The Postponed Compensatory Pause as a Manifestation of Positive Feedback in Atrioventricular Conduction

By Leonard B. Goldberg, M.D., Matthew N. Levy, M.D., and Josef Edelstein, M.D.

SUMMARY
A patient with hypertensive cardiovascular disease was found to have unusual varieties of premature atrial and ventricular contractions. If the premature atrial contraction resulted in a greatly prolonged P-R interval, such that the increment in P-R exceeded the decrement in the preceding R-P, the next sinus P wave, occurring after a normal P-P interval, was found to be blocked. Also, numerous interpolated ventricular extrastoles were observed in which the postextrasystolic P-R intervals were markedly prolonged and in which the compensatory pauses were postponed for one or two beats. We also present data from one dog in which a premature atrial activation produced a chain reaction such that complete A-V block occurred three beats later. We propose that the chain reaction which evoked the delayed block in the dog and the postponed compensatory pauses in the patient reflects the operation of a positive feedback mechanism in A-V conduction. Positive feedback is initiated by an extremely long P-R, which results in a very short R-P before the next cycle. This then leads to a still longer P-R, which then elicits a still shorter R-P. Block ultimately supervenes when the atrial activation wave arrives at the A-V junction during its effective refractory period.

RECENT STUDIES from our laboratory have suggested that positive feedback operates during atrioventricular (A-V) conduction. In a positive feedback system, a primary change in some characteristic results in a series of reactions which ultimately leads to an even greater secondary change in that characteristic. The A-V nodal Wenckebach phenomenon reflects the operation of such a system. A-V conduction time (P-R interval) progressively increases until an atrial activation is blocked. Conduction then occurs with a relatively short propagation time, followed by progressive increases as the Wenckebach cycle is repeated. Electrophysiological evidence indicates that the delay in conduction per se alters conditions in the A-V junction such that the next impulse will arrive at an earlier phase of the relative refractory period, and hence conduction will be even more prolonged during the subsequent beat. Such behavior is characteristic of a positive feedback system.

Positive feedback also influences other aspects of A-V conduction. In a recent study on experimental animals in our laboratory, it was observed that very early stimuli to the atria sometimes resulted in such prolonged A-V conduction that the increment in the P-R interval of the premature contraction exceeded the decrement in the preceding R-P interval. Under such conditions, conduction of the next atrial activation was usually blocked, even though the P-P interval for that beat had returned to the control cycle length. In other words, the failure of A-V conduction which frequently accompanies a very early premature atrial activation was postponed for one cardiac cycle.

The present report describes the electrocardiographic changes in a patient in whom a postponed block of a premature atrial contraction was observed. This response resembled that which we had observed previously in the dog, but to our knowledge it has not been reported previously in human subjects. Furthermore, our patient had frequent premature ventricular contractions. Many of these ventricular extrastoles were interpolated, and most of these displayed the type of "postponed compensatory pause" originally described by Katz et al. in 1944 and by Langendorf in 1953. However, there were also several examples of doubly postponed compensatory pauses; we believe that this has never been described previously. We propose that a positive feedback mechanism is intrinsic to A-V conduction, and it is this mechanism which is responsible for the singly and doubly postponed compensatory pauses after interpolated ventricular extrastoles, as well as for the postponed block after a premature atrial contraction.

From the Divisions of Cardiology and Investigative Medicine, Mt. Sinai Hospital of Cleveland, and Case Western Reserve University, Cleveland, Ohio.

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Address for reprints: Leonard B. Goldberg, M.D., Division of Cardiology, Mt. Sinai Hospital, 1800 E. 105 Street, Cleveland, Ohio 44106.

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Case Report

The electrocardiograms shown in figures 1 to 4 were recorded from a 72-year-old man who was admitted to the hospital primarily for treatment of severe esophagitis with achalasia. The patient had a long history of hypertension. On admission, he was found to be in congestive heart failure, for which he was treated with digitalis and diuretics.

