Ventricular Echo Beats in the Human Heart Elicited by Induced Ventricular Premature Beats

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SUMMARY
In three patients, all having as their only hemodynamic abnormality an elevated cardiac index at rest, ventricular echo beats could be elicited by ventricular premature beats induced during regular driving of the ventricle. The occurrence of ventricular echo beats was in each case limited to a small delay-range of the premature beat. Retardation of retrograde conduction of the premature ventricular impulse to the atria appeared to be a necessary condition for the initiation of the echo phenomenon.

One of the patients had retardation of antegrade A-V conduction of sinus and premature atrial beats. Atrial echo beats could be elicited in this case.

The genesis of the ventricular echo phenomenon in these cases could be explained by the dual conducting system theory.

Additional Indexing Words:
Ventricular reciprocation Ventricular return extrasystoles Reciprocal beats
Longitudinal dissociation of A-V conduction system Stimulation experiments
Retrograde A-V conduction

Ventricular echo beats frequently described in clinical literature are recognized when a characteristic sequence is found: a ventricular or an A-V nodal beat with a retrograde P wave is followed after a short interval by a QRS complex with a configuration suggesting supraventricular origin. Ventricular echo beats are seen most frequently in A-V nodal rhythm or atrionodal dissociation. Digitalis intoxication seems to be a provoking factor.

During studies on atrioventricular conduction we could produce ventricular echo beats by inducing right ventricular premature beats during regular driving of the ventricles in three patients and define the time relations of the occurrence of this phenomenon in each case.

Methods
Ventriculo-atrial conduction was studied in three patients after completion of diagnostic heart catheterization. Stimulation of the ventricles was possible after introducing a bipolar electrode catheter in the right ventricle. The specially designed stimulator described in earlier papers was used. Stimulus strength was two times threshold level; stimulus duration was 2 msec. The ventricles were driven regularly. After each eighth stimulus of this regular basic rhythm an extra stimulus with the same duration and current strength was given. The interval between this extra stimulus (V) and the last (eighth) stimulus of the basic (V) was gradually shortened. Leads I, II, III, and an intra-atrial lead obtained from a unipolar electrode catheter located high in or in the middle of the right atrium were recorded on an 8-channel Elema Mingograph recorder and stored on tape with an Ampex FR 1300 tape recorder.

Report of Cases
Patient A
This 26-year-old man had a systolic heart
murmur, which seemed to be innocent. Chest x-ray examination revealed no abnormalities. The electrocardiogram was completely normal. During right heart catheterization normal pressures were found. There was no left-to-right shunt. The cardiac index at rest was elevated: 5.5 L/min/m² (cardiac output, 10.4 L/min). Because the spontaneous sinus rate was above 100/min, the ventricles had to be driven at a relatively high rate to obviate interference with sinus impulses.

During driving at a basic rate of 130/min (basic cycle length, 460 msec) a ventricular echo

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**Figure 1**

*Patient A. Effect of shortening of the V₁V₂ interval on retrograde V-A conduction of premature beat V₂. Driving of the right ventricle with basic cycle length of 460 msec. Normal V-A conduction in V₁V₂ intervals down to 375 msec (Q-P interval, 200 msec). Sudden prolongation of V-A conduction time at V₁V₂ interval between 375 and 360 msec (Q-P interval, 410 msec). A₂ is then followed at 130 msec by a ventricular echo beat V* with normal QRS configuration. No V-A conduction is seen at the V₁V₂ interval of 358 msec.*
phenomenon could be elicited. The ventricular complexes of the basic rhythm were followed by inverted P waves in leads II and III (see fig. 2) with a Q-P interval of 200 msec, which was measured from the stimulus artifact to the beginning of the P wave in the intra-atrial lead.

Ventricular premature beats $V_2$, with $V_1V_2$ intervals between 460 and 375 msec, showed no increase of the Q-P interval (fig. 1). In the $V_1V_2$ interval range of 375 to 360 msec a sudden increase of the V-A conduction time was seen. The Q-P interval in this $V_1V_2$ interval range was 410 msec. The P waves ($A_2$) were inverted in leads II and III and were invariably followed 130 msec after their beginning by a QRS complex $V^*$ with a configuration identical to that found in sinus rhythm (figs. 1 and 2). This beat clearly was a ventricular echo beat. No V-A conduction was found at $V_1V_2$ intervals of less than 360 msec. The refractory period of the ventricle was reached at a $V_1V_2$ interval of 220 msec.

