential differences between two distant points in such leads. This does not hold for the semi-direct esophageal lead. In fact, if the esophageal electrode is so situated that components of both excitation waves are directed toward it, a tendency to summation of potentials would result.

Prematurity. The retrograde P waves in figures 1, 2A, and 6 occur prematurely with reference to the expected time of occurrence of the regular sinus P wave and interrupt the regularity of the sinus rhythm. Sinus arrhythmia may obscure this relationship (fig. 4). When the retrograde P is premature, the interval to the following sinus P is usually longer than a regular sinus P-P interval (figs. 1, 4 and 6). The evaluation of this interval too may be complicated by sinus arrhythmia.

Retrograde Conduction Time. The interval from the onset of the premature QRS in esophageal leads to the intrinsic deflection of the retrograde P was measurable in all instances except in two, where only fusion P waves were obtained. The interval varied from 0.09 to 0.29 second in all but one of these cases. In this, and in one other case (4 and 8, table 1), which will be discussed separately, there were two discontinuous orders of magnitude of this interval. In the simultaneously recorded E, II and V1 leads the interval from the earliest onset of premature QRS was 0.11 to 0.29 second. This is close to the range of normal atrioventricular conduction time. The variation of the interval for each case is 0.07 second or less in 9 cases, 0.10 second in one and 0.16 second in another. In cases 4 and 8, the range of variation within each of the two orders of magnitude of retrograde conduction was 0.02 to 0.08 second. These limits support the view that the P wave following the premature QRS is related to the latter and not fortuitous, and the simplest explanation of the relationship is retrograde conduction. In the standard leads, the hitherto accepted criteria10, 14, 20 for a P produced by retrograde conduction are its relation to the premature QRS, its prematurity, and its inversion in lead II and sometimes in lead III as well. It is also maintained10, 20 that the interval from the onset of the premature QRS to the onset of the retrograde P should be longer than the P-R interval of the same case. In only 2, possibly 3, of the 15 cases is retrograde conduction recognizable in lead II. The simultaneous records of esophageal leads and lead II prove that retrograde conduction may be apparent in the former and not discernible in the latter. We believe this explains why the frequency of retrograde conduction in the human has not been recognized previously. Examples are illustrated in figures 3, 4, 5, 6. Figure 4 is of particular
note in this connection. The ventriculoatrial conduction time in this case varied considerably, and only when it was long, was retrograde conduction distinctly recognizable in lead II (upper tracings). When the ventriculoatrial conduction time was sufficiently short, the retrograde conduction was not recognizable in lead II, while still distinct in the esophageal lead (last retrograde P wave, lower tracings). The significant thing about figure 4 is that the sometimes had difficulty in deciding whether or not a compensatory pause was present. In non-phasic sinus arrhythmia, a compensatory pause can be determined only within certain limits. Let us consider, for example, one of the cases of this series with a fairly regular rhythm. The P-P intervals immediately preceding the premature contractions varied within 0.07 second, which means that P-P'-P intervals varying within a range of 0.14 second might be con-

![Image](https://placekitten.com/g/300/400)

**Fig. 6** (Case 8, table 1). Simultaneous record of lead II, V₁ and an esophageal lead. The arrows point to the retrograde P waves which differ in contour from the sinus P waves and are slightly premature with respect to the expected sinus P. There are only poorly-defined deflections in II corresponding to the retrograde P waves of the esophageal leads. There were two discontinuous orders of magnitude of ventriculoatrial conduction time in this case—the short time being illustrated following the first premature complex and the longer conduction time following the second and third premature ventricular contractions. See text and figure 8.

tower tracings represent the range of ventriculoatrial conduction time found in 14 of the 15 cases. The ventriculoatrial conduction time in the upper tracings was exceptional in our series and yet it is these tracings that resemble previously reported cases of retrograde conduction.

The "Compensatory Pause"

The compensatory pause is often implied to have an absolute value, and the degree of discrepancy within which one may still speak of a compensatory pause is rarely emphasized. In the measurements of this study, however, we considered to be associated with a compensatory pause. If one arbitrarily allows for an additional variability of 0.01 second at both the upper and lower limits of the P-P interval for errors of measurement, this makes the range of variability of P-P'-P intervals with a compensatory pause equal to 0.18 second. The allowance of such a wide margin in the calculation of the compensatory pause cannot be avoided, and yet it would seem to allow for a number of fortuitous "compensatory" pauses. In the above instance the permissible range of variability of P-P'-P intervals was 20 per cent.
of P-P. If we apply the above analysis, there was a compensatory pause (fig. 1) more often than not (fig. 2A) following the premature ventricular contractions with definite retrograde conduction in this series.

