Further Observations on the Ventricular Echo Phenomenon Elicited in the Human Heart

Is the Atrium Part of the Echo Pathway?

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SUMMARY

In order to answer the question whether the atrium is a link in the pathway of ventricular reciprocation or whether the A-V node itself is the reflection point of the echo, a special stimulation program was followed in six patients in whom ventricular echo beats with stable time relations could be elicited by regular pacing of the right ventricle or by the application of appropriately timed right ventricular premature beats during regular pacing of the right ventricle. In these experiments the atrial septum was stimulated earlier than the expected arrival at the atrium of the retrograde impulse in the course of the echo sequence. The influence of stepwise (steps of 5 msec) advancing of this atrial “preexcitation” on the timing of the ventricular echo beat was studied.

We found that the atrial septum could be preexcited without interfering with the ventricular echo beat time relations over a time interval less than 5 msec in one patient, less than 10 msec in one, less than 20 msec in two, and less than 30 msec in two cases. When the time interval needed for the stimulated atrial impulse to reach the atrionodal margin is taken into account, it seems likely that the atrium participated in the echo pathway in the four cases with minimal atrial preexcitation intervals of 20 msec or less, and that in the two cases with a minimal atrial preexcitation interval of 30 msec the echo reflection point was localized in the upper part of the A-V node.

Additional Indexing Words:

A-V conduction Retrograde A-V conduction Ventricular reciprocation
Longitudinal dissociation of A-V conduction Stimulation experiments

In recent years intracavitary stimulation studies have outlined the conditions favoring the occurrence of the ventricular echo phenomenon in the intact human heart. The outcome of these studies suggested that the mechanism of reciprocation in the human heart could be explained with the concept of longitudinal dissociation in the upper part of the A-V junction. These clinical investigations did not give an answer to the question whether the atrium is a link in the echo circuit, as advocated by Mendez and co-workers, or whether the point of reflection of the echo is located within the A-V node, as proposed by Mignone and Wallace.

In six patients with ventricular reciprocation elicited by appropriately timed stimulation of the ventricles, we tried to obtain an answer to this question by stimulation of the atrium earlier than the excitation of the atrium expected in the course of the echo sequence. A stimulation program analogous to that used by Mendez and co-workers on the dog heart was performed. As is indicated schematically in figure 1, one would expect, in the case of the atrium serving as a link in the echo path, that “preexcitation” of the atrium (A_s), even over a very short time interval, would result in a
change of the echo beat interval (V-V*), the direction of this change (shortening or prolongation) depending on the functional state of the antegrade pathway at the time of arrival of A8. On the other hand, if the point of reflection is located within the A-V junction, preexcitation of the atrium over a time interval t, equal to the sum of the conduction times from reflection point to atrium in retrograde direction and from atrium to reflection point in antegrade direction, indicated by the broken line in figure 1, would have no influence on the echo beat time relations.

The results of these experiments show that participation of part of the atria in the echo circuit was very likely in four of the six patients studied.

Methods

Out of a total of 13 patients in whom the ventricular echo phenomenon could be elicited, six had echo beat time relations stable enough (constant time relations over a period longer than 4 minutes) to make further studies possible. The clinical data of these six patients are summarized in table 1. Three patients had sinus bradycardia or SA block (cases 1, 2, and 3). Three had a normal QRS configuration (cases 2, 5, and 6), one patient had a complete right bundle-branch block and ASD II (case 3), one had a complete right bundle-branch block with left anterior hemiblock (case 1), and one a complete right bundle-branch block with left posterior hemiblock (case 4). Case 6 had a hypersensitive carotid sinus manifested by extreme sinus bradycardia and block of A-V conduction during carotid sinus pressure. This patient had a slightly prolonged P-R interval (205 msec, as measured in the intracavitary leads) during sinus rhythm. The other five patients had a normal P-R interval.

In all experiments leads I, II, III, VI, and V6 were recorded on an eight-channel Elema Mingograph direct-writing recorder simultaneously with an intracavitary lead from a unipolar catheter located high in the right atrium and a His bundle electrogram obtained from a tripolar catheter (electrode distance 10 mm) advanced through the tricuspid orifice. The recordings were stored on tape with an Ampex FR 1300 tape recorder. The stimulus artifact in stimulated beats or the first visible deflection in any of the leads in nonstimulated beats was used as a reference in the measurements. The stimulus described earlier was used. Prior to the study of ventriculoatrial conduction and ventricular reciprocation, the characteristics of antegrade atrioventricular conduction were defined in all six patients with right atrial septal stimulation through a bipolar catheter with electrode distance 10 mm. Stimulus strength was two times diastolic threshold, and stimulus duration 2 msec.