**Patient B**

In this 30-year-old woman diagnostic right heart catheterization was performed. She had a systolic heart murmur which seemed to be innocent. Chest x-ray examination and electrocardiograms were normal. Right heart pressure was normal. There were no signs of shunts. The cardiac index was rather high: 4.9 L/min/m$^2$ (cardiac output, 8.4 L/min).

The right ventricle was driven at a rate of 100/min (basic cycle length, 600 msec). The Q-P interval (stimulus artifact to beginning of P wave in intra-atrial lead) of the ventricular complexes of the basic rhythm was 230 msec.

Ventricular premature beats $V_2$ showed a gradually increasing Q-P interval in the $V_1V_2$ interval range of 600 to 470 msec. At a $V_1V_2$ interval of 470 msec the Q-P interval was 275 msec. At this and shorter $V_1V_2$ intervals down to 440 msec the atrial depolarization $A_2$ caused by the ventricular premature beat $V_2$ was followed...
Patient B. Effect of shortening of the V1V2 interval on retrograde V-A conduction of premature beat V*. Driving of the right ventricle with a basic cycle length of 600 msec. Gradually progressive retardation of V-A conduction of V* is to be seen as shortening of the V1V2 interval. At the V1V2 interval of 470 msec the V2A2 interval is 275 msec. A2 is followed at 160 msec by a ventricular echo beat V*. The same sequence is seen when V1V2 interval is 450 msec. Slowing of retrograde conduction of V* is followed by acceleration of antegrade conduction of A2. No V-A conduction is observed at V1V2 interval of 440 msec. In this line the first P wave occurring after V* is a sinus P wave.

by a QRS complex, V*, identical to that seen in normal sinus rhythm. The P-Q interval of this beat, V*, was 160 msec (figs. 3 and 4). At a V1V2 interval of 450 msec, a somewhat shorter A2V* interval (100 msec) was seen once after a somewhat longer V2A2 interval (305 msec). The negative polarity of the P wave A2 in lead II is clearly seen in this sequence (fig. 3). At V1V2 intervals of 440 msec or less no retrograde conduction to the atria took place.

The refractory period of the ventricle was reached at a V1V2 interval of 220 msec.

Patient C

This 22-year-old man was treated for hyperthyroidism. This diagnosis was established by the finding of an elevation of the basic metabolic rate, the plasma protein-bound iodine, and the rate of uptake of 131I in the thyroid gland. Before treatment the ECG showed a first degree A-V block (P-Q interval of 0.28 sec at a rate of 100/min).

On a routine ECG made 6 years earlier, when no clinical signs of hyperthyroidism were found, a first degree A-V block was present. There were no further electrocardiographic abnormalities. On auscultation a grade II/VI systolic ejection murmur was heard along the left sternal border and at the apex. A loud third heart sound was present at the apex. Chest x-ray examination was normal. After 8 months of treatment with carbimazole the basic metabolic rate and the plasma protein-bound iodine became normal. At that time the ECG still showed a prolonged P-Q interval (0.26 sec at a rate of 68/min). On exercise (cycle ergometer, 100 watts) the P-Q interval shortened to 0.16 sec at a rate of 150/min. The auscultatory findings of the heart were unchanged. To exclude organic heart
ECHO elevated was cardiac disease venous and arterial heart catheterizations were performed. Normal pressures were found in the right and left heart. There were no signs of cardiac shunts. A left ventricular cineangiogram showed no abnormalities. The cardiac index was elevated 6.1 L/min/m² (cardiac output, 10.3 L/min). After completion of the hemodynamic study, antegrade and retrograde atrioventricular conduction were studied.

During driving of the right atrium 1:1 A-V conduction was found at rates up to 160/min. Increase in rate caused a progressive lengthening of the P-Q interval (at a rate of 100/min it was 260 msec; at a rate of 160/min, 365 msec).

At rate 170/min a Wenckebach phenomenon was observed; at rate 190, a 2:1 A-V block.

