
The author replies:
To the Editor:

Dr. Shaffer's analysis of figure 3 is correct, that the total deaths in the first twelve weeks were entirely in the high-risk subgroup. The total mortality during this period was 26% (14 of 54 patients). Our group considers patients in this subgroup with a combination of high-risk factors, e.g., unstable angina not responding to in-hospital medical management within 48 hours, prior stable angina, and ischemic ST change during pain as urgent candidates for coronary artery bypass surgery.

PETER C. GAZES, M.D.
Director, Cardiovascular Division
Medical University of South Carolina
Charleston, South Carolina

A-V Nodal Wenckebach Cycles and Reentry

To the Editor:

Damato and associates1, 2 ascribe the block of antegrade A-V nodal Wenckebach cycles to reentry. The concept is based chiefly on the "obligatory ventricular echo" (our term) which accompanies retrograde nodal Wenckebach cycles. If the rate is slow, all retrograde Wenckebach cycles end in an echo. Even if it appears to end in a blocked beat at faster rates, concealed reentry can often be demonstrated, as Damato has shown.

Antegrade cycles almost always end in a blocked beat, rarely in a manifest atrial echo. Antegrade cycles are common clinically while the retrograde variety is infrequent. The authors assume that antegrade periods end with a concealed reentry beat. We are asked to accept that over 95% of clinical Wenckebach cycles contain concealed reentry. We believe a more acceptable explanation is possible.

We have described mechanisms for the Wenckebach period without using reentry; furthermore we use Wenckebach mechanisms to explain reentry.3 4

The slow conduction is an exaggeration of the decremental conduction normally present in the node. The progressive delay from beat to beat is caused by a refractory period (RP) which normally lasts much longer than the action potential, by resetting of the RP and by the electrotonic prolongation of the action potential inherent in slow conduction. The relatively fast conduction of the first beat of the cycle is caused by retrograde electrotonic acceleration of repolarization of cells upstream to the block and by the nonactivated fully polarized cells downstream. The sharp increase of delay of the second beat results in part from the Ashman phenomenon.

To account for the obligatory association of the retrograde Wenckebach cycle with return beats we utilized a specific structure of the A-V node as described by Truex.5 The atrio-nodal region consists of spokes of nodal cells radiating into the atrium. A retrograde impulse undergoing Wenckebach delay will sooner or later be conducted through the atrium via some spokes and will be blocked in others. The impulse spreads from a locally activated atrial region to the previously blocked region where it is now conducted antegradely.

Because of the mechanisms inherent in the Wenckebach cycle, the cells on both sides of the block are recovered by this time so that antegrade conduction is faster than retrograde.

The anatomy also accounts for the rarity of atrial echoes. During antegrade Wenckebach cycles the entire atrio-nodal region is activated more or less simultaneously. If the impulse does not die out in the nodal spikes it will do so in the network in the body of the node. The net summates and neutralizes impulses. The random path through the net would not favor the formation of laterally dissociated pathways. The appearance of an atrial echo suggests a bypass tract, a grossly asymmetric activation of the atrio-nodal region or anatomic partitions within the node.

References

The author replies:
To the Editor:

Doctors Schaffer and DePasquale have raised interesting and challenging questions concerning the phenomena of A-V nodal Wenckebach cycles and reentry. As I interpret their comments, they ascribe Wenckebach periods to a combination of decremental conduction and electronic interchange between cells proximal and distal to an area of delay or block within the A-V node. Furthermore, they suggest that a specific anatomical substratum at the atrio-nodal junction accounts for the greater frequency of ventricular echo beats with retrograde A-V nodal Wenckebach cycles as well as the "rarity" of atrial echoes with antegrade Wenckebach cycles.

I view our differences concerning the mechanism of the Wenckebach cycle as more apparent than real.
During A-V nodal delay for single beats as well as successive beats of the Wenckebach cycle, an alteration in the configuration of the transmembrane action potential occurs. In certain nodal cells this alteration takes the form of notches during the repolarization or depolarization phases of the action potential. These notches which have been ascribed to electronic spread or current flow from distal or neighboring cells back to the region of the impaled cell have the effect of prolonging the duration of the impaled cell. During antegrade conduction delay, the timing of these notches coincide almost exactly with the upstroke of cells distal to the area of conduction delay. This relationship could be further substantiated by correlating the onset of notches with properly timed retrograde activation of distal cells during antegrade conduction delay.

If one defines reentry within the A-V node as a wave of depolarization or flow of current propagating simultaneously in opposite direction to the initiating wave of depolarization, then one can view (as we have) electronic interaction or prolongation as a reflection of reentry. This is to say, current is flowing in an opposite direction and is reflected as a notch or prolongation of the action potential of the impaled cell. Since notching or electrotonic interaction can be demonstrated throughout the entire Wenckebach cycle, then some degree of reentry is occurring on a beat to beat basis.

The critical question is whether this continuous reentry accounts, in part or whole, for the beat to beat prolongation characteristic of Wenckebach cycles. There is little doubt that the culmination of this reentry process is either an echo (manifest reentry) or a blocked beat (concealed reentry). Since Drs. Schaffer and DePasquale argue that the progressive delay from beat to beat is due in part to electrotonic prolongation of the action potential and since one could interpret electrotonic prolongation as a form of reentry, then I think that reentry as a mechanism for the Wenckebach phenomenon, and not the reverse, should be given serious consideration.

Atrial echo beats associated with or terminating antegrade Wenckebach cycles are not rare as suggested by Doctors Schaffer and DePasquale. In addition to our original six clinical cases we have continued to observe this phenomenon in approximately 40% of our clinical cases in which antegrade Wenckebach cycles are induced by antegrade Wenckebach cycles are induced by atrial pacing. In a planned study of this phenomenon we have been able, in 5 consecutive patients, none of whom had tachycardias, to convert ordinary Wenckebach cycles (concealed reentry) into Wenckebach cycles terminated by atrial echoes by delicately adjusting the cycle length of atrial pacing and the degree of antegrade A-V nodal delay.

As regards the concept of "nodal spokes" radiating into the atrium and the role of this suggested anatomic structure in the production of atrial and ventricular echo beats, it should be stated that no electrophysiological evidence is available to support such a concept. Furthermore, since there is some evidence to suggest that the atrium is not a required link in the production of ventricular echo beats the role of nodal spokes need not be invoked to explain this phenomenon. In addition, it has been demonstrated that echo beats can result from conduction delay and reentry within the bundle of His and other ventricular specialized conduction tissue. Thus, reciprocation within the bundle of His resulting in ventricular echo beats would also indicate that the concept of "nodal spokes" is not required.

ANTHONY N. DAMATO, M.D.
Chief, Cardiovascular Program
U.S. Public Health Service Hospital
Staten Island, N. Y.

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ANTHONY N. DAMATO

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