The functional and effective refractory periods of the A-V junction were estimated with atrial premature stimulation during regular atrial pacing. The influence of increases in atrial driving rate on A-H and H-V conduction times was measured.

Ventriculoatrial conduction was studied with a bipolar stimulation catheter (electrode distance 10 mm) located in the right ventricular apex. All patients had 1:1 ventriculoatrial conduction at the rate studied.

The ventricular echo phenomenon could be elicited by the application of one ventricular premature stimulus after eight beats of a regularly driven ventricular rhythm in four patients (cases
**Table 1**

**Clinical Data and Antegrade A-V Conduction Findings**

<table>
<thead>
<tr>
<th>Case</th>
<th>Sex</th>
<th>Age</th>
<th>Clinical diagnosis</th>
<th>Sinus rhythm Rate (A-V)</th>
<th>Max atrial rate with 1:1 A-H conduction Rate (A-V)</th>
<th>FRP A-V node</th>
<th>ERP A-V node</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M</td>
<td>57</td>
<td>S-A block, complete RBBB</td>
<td>60</td>
<td>80</td>
<td>450</td>
<td>290</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>+ LAH</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>73</td>
<td>Sinus bradycardia, normal QRS</td>
<td>46</td>
<td>60</td>
<td>410</td>
<td>&lt;280</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>65</td>
<td>Sinus bradycardia, complete RBBB,</td>
<td>50</td>
<td>70</td>
<td>445</td>
<td>&lt;260</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>ASD II</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>70</td>
<td>Complete RBBB + LPH</td>
<td>85</td>
<td>100</td>
<td>345</td>
<td>&lt;185</td>
</tr>
<tr>
<td></td>
<td></td>
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</tr>
<tr>
<td>5</td>
<td>F</td>
<td>25</td>
<td>Functional murmur, no ECG or</td>
<td>65</td>
<td>65</td>
<td>435</td>
<td>&lt;275</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>hemodynamic abnormalities</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>M</td>
<td>64</td>
<td>Hypertensive carotid sinus, normal QRS</td>
<td>65</td>
<td>80</td>
<td>430</td>
<td>290</td>
</tr>
</tbody>
</table>

Abbreviations: RBBB = right bundle-branch block; LAH = left anterior hemiblock; LPH = left posterior hemiblock; ASD = atrial septal defect; A-V = A-V interval; H-V = H-V interval; FRP A-V node = functional refractory period A-V node; ERP A-V node = effective refractory period A-V node. Intervals given in msec.

1, 3, 4, and 5). Stimulation was bipolar, and stimulus strength (two times diastolic threshold) and duration (2 msec) were the same for the premature stimuli and those of the basic rhythm.

In the two other patients regular ventricular driving with a rate just above the spontaneous sinus rate resulted in ventricular reciprocation. In one (case 2), the time relations of the ventricular echo beat occurring after each driven ventricular beat were stable enough to allow further studies. In the other (case 6), interference with sinus activity necessitated a stimulation program in which right atrium and ventricle were stimulated synchronously. A premature stimulus was applied only to the ventricle after each eighth beat of the regular synchronous rhythm, giving rise to a ventricular echo beat.

Preexcitation of the atrium (i.e., stimulation of the atrium earlier than the expected time of activation of the atrium in the course of the ventricular reciprocation) was performed by the application of a premature stimulus A₂ by way of a bipolar catheter (with electrode distance 10 mm) located with its tip against the middle of the right atrial septum, using the ostium of the coronary sinus and the limbus of the oval fossa as landmarks. The atrial premature stimulus A₂ was advanced with steps of 5 msec. After each ventricular echo sequence with atrial preexcitation, stability of time relations was always checked by eliciting a ventricular echo sequence without atrial preexcitation. The influence of the atrial preexcitation A₂ on the echo beat interval was measured (interval between ventricular premature beat V₂ and ventricular echo beat V* in cases 1, 3, 4, 5, and 6, or between the ventricular beat V of the regular driven rhythm and the ventricular echo beat V* in case 2).

### Results

#### Antegrade A-V Conduction

**Spontaneous Rhythm**

A slightly prolonged A-V interval was seen only in case 6 (see table 1). The other five patients had normal A-V intervals, even those with a prolonged H-V interval (cases 1 and 4).