Figure 1 demonstrates a normal sinus rhythm interrupted by two extrasystoles. The second extrasystole (E₂) blocks a P wave and is followed by a classical compensatory pause. The first extrasystole (E₁) follows the preceding sinus conducted beat with a shorter coupling interval. The next P wave appears in the ST segment of E₁. The next QRS, R₄, occurs 0.67 sec after the onset of the preceding P wave. This postextrasystolic QRS complex (R₄) might represent either (1) a junctional premature beat, (2) a junctional escape beat, (3) a ventricular echo beat, or (4) a conducted beat with a very long A-V propagation delay. R₄ does not conform to the usual definition of a junctional premature beat, because the E₁-R₄ interval (0.78 sec) exceeds the B-R interval (0.70 sec) between two normally conducted beats. The interval for a junctional premature beat should be less than the prevailing R-R interval. It is also unlikely that R₄ is a junctional escape beat, because such a beat would be expected to terminate the longest R-R cycle. The E₂-R₄ interval is less than either R₄-R₅ (0.94 sec) or E₂-R₅ (0.91 sec). Both of these intervals are terminated by normally conducted beats. From the tracing in figure 1, the possibility that R₄ is a ventricular echo cannot be excluded. However, the records shown in figures 2 to 4 make such a mechanism unlikely, because atrial premature and sinus beats are observed which conduct with P-R intervals approximately as long as the P₂-R₅ interval in figure 1. Therefore, the most likely possibility is that the postextrasystolic QRS (R₄) in figure 1 is a delayed supraventricular response to the P wave which follows the interpolated extrasystole (E₂). The subsequent atrial depolarization is not conducted, presumably because it reaches the A-V junction during its effective refractory period.

The two extrasystoles which appear in figure 1 are associated with different types of compensatory pauses. The pause after E₂ immediately follows the extrasystole, and is the common variety of compensatory pause. On the other hand, pause R₃-R₄ does not occur immediately after the extrasystole, but it is delayed by one cardiac cycle. This type of pause has been termed the "postponed compensatory pause" by Katz et al.*

Figure 2 shows another segment from the same long rhythm strip. The fourth QRS complex (E₄) is an interpolated extrasystole which has a configuration similar to the first premature beat in figure 1. The postextrasystolic P-R interval is markedly prolonged (0.62 sec). The next P wave follows R₄ with an R-P interval of 0.12 sec. It is propagated to the ventricles after a conduction delay of 0.65 sec, slightly longer than the preceding P-R interval. This causes the next sinus P wave to follow R₄ by 0.09 sec, which is 0.03 sec shorter than the preceding R-P interval. This P wave is not conducted to the ventricles. Note that the pause associated with the blocked P wave follows a 3:2 postextrasystolic A-V nodal Wenckebach sequence. After a pause, a normal sinoventricular rhythm is re-established. Hence, the pause between R₄ and R₅ constitutes a postponed compensatory pause. It differs from the postponed compensatory pause in figure 1, however, in that it is doubly postponed.

In figure 3, the two strips are continuous, overlapping at the arrows. The lower strip shows an interpolated extrasystole (E₃) with a singly postponed compensatory pause, similar to the first extrasystole in figure 1. In the upper strip, a normal sinus rhythm is interrupted after R₃ by a premature atrial contraction. A supraventricular QRS (R₅) appears 0.71 sec after the onset of the premature P wave and 1.03 sec after the preceding QRS. This might represent either a junctional escape beat or a conducted beat after a greatly prolonged A-V conduction time. The latter interpretation is more plausible, because the P-R interval is approximately the same as those observed in this patient after numerous interpolated ventricular extrasystoles (figs. 1 to 3) and during the last beat of a series of Wenckebach cycles, to be described below (fig. 4).

The tracing shown in figure 4 was obtained from a monitor tape, which accounts for the observed distortion. In figure 4 there is a 5:4 A-V nodal Wenckebach sequence. The tracing is shown primarily to call attention to the long P-R interval (0.68 sec) in the last conducted beat of the Wenckebach cycle. This interval is in the same range as those for the postextrasystolic beats in figures 1 to 3. This lends further credence to our contention that the postextrasystolic QRS complexes in figures 1 to 3 are conducted beats rather than junctional escape beats.
Discussion

In 1926, Lewis and Master described the state of conductivity of the atrioventricular junction by means of a graph of the P-R interval plotted as a function of the preceding R-P interval. They referred to such a graph as the "recovery curve" of the A-V junction. Since that time, electrocardiographers have employed such recovery curves to explain the changes in the recovery curve.
in the P-R interval which occur in certain disturbances of cardiac rhythm, notably premature atrial contractions and the A-V nodal Wenckebach phenomenon. The curve in figure 5A illustrates the general shape of the recovery curve. For prolonged values of R-P, the P-R intervals are relatively short, and changes in R-P have little influence on P-R. However, at low values of R-P, the recovery curve is steep. Hence, near the left end of the curve, relatively small changes in R-P produce large alterations in P-R. Conditions which depress A-V conductivity are reflected by a shift in the recovery curve upward and to the right, whereas those conditions which facilitate A-V conductivity shift the curve in the opposite direction.