In some instances, the retrograde atrial activation seemed to occur early enough to discharge the sinus node, and yet an apparent compensatory pause occurred. In 4 of the cases with fairly regular sinus rhythm, premature ventricular contractions occurred followed by a compensatory pause when the onset of the intrinsic deflection of the retrograde P was at least 0.14, 0.16, 0.16 and 0.17 second premature.

Though the atrium is activated by the retrograde stimulus, it is possible that the sinus node may still discharge spontaneously before the retrograde stimulus reaches it. This undoubtedly accounts for the compensatory pause in a number of the cases. Since the retrograde atrial contraction follows a premature ventricular contraction its degree of prematurity at ordinary heart rates must be limited—unlike spontaneous premature atrial contractions which may occur much earlier in the cardiac cycle. This limited prematurity of the retrograde stimulus (table 1) makes the spontaneous discharge of the sinus node more likely.

### Table 1.—Summary of Data in Cases of Retrograde Conduction

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Diagnosis</th>
<th>Prematurity of Retrograde P (P-P minus P-P') Seconds X100</th>
<th>B*</th>
<th>R'-I† Seconds X100</th>
<th>P-R‡ Seconds X100</th>
<th>&quot;Compensatory Pause&quot;</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td>A*</td>
<td>Min.</td>
<td>Max.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>No heart disease</td>
<td>03 to 07</td>
<td>02 to 04</td>
<td>03 to 07</td>
<td>24 to 27</td>
<td>19</td>
</tr>
<tr>
<td>2</td>
<td>No heart disease</td>
<td>01 to 17</td>
<td>-10 to 14</td>
<td>02 to 20</td>
<td>16 to 18</td>
<td>17</td>
</tr>
<tr>
<td>3</td>
<td>No heart disease</td>
<td>-01 to 22</td>
<td>-08 to 09</td>
<td>-01 to 22</td>
<td>09 to 19</td>
<td>12</td>
</tr>
<tr>
<td>4§</td>
<td>No heart disease</td>
<td>-13 to 26</td>
<td>-21 to 23</td>
<td>-14 to 26</td>
<td>16 to 24</td>
<td>12</td>
</tr>
<tr>
<td>5</td>
<td>?</td>
<td>01 to 23</td>
<td>0 to 21</td>
<td>01 to 29</td>
<td>12 to 15</td>
<td>15</td>
</tr>
<tr>
<td>6</td>
<td>Hypertension, no evidence</td>
<td>-02 to 11</td>
<td>-02 to 10</td>
<td>0 to 13</td>
<td>22 to 29</td>
<td>16</td>
</tr>
<tr>
<td>7</td>
<td>Hypertensive heart disease</td>
<td>0 to 07</td>
<td>-01 to 07</td>
<td>01 to 07</td>
<td>20 to 25</td>
<td>15</td>
</tr>
<tr>
<td>8§</td>
<td>Coronary heart disease</td>
<td>03 to 17</td>
<td>03 to 14</td>
<td>07 to 18</td>
<td>11 to 14</td>
<td>12</td>
</tr>
<tr>
<td>9</td>
<td>Coronary heart disease</td>
<td>-03 to 01</td>
<td>-03 to 01</td>
<td>-03 to 01</td>
<td>?</td>
<td>18</td>
</tr>
<tr>
<td>10</td>
<td>Coronary heart disease</td>
<td>-01 to 01</td>
<td>-02 to 0</td>
<td>-01 to 01</td>
<td>?</td>
<td>13</td>
</tr>
<tr>
<td>11</td>
<td>Myocardial infarct</td>
<td>05 to 17</td>
<td>03 to 14</td>
<td>07 to 17</td>
<td>17 to 21</td>
<td>14</td>
</tr>
<tr>
<td>12</td>
<td>Myocardial infarct</td>
<td>0 to 20</td>
<td>02 to 17</td>
<td>04 to 19</td>
<td>13 to 20</td>
<td>18</td>
</tr>
<tr>
<td>13</td>
<td>Rheumatic heart disease, digitalis</td>
<td>01 to 16</td>
<td>01 to 14</td>
<td>02 to 16</td>
<td>10 to 16</td>
<td>13</td>
</tr>
<tr>
<td>14</td>
<td>Rheumatic heart disease, digitalis</td>
<td>02 to 07</td>
<td>01 to 26</td>
<td>03 to 28</td>
<td>17 to 26</td>
<td>20</td>
</tr>
<tr>
<td>15</td>
<td>Ventricular aneurysm, etiology undetermined</td>
<td>-04 to 31</td>
<td>-04 to 23</td>
<td>10 to 39</td>
<td>10 to 26</td>
<td>14</td>
</tr>
</tbody>
</table>