**Increases of Atrial Drivng Rate**

A normal response of A-H conduction (A-H Wenckebach occurring at rates higher than
120) was seen in cases 1 to 5. In case 6 A-H Wenckebach occurred at rate 120. Case 4 showed 1:1 A-H conduction even at rate 190; at rates higher than 120, however, a second-degree H-V block (Mobitz II, 2:1, or 3:1 H-V block) was seen.

**Refractory Periods of A-V Junctional Tissue between Atrium and His Bundle**

As shown in table 1, the functional refractory period of the A-V junction (defined as the shortest H1-H2 interval possible), the effective refractory period to the A-V junction (defined as the longest A1-A2 interval possible with no A-H conduction of A2), and the influence of changes of basic rate on these variables were normal in cases 1 to 5. In case 6 both refractory periods were prolonged.

**Studies of Retrograde V-A Conduction and Ventricular Echo Phenomenon**

**Case 1**

At right ventricular driving rates lower than 90 per minute, 1:1 V-A conduction was seen; at higher rates a V-A Wenckebach phenomenon, interference by sinus node activity, or both, occurred. During driving with cycle length 900 msec, a ventricular echo phenomenon with stable time relations could be produced by application of a right ventricular premature beat V2 740 msec after the last (V1) of eight beats of the basic rhythm (fig. 2B). Stimulation of the right side of the atrial septum (A8) 10 msec earlier than the expected first deflection of the atrial activation A2 (conducted retrogradely from V2) resulted in a ventricular activation V8 with a V2-V8 interval 10 msec shorter than the echo beat interval V2-V*, 610 and 620 msec, respectively (fig. 2C).

**Case 2**

At a right ventricular driving rate of 50 per minute, every ventricular beat was followed by a ventricular echo sequence with V-A interval 410 msec and A-V* interval 210 msec (fig. 3A). "Preexcitation" of the right atrial septum with 20 msec (V-A8 390) gave rise to a ventricular beat V8 occurring 10 msec earlier than the expected ventricular echo beat V* (V-V* 620 msec and V-V8 610 msec), as shown in figure 3A and B. Further shortening of V-A8 was accompanied by a corresponding shortening of V-V8, until at a V-A8 interval of 300 msec A8 was not conducted to the His bundle.

**Figure 2**

Case 1. (A) Sinus rhythm (P-P interval about 1,000 msec). Complete right bundle-branch block with left anterior hemiblock. The H-V interval is prolonged (70 msec). (B) Ventricular echo phenomenon occurring after right ventricular premature beat V2 (induced 1:8 during regular driving of right ventricle with basic cycle length [BCL] 900 msec); V1V2 740 msec. Note slowing of retrograde conduction of V2 (V2A2 355 msec, V1A2 270 msec). Echo beat interval V2V* 620 msec. (C) Preexcitation of the right atrial septum with 10 msec (V4A8 345 msec) results in shortening of the echo beat interval with 10 msec (V2V8 610 msec). Since the retrograde activation time of the His bundle after V2 is unknown (His complex hidden in ventricular complex V8), retrograde conduction of V2 to the atrium is represented by a broken line in the diagram.
phenomenon was related to a sudden prolongation of the \( V_2-A_2 \) interval on shortening the \( V_1-V_2 \) interval from 575 to 565 msec (\( V_2-A_2 \) 255 msec at \( V_1-V_2 \) 575 msec; \( V_2-A_2 \) 605 msec at \( V_1-V_2 \) 565 msec).

The echo beat sequence taking place at \( V_1-V_2 \) 500 msec was studied. At this \( V_1-V_2 \) interval, \( V_2-A_2 \) was 655 msec (considerably prolonged as compared with \( V_1-A_1 \) of 245 msec). \( A_2 \) (negative in leads II and III) was followed at 70 msec by a His complex \( H^* \) and at 115 msec by the ventricular echo beat \( V^* \) having the same QRS configuration and H-V interval (45 msec) as the ventricular beats of the sinus rhythm. Preexcitation of the atrial septum with 30 msec (\( V_2-A_8 \) 625 msec) resulted in a ventricular complex \( V_8 \) occurring 10 msec earlier than the expected ventricular echo beat \( V^* \) (\( V_2-V^* \) 770 msec, \( V_2-V_8 \) 760 msec) (fig. 4). Further shortening of \( V_2-A_8 \) resulted in further shortening of \( V_2-V_8 \).