In our previous analysis of positive feedback in A-V conduction, we found it convenient to add a second component to the graph. This additional component is the diagonal line in figure 5A, which represents the locus of all points for which the sums of P-R and R-P equal the prevailing P-P interval. The combination of the recovery curve and the diagonal line facilitates the analysis of the changes in the P-R interval after premature atrial or ventricular contractions or during the A-V nodal Wenckebach phenomenon.

Ordinarily, the recovery curve and the diagonal line intersect at one or two points. If there is only one point of intersection, it would correspond to the stable equilibrium point, E_0, shown in panel A; this has been explained in detail previously. The coordinates of point E_u represent the steady state values of the R-P and P-R intervals, respectively. If there are two points of intersection, the upper point, E_u, would represent an unstable equilibrium condition. A premature beat for which the coordinates fall anywhere below point E_u would initiate a sequence of events such that the R-P and P-R values of the subsequent beats would rapidly approach the stable equilibrium point, E_0. An example of the sequence of changes evoked by an atrial premature beat is illustrated in one of our previous papers (fig. 3 in reference 1), and an example for an interpolated ventricular extrasystole is illustrated in another of our previous publications (fig. 7 in reference 2). The resulting sequence of changes in P-R and R-P intervals after such premature beats has sometimes been termed the reverse Wenckebach phenomenon.

In our previous study of the changes in the P-R interval after premature atrial contractions in the dog, we observed that a postponed block occurred when

![Figure 4](image_url)

*A-V nodal Wenckebach phenomenon, with a 5:4 block, in the same patient as in figures 1 to 3. Note the P-R interval of 0.68 sec in the last beat of the Wenckebach cycle.*

![Figure 5](image_url)

*In panel A, the recovery curve represents the changes in P-R interval as a function of the preceding R-P interval for a series of premature atrial activations. The diagonal line represents the locus of all points for which the R-P and following R-P intervals equal the basic cycle length. The lower point of intersection, E_u, is the stable equilibrium point; the upper point of intersection, E_u, is the unstable equilibrium point. Panels B and C illustrate the sequence of changes which occur when the R-P and P-R coordinates of a premature beat lie above E_u. In panel B, the compensatory pause is singly postponed; in panel C, it is doubly postponed.*
the R-P and P-R coordinates for the premature beat were located above the upper point of intersection, E_u (e.g., see fig. 2 in reference 1). Any point on the recovery curve above E_u represents a premature atrial beat that is characterized by an increment in P-R that exceeds the decrement in the preceding R-P. When this condition prevailed in our animal experiments, the atrial activation occurring after the premature beat was usually blocked at the A-V junction, i.e., the block was postponed for one beat. This block occurred despite the fact that the cycle length had already returned to the control value.

The sequence of events occurring after this type of premature atrial activation is illustrated in figure 5B. Point A, which lies above E_u, represents such a premature atrial contraction. If the basic cycle length is reinstituted on the next beat, the R-P interval may be ascertained by drawing a horizontal line from point A on the recovery curve to the diagonal line, since this line represents the locus of points for which P-R + R-P equals the basic cycle length. If this next R-P value is less than the minimum value for R-P on the recovery curve, the atrial activation would arrive at the A-V junction during its effective refractory period and would be blocked. This is indicated in panel B by the fact that the vertical line lies totally to the left of the recovery curve.

The atrial premature beat in the upper segment of figure 3 fulfills the criterion for a point on the recovery curve above the upper, unstable equilibrium point. The first R-P interval (R_1P_2) was 0.44 sec, which was the steady state value. The next R-P (from R_2 to the premature P) measured 0.32 sec, a decrement of 0.12 sec. The steady state P-R interval was 0.24 sec, and it increased to 0.71 sec during the premature beat, an increment of 0.47 sec. Hence, the increment in P-R greatly exceeded the decrement in R-P, thereby satisfying the criterion for a point above E_u on the recovery curve (analogous to point A in fig. 5B). As a consequence of the marked augmentation of P-R, the subsequent R-P was only 0.05 sec. Since the next P wave was blocked, this atrial activation wave must have arrived at the A-V junction during its effective refractory period.