* A: Based on the single immediately preceding P-P interval.

B: Based on all available immediately preceding P-P intervals (1 to 10), giving a minimum (Min.) and maximum (Max.) value. Zero and negative values indicate that the retrograde P was recognizable by contour even though not premature.

† From the onset of the premature QRS in the esophageal lead to the onset of the intrinsic deflection (I') of the retrograde P following it, recorded only when the latter was premature with respect to the expected sinus P wave.

‡ The mean of several P-R intervals.

§ To show the division of R'-I’ into two distinct groups in these two cases measurements from a lead from a single esophageal level were recorded. Measurements in other cases sometimes include leads from more than one esophageal level.
respectively, as measured on the basis of the shortest of the P-P intervals immediately preceding the premature contraction. Since the normal duration of the P wave is less than 0.14 second, such excitation would be expected to discharge the sinus node unless some delay in activation occurs at the latter. The P'-P interval following the retrograde atrial activation is somewhat longer than the P-P interval just as it is following spontaneous premature atrial contractions. It is possible that a slightly decreased P-P' interval plus a slightly increased P'-P interval should sometimes add up to approximately twice P-P by chance, even though the sinus node is discharged by retrograde atrial activation.

The Ventriculoatrial Conduction Time

As an index of ventriculoatrial conduction time we measured the interval from the onset of the premature QRS to the onset of the intrinsic deflection of the retrograde P (R'-I' interval). The onset of the retrograde P is frequently impossible to time precisely in the esophageal leads. We use the designation R'-I' rather than R'-P' because the latter has been recorded in the literature as a measurement from the onset of the ectopic QRS to the onset of the retrograde P. The over-all range of R'-I' and the variation in a given case have already been discussed and are summarized in Table 1. In 10 cases the earliest onset of the premature QRS in the simultaneously recorded esophageal lead and lead II (and often V3) was measured, and the minimum R'-I' was equal to or smaller than the P-R immediately preceding it in 5 cases (cases 3, 5, 8, 13, 14, Table 1). The interval exceeded the preceding P-R by 0.01 second in one case, 0.05 and 0.07 second in 2 cases each. The minimum R'-I' is an approach to the ventriculoatrial conduction time least affected by the refractoriness of the conducting tissues. In cases 4 and 8, with two groups of R'-I' intervals (see below), the minimum R'-I' was within 0.03 of P-R in the group of shorter intervals, but exceeded P-R by 0.31 and 0.10 second, respectively, in the group of longer intervals. It has been stated that the R'-P' interval resulting from retrograde conduction is longer than the P-R interval in the same case.20

The P-R and R'-P' intervals are not strictly comparable as an indication of the relative speeds of conduction, since the pathways of antegrade and retrograde conduction are different. Even if it is assumed that the pathways are comparable in length, the two intervals do not necessarily represent relative speeds of conduction under similar conditions, because the conduction from ventricle to atrium occurring early in the cardiac cycle is more apt to encounter refractoriness in the A-V node. For comparison with P-R the same limitations apply, of course, to R'-I' as to R'-P'.

Winternitz and Langendorf22 reviewed the literature and tabulated the R-P' intervals in reported cases of retrograde conduction occurring with incomplete and complete atrioventricular block. Of the twenty-three cases cited by these authors, the R-P' interval was 0.20 second or less in sixteen. This evidence added to our observations in cases where antegrade conduction was not abnormal does not support the thesis that the retrograde ventriculoatrial conduction time in the human is consistently greater than the normal atrioventricular conduction time.