**Case 4**

In this case with complete right bundle-branch block and left posterior hemiblock, showing varying degrees of antegrade H-V block at atrial driving rates of 120 and higher, 1:1 V-H conduction was seen even at ventricular driving rate 160. The retrograde His potential could be identified occurring 115 msec after the beginning of the ventricular complex. On increasing the ventricular driving rate, the V-A interval rose from 220 msec at rate 100 to 315 msec at rate 140, the V-H interval remaining constant. At rate 150 and 160, an H-A Wenckebach phenomenon was found.

During driving with rate 130 (cycle length 463 msec), ventricular echo beats were seen after ventricular premature beats \( V_2 \) in the \( V_1-V_2 \) range 330 to 310 msec. The \( A_1-A_2 \) intervals in this \( V_1-V_2 \) range were not longer than at longer \( V_1-V_2 \) intervals; there was no sudden increase in the \( V_2-A_2 \) interval, as in case 3. The time relations of the occurrence of the echo phenomenon in this patient were therefore of the type described in patient B in an earlier paper.\(^1\)

At a \( V_1-V_2 \) interval of 320 msec, the echo beat interval \( V_2-V^* \) was 570 msec (fig. 4). Preexcitation of the atrial septum by only 5 msec (\( V_2-A_2 \) 335 msec, \( V_2-A_8 \) 330 msec) gave rise to a ventricular beat \( V_8 \) 25 msec later than the expected ventricular echo beat \( V^* \) (\( V_2-V^* \) 570 msec, \( V_2-V_8 \) 595 msec). Shortening of the \( V_2-A_8 \) interval by 10 msec (\( V_2-A_8 \) 320 msec) was accompanied by a further 10-msec prolongation of the \( V_2-V_8 \) interval (605 msec). An atrial excitation induced 45 msec earlier than the expected \( A_2 \) (\( V_2-A_8 \) 290 msec) was not conducted through the A-V junction.

**Case 5**

A 1:1 V-A conduction was seen at ventricular driving rates 80 to 100/min. Higher driving rates were accompanied by a V-A Wenckebach phenomenon. During right ventricular

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driving with cycle length 950 msec, ventricular echo beats V* were found after ventricular premature beats V2 in the V1-V2 range 500 to 395 msec.

As in case 4, a sudden increase of the V2-A2 interval on shortening the V1-V2 interval did not seem to be a necessary prerequisite for the occurrence of the reciprocating mechanism. The ventricular echo sequence found at V1-V2 450 was studied.

Preexcitation of the atrial septum with 20 msec (V2-A2 330, V2-A8 310 msec) shifted the ventricular activation V5 to a time 30 msec later than the expected echo beat V* (V2-V* 535, V2-V5 565 msec) (fig. 5B and C). When the atrial septum was activated 5 msec earlier (V2-A8 305 msec), the atrial impulse was blocked in the A-V junctional tissue (fig. 5D).

**Case 6**
Spontaneous ventricular reciprocation was found at right ventricular driving rates 100
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however, was therefore driven synchronously. After each beat of this basic rhythm (V1), a stimulus V2 was applied only to the right ventricle. At a basic rhythm with cycle length 550 msec, ventricular reciprocation was found at V1-V2 intervals 550 to 220 msec (refractory period ventricle 210 msec). The echo sequence occurring at V1-V2 500 was studied (fig. 4). Ventricular activation V8 was postponed to 20 msec after the expected ventricular echo V2-V* 535, V2-V8 555 msec) when the atrium was preexcited 30 msec (V2-A2 330, V2-A8 300 msec).

Preexcitation of the atrium with 40 msec V2-A8 290 msec) resulted in block of the atrial impulse in the A-V junction.

Conclusion

In conclusion, in two of the six patients preexcitation of the atrium without interfering with the time relations of the echo sequence was possible only over a period shorter than 30 msec, in two patients over a period less than 20 msec, and in two over an interval less than 10 msec (table 2). In three patients atrial preexcitation resulted in ventricular activation earlier than the expected ventricular echo beat; in the other three patients atrial preexcitation led to postponement of the ventricular response.

Discussion

Longitudinal dissociation in the A-V junction, as proposed by Scherf and Shookhoff,6 Moe and co-workers,8 and by Rosenblueth,7 has been accepted as an explanation for reciprocal beating. In the concept of Mendez and Moe two functionally different pathways (α and β) in the upper part of the A-V node are connected on one side with the atrium and on the other with a final common pathway which merges with the His bundle. By the differences in functional properties of these two pathways, the possibility arises that a critically timed ventricular or atrial excitation invades only one of the two pathways leaving the other open for reentry from its atrial end (in the case of the ventricular excitation), leading to a ventricular echo beat, or from the other end (in the case of the atrial excitation), leading to an atrial echo beat.