Atrial premature beats, A₂, induced during regular driving of the atrium with a rate of 100/min, showed progressive retardation of A-V conduction, when the interval (A₁A₂) after the last atrial beat (A₁) of the basic rhythm was gradually decreased. Figure 5 is a diagram in which the A₁A₂ intervals and the corresponding V₁V₂ intervals are plotted. Retardation of A-V conduction was most marked in the A₁A₂ interval range of 275 to 230 msec. Atrial premature beats with A₁A₂ intervals between 250 and 230 msec gave rise to ventricular activations, V₂ (open circles), which were followed at 60 msec after their beginning by an atrial echo beat.

The refractory period of the atrium was reached at a delay of 225 msec. Atrial premature beats with an A₁A₂ interval a few milliseconds longer were still conducted to the ventricles.

The functional disturbance of A-V conduction, therefore, revealed itself in this patient not in early blockade of premature atrial impulses, but in a retardation of conduction, which became progressively more marked at shortening of the A₁A₂ interval.

Induction of ventricular premature beats during regular driving of the ventricle yielded complex results. At a rate of 100/min (basic cycle length, 600 msec) the V-A conduction time (time interval stimulus artifact to beginning of P wave in the intra-atrial lead) of the beats of the basic rhythm was found to be shorter than the antegrade A-V conduction time during driving of the atrium at the same rate, 160 and 260 msec, respectively.

Figure 6 shows the relation between the interval (V₁V₂) of the premature beat V₂ after the last beat of the basic rhythm V₁ and the interval between the resulting atrial activation (A₁A₂) at the rate of 100/min). In the V₁V₂ interval range of 600 to 450 msec most V₂ activations were conducted back to the atria with only a slight retardation (fig. 6, closed circles). In the V₁V₂ interval range of 480 to 450 msec some premature beats (fig. 6 open circles) were conducted in retrograde direction with a considerable prolongation of the V-A interval, the A₁A₂ interval varying between 700 and 720 msec. The same retardation of retrograde conduction was
Patient C. Antegrade A-V conduction of atrial premature beats $A_2$. Regular driving of right atrium with basic cycle length of 600 msec. Relation between interval atrial premature beat $A_2$ to last atrial beat $A_1$ of basic driven rhythm and the resulting $V_1V_2$ interval. Note the progressive slowing of A-V conduction at $A_1A_2$ intervals shorter than 300 msec. Ventricular complexes $V_1$ with long $V_1V_2$ intervals (open circles) in the $A_1A_2$ interval range of 250 to 230 msec were followed by atrial echo beats.

seen in the $V_1V_2$ interval range of 450 to 400 msec. In this range no premature beats with nearly normal V-A interval were found. Sometimes no V-A conduction takes place. The $A_2$ activations occurring after a long V-A interval (fig. 6, open circles) are always followed by a ventricular excitation with a QRS-complex $V^*$ of normal width and configuration, which can be recognized as a ventricular echo beat (fig. 7, delay of 470 msec, and fig. 8). The $A_2V^*$ interval varied between 260 and 300 msec, the shorter intervals being found after the shorter $V_1V_2$ intervals. No retrograde activation of the atria took place in premature beats with $V_1V_2$ intervals between 400 and 370 msec (fig. 7, $V_1V_2$ interval, 390 msec). $V_2$ complexes with intervals smaller than 370 msec down to that interval at which the refractory period of the ventricle was reached (190 msec) were followed again by an atrial activation (fig. 6 and fig. 7, $V_1V_2$ interval 350, 320, and 200 msec). The $A_1A_2$ intervals in this whole interval range showed only a small variation: 40 msec (435 to 475 msec).

That these atrial activations were not sinus or atrial escape beats is evident in the last tracing of

Figure 7 (V1V2 interval of 190 msec) in which the extra stimulus is given in the refractory period of the ventricle. No atrial escape beat is to be seen. Premature beats with V1V2 intervals between 370 and 290 msec are sometimes not conducted retrogradely.

Discussion

Experimental evidence has been given that the echo phenomenon is due to a longitudinal functional dissociation within the A-V node, as suggested by Scherf and Shookhoff in 1926. Moe and co-workers postulated the existence of a dual A-V nodal conduction
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Figure 7

Patient C. Retrograde V-A conduction of ventricular premature beats V₁ during regular driving of the right ventricle with basic cycle length 600 msec (compare with fig. 6). Only slight prolongation of V₂A₂ interval at V₁V₂ intervals down to 470 msec. At V₁V₂ interval 470 msec the V₂A₂ interval may be considerably prolonged: 350 msec. A₂ is followed by a ventricular echo beat V* after 340 msec. No V-A conduction is seen at V₁V₂ intervals 425 and 390 msec. V-A conduction is present again at V₁V₂ intervals between 370 and 195 msec. There are no ventricular echo beats in this V₁V₂ interval range. Note constancy of A₁A₂ interval. At V₁V₂ interval 190 msec the ventricle is found to be refractory.