In experimental animals retrograde conduction to the atria from artificially induced premature ventricular contractions has been observed repeatedly.4, 7, 18, 21, 23, 27, 28, 30 Some observers found the ventriculoatrial time slower than the atrioventricular time,4, 27, 28 and others found the reverse.7, 30 Comprehensive summary and evaluation of the experimental data is difficult. The conditions of the experiments of the different observers are not strictly comparable, and sometimes the experimental conditions during ventriculoatrial conduction differ from those during atrioventricular conduction. Furthermore, there may be differences among animal groups; von Skramlik found atrioventricular conduction faster than ventriculoatrial conduction in frogs,29 but the reverse in fish.30 Comparative data of atrioventricular and ventriculoatrial conduction times in mammals are limited. Both Bayliss and Starling4 and Stassen27 found the ventriculoatrial conduction time longer than the atrioventricular time in dogs. The former authors, however, were careful to point out that they made only few observations, and the accuracy of their mechani-
RETROGRADE CONDUCTION

![Graph showing R'-I' seconds vs. P-P seconds and R'-R' seconds vs. P-P seconds]

**Fig. 7** (Case 4, table 1). Relation of ventriculoatrial conduction time (R'-I') to cardiac cycle length (P-P) and to time of occurrence of premature ventricular beat (R-R'). Two discontinuous orders of magnitude of R'-I'.

**Fig. 8** (Case 8, table 1). Relation of ventriculoatrial conduction time (R'-I') to cardiac cycle length (P-P) and to time of occurrence of premature ventricular beat (R-R'). Two discontinuous orders of magnitude of R'-I'.

Because recording methods were limited, the recording of the onset of atrial or ventricular contraction being questionable. Stassen's measurements were made following ventricular standstill induced by vagal stimulation. Scherf and Shookhoff observed that retrograde conduction occurs readily from induced premature ventricular beats in dogs, and ventriculoatrial intervals as short as 0.08 second were observed. In these experiments A-V nodal rhythm had also been produced, however, so that comparison with the atrioventricular conduction time is not possible. Hukuhara and Komita denied that retrograde conduction occurs in dogs.

In one of our cases (fig. 7) in which numerous premature ventricular contractions occurred at different times in the cardiac cycle, ventriculoatrial conduction time increased the earlier the ectopic ventricular beat occurred. This was interpreted to mean that the earlier a premature contraction occurred the more refractory was the A-V node and the slower the retrograde conduction. In the same case there was considerable sinus arrhythmia and the ventriculoatrial conduction time was proportional to the immediately preceding P-P interval. This is similar to the general relationship that exists between heart rate and atrioventricular conduction time, and suggests a vagal influence on retrograde conduction as well as antegrade conduction. In this same case, however, the P-R variation was only 0.01 second and not discernibly related to cycle length. In one other case in which the measurements could be made, there was a suggestion of the inverse relationship between R-R' and R'-I' (group of shorter R'-I' intervals, fig. 8), but the total range of R'-I' is rather narrow here compared to the error of measurement. The relationship between heart rate and R'-I' cannot be evaluated here because of the narrow range of P-P. In the remaining cases there was insufficient variation in heart rate or prematurity or there were too few observations to test these relationships.

**Two Cases with Two Orders of Magnitude of Ventriculoatrial Conduction Time**

The R'-I' intervals fall into two discontinuous groups in the two cases (Nos. 4 and 8, table 1), whose electrocardiograms are illustrated in figures 3, 4, and 6 and the data of which are plotted in figures 7 and 8. This dichotomy strongly suggests two pathways of
retrograde conduction, pathways with either absolute differences in rate of conduction or differences in duration of relative refractoriness. So far as we know this has not previously been described.