The atrium is believed by Mendez and Moe8,12,13 to be a necessary link in the ventricular echo pathway, as was postulated by Rosenblueth.7 In their experiments on dog hearts, Mendez and co-workers8 preexcited

Figure 5

Case 5. (A) Sinus rhythm with P-P about 1,150 msec. Normal AH, HV, and AV intervals. (B) Ventricular echo phenomenon elicited by right ventricular premature beat V2 during regular driving of the right ventricle with BCL 950 msec, V1V2 450 msec. Echo beat interval V2-V* 535 msec, V2-A2 330 msec. (C) Preexcitation of the right atrial septum with 20 msec (V2-A8 310 msec) induces a ventricular activation V8 occurring 30 msec later than the ventricular echo beat (V2V8 565 msec). (D) An atrial impulse 5 msec earlier than in strip C (V2 A8 305 msec) is blocked in the A-V node.

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### Table 2

**Minimal Atrial Preexcitation Intervals**

<table>
<thead>
<tr>
<th>Case</th>
<th>Method</th>
<th>Basic cycle length</th>
<th>V2 interval of basic rhythm</th>
<th>V1-V2 zone with echo phenomenon</th>
<th>V1-V2 interval studied</th>
<th>Minimal atrial preexcitation interval†</th>
<th>Resulting V1-V2 minus V2-V*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>V driving†</td>
<td>900</td>
<td>270</td>
<td>—</td>
<td>740</td>
<td>10</td>
<td>-10</td>
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<tr>
<td></td>
<td>V2 1:8</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>V driving</td>
<td>1,000</td>
<td>410</td>
<td>1:1 ventricular echo phenomenon</td>
<td>—</td>
<td>20</td>
<td>-10</td>
</tr>
<tr>
<td></td>
<td>V2 1:8</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>V driving</td>
<td>750</td>
<td>245</td>
<td>565 to 360</td>
<td>500</td>
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<td>-10</td>
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<tr>
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<td>V2 1:8</td>
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<tr>
<td>4</td>
<td>V driving</td>
<td>463</td>
<td>295</td>
<td>330 to 310</td>
<td>320</td>
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<td>+25</td>
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<tr>
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<td>V2 1:8</td>
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</tr>
<tr>
<td>5</td>
<td>V driving</td>
<td>950</td>
<td>220</td>
<td>550 to 395</td>
<td>450</td>
<td>20</td>
<td>+30</td>
</tr>
<tr>
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<td>V2 1:8</td>
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<tr>
<td>6</td>
<td>V-A driving§</td>
<td>550</td>
<td>340</td>
<td>550 to 220</td>
<td>500</td>
<td>30</td>
<td>+20</td>
</tr>
<tr>
<td></td>
<td>(V1-V2 550)</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

All values given in msec.

†Minimal atrial preexcitation interval interfering with the echo sequence time relations.

‡Right ventricular driving, test stimulus V2 1:8 beats of the basic rhythm.

§Synchronous driving of right ventricle and atrial septum.

The atrium in the ventricular echo sequence by driving and premature stimulation of the His bundle. They found that stimulation of the atrial septum as closely as possible to the A-V node, even a few milliseconds before the expected arrival of the retrogradely conducted impulse, resulted in a ventricular response in advance of the expected echo beat or even prevented the echo response. In microelectrode studies on the rabbit heart, Mendez and Moe\(^\text{12}\) never found units of the return pathway discharging earlier than the atrium during ventricular reciprocation.

On the other hand, Mignone and Wallace\(^\text{8}\) could preexcite the atrium in the dog heart 10 to 30 msec without influencing the time relations of the echo sequence. These authors, however, used the atrial complex in the bipolar His bundle lead for their measurements. Since this lead gives no exact information about the time of arrival of the impulse at the atrial margin of the A-V node, additional mapping of the atrial area around the A-V node during antegrade and retrograde conduction was necessary. This was done in only three of the nine dogs studied and not simultaneously with the actual echo studies. Small errors might therefore have been introduced.