system basing their conduction on indirect studies in the dog heart. The possibility of inhomogeneous conduction in the A-V node of experimental animals was recognized by several other workers.¹¹⁻¹³ The results of their experiments led Moe and associates to the concept that in the upper part of the A-V node two functionally different and separated pathways could be present, converging in a final common pathway which feeds the subnodal conduction system. In later experiments comprising multiple microelectrode records from

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Driving right ventricle in patient C. Ventricular echo beat V* following ventricular premature beat V2 with V1V2 interval 435 msec; basic cycle length, 600 msec. V-A conduction of V* is considerably prolonged: 390 msec. A2V* interval: 240 msec. Note negative polarity of P wave of A2 in leads II and III.

In the occurrence of ventricular echo beats retrograde conduction took place only along one pathway (α), the other (β) failing to participate presumably by a longer effective refractory period. The resulting atrial response could then be transmitted antegradely in the β pathway, which was open to conduction at that time. Unless the refractory period of the final common pathway prohibited further antegrade conduction, activation of the ventricles followed. In our three patients ventricular echo beats could be elicited by induced ventricular premature beats. The time relations of their occurrence could be defined. The genesis of these echo beats could be explained following the lines of the concept of Moe and associates.

In patient A the ventricular beats of the basic rhythm and the ventricular premature beats in the V1V2 interval range of 400 to 375 msec showing no increase in retrograde conduction time may be conducted to the atrium along a relatively fast conducting pathway β in the upper part of the A-V node (see diagram in figure 1). One can assume that this pathway is found to be refractory at V1V2 intervals shorter than 375 msec. Retrograde conduction to the atrium may then take place exclusively along a slower conducting pathway α with a relatively shorter effective refractory period. The negative polarity in leads II and III of the P wave resulting from this atrial activation indeed suggests a retrograde conduction to the atrium. The impulse may then invade the atrial end of pathway β, in which antegrade conduction at this time is possible. Further antegrade conduction through the final common pathway results in a ventricular complex with normal configuration V*, which is in fact a ventricular echo beat. This sequence is found in the V1V2 interval range of 375 to 360 msec (figs. 1 and 2). The blockade of retrograde conduction seen
at V₁V₂ intervals shorter than 360 msec may be localized in the α pathway or at a lower level.

In patient B (fig. 3) no sudden increase in V-A conduction time was seen before the occurrence of ventricular echo beats, as in patient A. Gradual shortening of the V₁V₂ interval from 600 to 470 msec resulted in a gradual increase of the V₂A₂ interval from 230 to 275 msec. In the V₁V₂ interval range of 470 to 440 msec the V₂A₂ interval varied between 275 and 305 msec and atrial complexes A₂ were followed by ventricular echo beats V* after a time interval, that varied between 100 and 160 msec, showing an inverse relationship with the V₂A₂ interval (figs. 3 and 4). In this patient (patient B) a mechanism somewhat different from that in patient A may be responsible (see diagram in fig. 3). The gradual and not stepwise increase in V-A conduction which is observed when the V₁V₂ interval gradually is shortened from 600 to 440 msec suggests that retrograde activation of the atria takes place along only one pathway (α in the diagram), in the upper part of the A-V node, the other pathway (β) not being capable of conduction in a retrograde direction.

Antegrade conduction of the resulting atrial activation A₂ in the β pathway may be possible at V₁V₂ intervals in the range 600 to 470 msec, but further antegrade conduction to the ventricles is presumably not possible while the final common pathway is still in its refractory phase, caused by the retrograde conduction of the V₂ impulse.

When retrograde conduction in the α pathway at shortening of the V₁V₂ interval is progressively retarded, the activation of the atrium A₂ and the following antegrade activation of the β pathway take place at a progressively later time. In the V₁V₂ interval range of 470 to 440 msec the impulse traveling antegradely through the β pathway, therefore, may find the final common pathway now nonrefractory and be conducted through it to the ventricles. The resulting normal ventricular complex V* is a ventricular echo beat. At V₁V₂ intervals shorter than 400 msec retrograde conduction may be blocked in the α pathway or lower.