Retrograde atrial activation did not follow all premature ventricular contractions in the tracings from which figure 7 was charted. Sometimes a regular sinus P wave occurred before retrograde conduction became effective. The intervals the smaller order of magnitude is more apt to occur. A sinus P wave or an R'-I' interval of the larger order of magnitude follows a premature beat only when the more rapid order of conduction fails, and such failures may be expressed as a proportion of the total number of premature beats in a given range of heart rates. In this case this proportion was found to be distinctly higher at slower heart rates than at fast rates. Exactly what factors asso

Fig. 9 (Case 3, table 1). Simultaneous records of lead II, V1 and an esophageal lead. Two short runs of ventricular tachycardia. In the esophageal lead the first QRS of each run has superimposed on it a sinus P and is not followed by a retrograde P whereas each subsequent QRS is followed by a retrograde P. In II and V1 neither the superimposed sinus P nor the retrograde conduction is recognizable within the ectopic ventricular complexes.

intervals from the premature beats to these regular sinus P waves were consistently intermediate between the two orders of magnitude of R'-I', and none exceeded the group of long R'-I' intervals. That is, if the more rapid retrograde conduction failed to occur a regular sinus P might intervene but if the latter did not occur then the slower retrograde conduction never failed.

In figure 7 it is apparent that at the longest P-P intervals the larger order of magnitude of R'-I' is more apt to occur while at smaller P-P associated with slower heart rate determine this is not apparent. The R-R' interval does not seem to influence the order of magnitude of R'-I' (fig. 7).

Ventricular Tachycardia and Retrograde Conduction

One of the subjects of this study, who had no other evidence of heart disease, manifested frequent brief paroxysms of ventricular tachycardia in addition to frequent isolated premature ventricular contractions. The contour of
the ventricular complexes during tachycardia closely resembled that of the isolated premature beats. In figure 9 are two brief runs of ventricular tachycardia recorded simultaneously in lead II, V1 and an esophageal lead. In the esophageal lead, superimposed upon the first QRS of each run of tachycardia is the regular sinus P wave, resulting in a W-shaped QRS. That the middle prong of the W is a sinus P wave is apparent from the timing of the previous P-P interval. Each subsequent QRS complex is followed by a retrograde P wave. This retrograde conduction is not apparent in either the simultaneously recorded lead II or V1.

Not all the runs of tachycardia followed this pattern, as illustrated in figure 10 from a record obtained within 15 minutes of figure 9. Here a number of consecutive premature ventricular beats are not followed by retrograde conduction.

The differences between figures 9 and 10 are possibly due to slight differences in timing of the sinus P wave with respect to the ectopic beats and/or slight differences in refractoriness of the A-V node to retrograde conduction. The different mechanisms shown in the esophageal leads in figures 9 and 10 are not apparent in leads II or V1.

Retrograde conduction has been considered a possible mechanism of the perpetuation of certain tachycardias after the manner of a circus rhythm. The present study indicates that retrograde conduction is common enough to be considered a possible factor. However, in the case illustrated in figures 9 and 10, the exhibited retrograde atrial activation seems to be an incidental occurrence, since runs of ventricular tachycardia occurred with or without retrograde conduction to the atria. This, of course, does not rule out a circuit through possible A-V nodal or ventricular pathways in this case; it merely excludes the atria as an essential part of the circuit.

The Conditions for Retrograde Conduction

The supposed rarity of retrograde conduction has in the past received two explanations: (1) unidirectional block at the A-V node exists normally, and (2) the relationship between the rate of the human heart and the duration of the refractory period of the atria and node, would only rarely result in atrial response to stimulus from a premature ventricular beat. If our interpretations in this study are correct then the theory of consistent unidirectional block in the human A-V node as a “normal” occurrence is no longer tenable. We would consider the capacity to conduct stimuli in either direction a more “normal” state.

Unidirectional block does occur in A-V dissociation with interference, and while some of these cases occur with digitalis intoxication, rheumatic fever and other abnormal situations, some cases seem to occur in normal hearts. Whatever is the explanation for the unidirectional block in the latter group of cases, its occurrence in exceptional circumstances is not proof of the consistent “normal” existence of unidirectional block in the A-V node. A-V dissociation with interference occurs in “normal”
cases sometimes with pronounced sinus arrhythmia, and possibly intense vagal stimulation is involved in these cases. Experimentally, it is necessary to injure the myocardium in order to produce unidirectional block. Englemann noted a long time ago in frogs that conduction is equally rapid in either direction but that unidirectional block sets in as the animal is dying or as a result of nerve stimulation or toxins. In some cases of incomplete or complete atrioventricular block retrograde conduction to the atria may occur from beats of idioventricular origin. In other words, in these cases there is unidirectional block in the reverse direction, and this is comparable to the unidirectional block produced experimentally as a result of myocardial injury.