As Moe and Mendez\(^\text{13}\) have pointed out, the fact that the atrium can be preexcited without altering the echo beat interval (V2-V*) does not necessarily prove the existence of a subatral communication unless it can be established that the ventricular response is indeed an echo and not a response to the premature atrial excitation propagated with a "compensatory" delay depending on the functional state of the final common pathway. One has to keep in mind that the models given in figure 1, based on investigations in the rabbit and dog heart, do not literally represent an anatomic substrate. The three-dimensional structure of the atrial nodal margin is complex and different in the rabbit, dog, and human hearts. The concept of temporal dissociation of conduction within the A-V junction, as represented in the models of figure 1, is functional and not necessarily affected by structural differences. The atrial septum was chosen as the atrial stimulation site in order to apply the "preexcitatory" stimulus as near as possible to the A-V node.

One might argue that the direction in which the atrial wavefront approaches the atrionodal margin might have influence on the conduction of the impulse within the A-V node, as was demonstrated in the isolated
rabit heart by Janse, who observed differences in effectiveness of impulse transmission depending on the site of input into the A-V node. If this were true for the human heart, antegrade conduction within the A-V node might be different for the atrial activation occurring in the course of the echo phenomenon and for the atrial activation caused by stimulation of the atrial septum. Obviously, this problem is very hard to solve in the intact human heart and in this specific experimental setting. However, studies in a different group of 16 patients yielded results suggesting that in the human heart the direction of the atrial wavefront has less influence on A-V nodal conduction than in the rabbit heart. In these cases the A-V conduction of atrial activations from several stimulation sites in the right and left atrium, as judged from the effect of increases in driving rate and application of atrial premature beats with gradually shortening coupling interval, showed no significant difference, even when A-V conduction was critical (high driving rate, short coupling interval). The functional and effective refractory periods of the A-V junction were also the same for different stimulation sites in the atria. Moreover, we recently studied a patient in whom the atrial echo beat phenomenon (also thought to be an expression of longitudinal dissociation in the upper part of the A-V node) could be elicited by appropriately timed atrial premature beats during regular driving of the atrium. The premature beat interval range in which the echo beat phenomenon was observed in this patient and the time relations of the echo beat itself were the same, whether the middle of the atrial septum or the right posterior side of the free wall of the right atrium was stimulated.

In the experiments of this communication, we found that the atrial septum could be preexcited without interfering with the echo beat sequence less than 5 msec in one case, less than 10 msec in one, less than 20 msec in two, and less than 30 msec in two cases. The stimulation site in the atrial septum for obvious reasons was not identical to the part of the atrium first activated in the course of the echo sequence. Therefore, in order to obtain the actual preexcitation times, the time needed by the impulse to travel from the stimulation site in the atrial septum to the atrial margin of the A-V node has to be subtracted from the values given above. This time could not be measured. It depends on the distance between the stimulating electrodes and the A-V nodal margin and on propagation velocity. The handicap of all stimulation studies during heart catheterization is that the location of intracavitary electrodes cannot be defined exactly. While electrode position was stable in every individual patient, the distance from electrodes to A-V node certainly was not the same in all six patients and might have varied from a few millimeters to as much as 2 cm. Atrial stimulation was performed by way of a bipolar catheter, and stimulus strength was not greater than two times diastolic threshold. It therefore seems to be improbable that the stimulated area of the atrial septum was very large, which fact could have influenced the time interval between stimulus and moment of arrival of the atrial impulse at the A-V node margin.

Conduction velocity in atrial muscle has been found in the isolated normal human heart to be about 1 m/sec. This value is consonant with our findings during heart catheterization using unipolar leads from a multipolar catheter positioned against the atrial septum. Travel time from electrodes to A-V node, therefore, might have varied in these six patients from a few milliseconds to as much as 20 msec.

In the four cases in which the atrial septum could be preexcited less than 20 msec without interference with the echo time relations, atrial participation in the echo pathway seems to be very likely. In the other two cases with a minimal atrial preexcitation time of 30 msec, the probability is higher that the reflection point was localized within the A-V node. Considering that the preexcitation time then comprises the conduction times from reflection point to atrium, from atrial electrodes to atrial margin of the A-V node, and from atrial margin of the A-V node to
reflection point, one might draw the conclusion that, if the reflection point is localized within the A-V node, it must be very near the atrial margin. The probability that the atrium was indeed the reflection point is higher when atrial conduction velocity was lower than 1 m/sec, which value has been found in normal hearts. The fact that three of the six patients had abnormal sinus node function may be of some importance in these considerations.

The results of this study indicate that the answer to the question posed in the title, whether the ventricular echo pathway comprises atrial tissue or is confined to the A-V node, must be that both possibilities can be found in the human heart.

References


Further Observations on the Ventricular Echo Phenomenon Elicited in the Human Heart: Is the Atrium Part of the Echo Pathway?
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