Patient C clearly had a disturbance of antegrade A-V conduction. During sinus rhythm and during driving of the atrium the P-Q interval was prolonged. Early premature atrial beats gave rise to considerable retardation of A-V conduction and to atrial reciprocation. The genesis of this conduction disturbance was not clear. Six years earlier, when no signs of hyperthyroidism were present, the ECG already showed a first degree A-V block.

Ventricular echo beats were found after ventricular premature beats, during driving of the ventricle with a rate of 100/min, in the V₁V₂ interval range of 480 to 400 msec. In the V₁V₂ interval range of 480 to 450 msec the retrograde V-A conduction time of the premature beats could be either short (10 to 20 msec longer than the V-A interval of the beats of the basic rhythm) or long (240 to 270 msec longer than the basic V-A interval) (figs. 6 and 7, V₁V₂ interval, 470 msec). Atrial activations A₂ with long V₂A₂ intervals were followed by ventricular echo beats V* (fig. 8). The occurrence of these two distinct patterns in this interval range suggests the existence of two pathways in the upper part of the A-V node, one (β) conducting fast, the other (α) conducting slowly (see diagrams in fig. 7). Retrograde conduction along pathway β may be blocked occasionally at V₁V₂ intervals between 480 and 450 msec and always at shorter intervals. When the β pathway is blocked, retrograde conduction may occur along the slower pathway (α), which has a shorter effective refractory period. The resulting atrial response A₂ can then reflect antegradely through pathway β to the ventricles giving rise to the ventricular echo beat V*.

In the V₁V₂ interval range of 400 to 365 msec no retrograde activation of the atria is seen, presumably because of blockade of the impulses in both pathways β and α.

At V₁V₂ intervals shorter than 365 msec retrograde conduction is again present. The simplest explanation of this remarkable finding may be based upon the assumption of a
retardation of retrograde conduction at a lower level than the junction of pathways \(\alpha\) and \(\beta\) long enough for the impulse to find pathway \(\beta\) excitable again.

It is possible, however, that a far more complex mechanism is responsible. The relative constancy of the \(A_1A_2\) intervals (varying only between 435 and 470 msec) in the whole \(V_1V_2\) range (365 to 195 msec) in which this phenomenon is seen, is most likely the result of progressive retardation at shorter \(V_1V_2\) intervals. The progressive widening of the QRS complex at shorter \(V_1V_2\) intervals (figs. 7 and 9) suggests that this retardation might be at least partly localized at a level below the His bundle. This is not in contradiction of the findings of Moore,\(^{15}\) who showed in the isolated rabbit heart that retrograde conduction of ventricular premature beats could be considerably delayed in the Purkinje and bundle-branch system.

In the postulated mechanism of ventricular reciprocation in these three patients, the atrium serves as a necessary link in the re-entry pathway. An alternative hypothesis is that reflection of the impulse takes place in the A-V node at a lower level than the atrium. Experiments on dog hearts by Mignone and Wallace\(^{13}\) support the possibility of such a mechanism. The finding of normal ventricular activation following an induced ventricular premature beat without an interpolated retrograde atrial activation would be the only proof for the existence of subatrial reciprocation in our experimental setup. This sequence, however, was never seen.

In patients A and B ventricular echo beats were found accidentally. There were no clinical signs indicating an abnormality of the A-V conducting system. In additional stimulation experiments (not mentioned above) no disturbance of antegrade A-V conduction could be demonstrated in these two patients. The only abnormal hemodynamic finding in

\[\text{Figure 9}\]

Driving right ventricle in patient C. Retrograde V-A conduction of ventricular premature beat \(V_2\) at the \(V_1V_2\) interval of 200 msec. \(V_2A_2\) interval: 390 msec; basic cycle length: 600 msec. Note the remarkable broadening of the QRS complex of \(V_2\). No ventricular reciprocation is seen.

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both cases was an elevated cardiac index at rest.

Patient C, who had been treated for hyperthyroidism, also had a high cardiac index at rest. This man, however, had retarded antegrade A-V conduction. Atrial echo beats could be elicited by atrial premature beats.

The findings in patients A and B suggest that the occurrence of ventricular echo beats does not necessarily implicate a pathologic disturbance in the A-V conducting system but may be explained as a normal physiologic mechanism.

References
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