The interrelations of the heart rate, the refractory periods, and the time of occurrence in the cardiac cycle of the premature ventricular contraction remain to explain the cases in which retrograde conduction does not occur. In most of the 18 cases in which we did not find retrograde conduction, the reason was quite obvious. In these cases the premature QRS occurred immediately before or after a sinus P so that one might expect the atrium and/or A-V node to be refractory to retrograde activation. In some cases only few of many recorded premature ventricular contractions showed retrograde conduction although long records were obtained, and in some cases records were repeated at different times. In these cases the relationships of the factors under discussion happened to be favorable for these few beats, for example, adequate slowing of the heart rate and sufficient prematurity of the premature ventricular contraction.

Possible Objections to the Interpretations

Two possible objections to our interpretations must be considered: (1) The premature beats we have illustrated may be of nodal origin with aberrant conduction. This cannot be completely ruled out for any so-called ventricular premature beat. However, this series includes only those cases in which the contour of the QRS of the premature contractions differed significantly from that of the regularly occurring QRS complexes. In one case there were premature ventricular contractions from multiple foci with no conventional doubt of their ventricular origin, and contractions from all foci gave rise to retrograde conduction to the atria (fig. 11).

(2) The P waves following the premature ventricular contractions may be fortuitous pre-
mature atrial contractions not due to retrograde conduction. Against this objection, there were at least several premature ventricular contractions with retrograde conduction in each case in which this interpretation was made. In a number of the cases many premature ventricular contractions with retrograde conduction were studied and in some of the subjects the retrograde conduction was demonstrated repeatedly at different times. In only 2 of the 15 cases, which we believe showed retrograde conduction, did long records show premature atrial contractions that were clearly spontaneous and occurred at times other than immediately following the premature QRS complexes. In one of these there was a single complex of this type, and in the second case there were a number. The P waves which we interpreted as retrograde in these two cases were different in contour from the spontaneous premature atrial contractions occurring at other times. It has already been shown that R'-I' in the 15 cases as a whole is not far from the range of normal P-R, and R'-I' in a given case falls within a narrow range. These facts also are against the fortuitous nature of the atrial contractions and favor retrograde conduction.

**Summary and Conclusions**

1. Retrograde conduction to the atria from premature ventricular contractions was demonstrated in 15 of 35 unselected individuals by means of esophageal leads. The large, peaked P waves obtained in esophageal leads from the atrial level are favorable for this study. In 12 of these 15 cases the esophageal leads were recorded simultaneously with lead II, and in most of these cases the retrograde conduction is not recognizable in lead II.

2. The P wave due to retrograde conduction in the esophageal leads (a) may differ in contour from the sinus P wave, (b) may be premature with respect to the expected sinus P wave in which case the interval to the next sinus P wave is longer than a sinus P-P interval, and (c) occurs within a limited range of time following the premature QRS.

3. A compensatory pause may or may not be present with retrograde conduction. Some of the cases with a compensatory pause may be explained by the spontaneous discharge of the sinus node before the retrograde impulse reaches it. The limited degree of prematurity of the retrograde atrial activation following an antecedent premature ventricular contraction favors spontaneous discharge of the sinus node. In some cases with an apparent compensatory pause, however, the retrograde atrial activation was sufficiently premature so that this explanation does not hold, and in these cases the "compensatory pause" is unexplained or fortuitous. The range of variation which enters into the calculation of the compensatory pause is discussed.

4. There is no evidence that the ventriculoatrial conduction time is consistently longer than the atrioventricular conduction time in human beings. Therefore, the human A-V node "normally" conducts in either direction, and there is no evidence for "normal" unidirectional block. The failure of retrograde conduction to occur in some cases may be explained by the interrelationships of heart rate, refractory periods, and time within the cardiac cycle of occurrence of the premature ventricular contraction.

5. In 2 cases in which adequate measurements were possible, the ventriculoatrial conduction time increased the earlier the premature ventricular contraction occurred. This was interpreted to indicate that the earlier a premature contraction occurred, the more refractory was the A-V node. In one of these cases with pronounced sinus arrhythmia, the ventriculoatrial conduction time was also proportional to the cycle length. This suggests a vagal influence on retrograde as well as antegrade conduction.

6. In 2 cases there were two discontinuous orders of magnitude of ventriculoatrial conduction time, and it is postulated that this was due to two separate pathways for retrograde conduction.

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