A similar mechanism probably accounts for the postponed compensatory pause which may follow an interpolated ventricular extrasystole; examples are shown in figure 1 and figure 3 (lower strip). In figure 1, after the interpolated extrasystole (E_1), concealed retrograde conduction into the A-V junction prolonged the postextrasystolic P-R interval (P_3-R_3) to 0.67 sec, from a steady state level of 0.21-0.22 sec. This excessively long P_3-R_3 interval resulted in a critical curtailment of the subsequent R-P to 0.03 sec, and hence the next P wave (P_4) was blocked. Thus, the mechanism for the postponed compensatory pause after an interpolated ventricular extrasystole resembles that for the delayed block of a premature atrial contraction, as diagrammed in figure 5B. However, the quantitative relationship of R-P to P-R for a ventricular extrasystole is quite variable and hence differs from that for a premature atrial contraction. This was recognized by Katz et al. in 1944.

The mechanism responsible for the doubly postponed compensatory pause (fig. 2) may be appreciated by referring to figure 5C. Point A in this diagram corresponds to the postextrasystolic beat with a greatly prolonged P-R interval (0.62 sec). Because of the marked increase in P-R, the next R-P amounted to only 0.12 sec. However, this R-P interval was evidently still compatible with conduction through the A-V junction, but it resulted in a still greater P-R interval of 0.65 sec (corresponding to point B in fig. 5C). By virtue of the additional increment in P-R of the second (point B) relative to the first (point A) postextrasystolic beat, the R-P interval was further reduced to 0.09 sec (fig. 2). This is represented in figure 5C by the horizontal line from point B to the diagonal line. Evidently, this R-P interval was not compatible with A-V conduction, since the third postextrasystolic P wave was blocked.

No instances of doubly postponed blocks were observed in this patient after premature atrial contractions, nor have any been described to our knowledge. In reviewing our previous series of animal experiments, however, we have uncovered one example of a triply postponed block after a premature atrial excitation. A record is presented in figure 6. The upper tracing is the right atrial electrogram, the lower the right ventricular electrogram, each recorded from separate bipolar electrode catheters in an open-chest, anesthetized dog. The animal was paced from the right atrium at a basic cycle length of 300 msec, and once every 100 beats a shorter cycle length (in this instance, 237 msec) was interposed. The P-P, P-R, and R-P intervals were printed out to the nearest msec on a beat-by-beat basis with the aid of a digital computer (PDP 12). It is apparent from figure 6 that the short cycle (P_3-P_4) was accompanied by a curtailed R-P interval (113 msec), and it was followed by a prolonged P-R interval (195 msec). With each succeeding beat, R-P was further abridged (to 110, 107, and 98 msec) despite the return to the control cycle length after the single abbreviated cycle. The R-P interval of 98 msec must have been incompatible with A-V conduction, since the next atrial activation (P_5) was blocked.

Hence, the postponed blocks after premature atrial contractions and the postponed compensatory pauses after interpolated extrasystoles appear to be manifestations of positive feedback during A-V con-
In terms of its gross electrocardiographic counterparts, a premature atrial or ventricular contraction causes a prolongation of the P-R interval. If the basic cycle length remains constant after the extrasystole, the next R-P interval will be curtailed. Shortening of the R-P in turn tends to produce a still greater prolongation of P-R, thereby constituting a positive feedback system. When a P-R prolongation evoked by an extrasystole is such that it lies on the recovery curve above the upper, unstable point of equilibrium (E_u), the increment in P-R with each beat will exceed the decrement in the preceding R-P. This constitutes positive feedback with an open-loop gain greater than unity. Such A-V conduction is highly unstable and will terminate in a block of the atrial activation wave in the A-V junction within a very few beats.

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Figure 6

The right atrial (P) and right ventricular (R) electrograms recorded from intracavitary electrode catheters in an anesthetized dog. The atria were paced at a basic cycle length of 300 msec, and one premature atrial activation (P2-P1 = 237 msec) was produced after each sequence of 99 basic cycles. Note that the premature P wave (P1) was not blocked, but that the third P wave (P3) after the premature P wave was blocked.

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L B Goldberg, M N Levy and J Edelstein

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