Mechanisms of Supraventricular Tachycardia*

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"Circus movement" or continuous cyclic conduction along a closed pathway was proposed by Mines (6) in 1913 to explain paroxysmal tachycardia. In 1943, Barker *et al.* (1) suggested that the S-A node or the A-V node, on the basis of their properties of slow conduction, were the turn around sites of such a circus or reentry arrhythmia. Insofar as the A-V node was concerned, this postulation was confirmed and expanded by the investigations of Moe *et al.* (7) who showed that under conditions of slowed conduction the A-V nodal pathways could dissociate functionally. More recently, Mendez and Moe (5) using microelectrode recording techniques have found that areas within the A-V node can be identified as the sites of rebound or echo beats which returned to the atrium. Wit *et al.* (13) were able to induce paroxysmal tachycardias in isolated portions of rabbit atria containing the A-V node. Clinically, Goldreyer and Bigger (2), induced and terminated, in patients with a predisposition to paroxysmal supraventricular tachycardia, periods of supraventricular tachycardia with appropriately placed atrial premature beats. Their evidence from His bundle recordings pointed to the A-V node as a site of reentry for these paroxysmal supraventricular tachycardias in man.

In figure 1, a schematic representation is shown of the series of events that might occur in the course of an induced period of supraventricular tachycardia. After a normally conducted sinus beat (SB), an atrial premature beat (APB) occurring spontaneously or electrically induced is conducted slowly through the A-V node. If the A-V nodal delay is great enough to produce functional longitudinal dissociation, a reentrant pathway can be set up either giving rise to one rebounding beat into the atrium, an echo beat (EB), or a continuous circuit may ensue within the A-V node with atrial and ventricular responses being thrown out of the node with each intra-nodal circuit. The interruption of the circuit can occur due to another atrial premature beat thus terminating the paroxysm and reinstituting sinus activity. There is both clinical and experimental evidence supporting the intra-A-V nodal circuit as the prime mechanism involved in reentry arrhythmias such as paroxysmal atrial tachycardias or in reciprocal beating. On the other hand, there are studies which clearly indicate that the atrium participates in the reentry pathway. For example, in the study of Wit *et al.* (13), these authors found that excision of a large portion of atrial muscle prevented the induction of paroxysmal tachycardia in the isolated rabbit preparation, and Goldreyer and Bigger (3) also indicated on the basis of A-V nodal conduction times during paroxysmal supraventricular tachycardia in patients that the atrium was involved in the reentry path. Thus, in our schematic representation, we have indicated by the dotted lines the possibility that activity echoing back from the A-V node, the first site of reentry, can enter the atrium, which serves as the second site of reentry, to continue the paroxysmal tachycardia. It is at this point, where intra-atrial reentry is implicated that one encounters a controversy that has occupied a great deal of journal space

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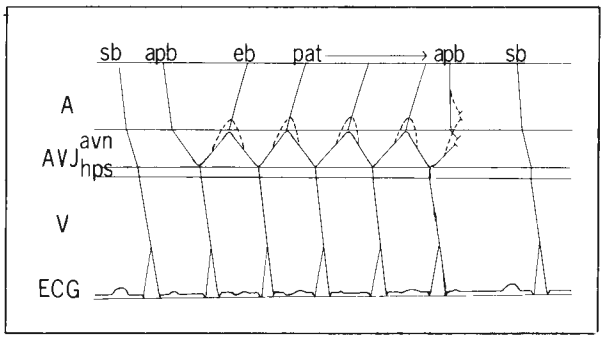

Fig. 1—A schematized version of impulse conduction during induced atrial tachyarrhythmias. The modified ladder diagram depicts three major cardiac compartments: the atrium (A); the A-V junction (A-VJ) composed of the A-V node (avn) and the His-Purkinje system (hps); and the ventricle (V). The electrocardiogram is depicted below (ECG). Other abbreviations are: sb = sinus beats; apb = atrial premature beats; eb = echo beats; pat = paroxysmal atria tachycardia. See text for discussion.

recently. In looking at the schematic, particularly the atrial bridge, one notices that at the point at which atrial activation must reverse itself atrial refractoriness must be infinitely rapid or another area of slow conduction must be postulated to account for the atrial bridge. In a recent issue of *Chest,* Surawicz (11) has concisely addressed himself to this point and indicates that the electrophysiologist must return to the drawing board, so to speak, in order to elucidate mechanisms by which the atrial bridge hypothesis can be substantiated. Below, we would like to present evidence for perhaps one mechanism by which an intra-atrial bridge or a second reentry site may be involved in paroxysmal supraventricular tachycardias.

Our investigations were based on an interesting observation and clever postulation made by Vassalle, Greenspan, and Hoffman in 1963 (12). They observed that a ventricular escape beat which terminated a vagal arrest of the heart often initiated atrial fibrillation. They postulated that in some way the retrograde impulse entering the atrium proceeded in a heterogeneous fashion so that the dissociated atrial wave front could establish a return pathway(s) within the atrium thereby initiating atrial flutter or fibrillation.

We were able to reproduce this phenomenon and analyze atrial activation patterns (fig. 2). In panel A, close bipolar electrograms were recorded in various parts of the dog atria in order to find the earliest and latest epicardial areas of activation. These corresponded to the high right atrium in the area of the sinus node and the inferior-posterior left atrium, respectively. The total antegrade atrial activation time, that is the time from the beginning of the earliest activation to the end of the latest activation or total atrial activation time (AA), was 100 msec. In contrast, during ventricular pacing with retrograde atrial activation, AA was only 65 msec. Note that the sequence of activation during retrograde conduction showed low atrial activity occurring first and sinus activity as well as posterior left atrial activation occurring last.

In panel C, an asystolic interval with a ventricular escape time equal to 12 seconds due to vagal stimulation was terminated by a ventricular escape beat which produced a normal retrograde atrial activation pattern. Note that the sequence of activation as well as the total atrial activation is the same during this retrograde atrial activation as that seen with ventricular pacing in panel B. No arrhythmia occurred subsequent to this ventricular and atrial beat. In the same animal, panel D, with a 13-second asystolic period, a ventricular escape beat was associated with activation of the atria which differs markedly from the normal retrograde atrial activation pattern. Although the first area to be activated in the low right atrium is in the area of the coronary sinus ostium, the other low right atrial region approximately 3–5 mm away is activated later. Note that in panel C, both areas were activated almost simultaneously. Total duration of retrograde atrial activation now occupies 100 msec instead of the normal 65 msec. Subsequent to this initial aberrant atrial activation pattern atrial fibrillation ensued. In those animals in which ventricular escape beats often produced runs of atrial fibrillation, this phenomenon of dispersion of atrial activation was regularly seen and was clearly related to the occurrence of atrial arrhythmias.

We found that the dispersion of atrial activation was not limited to ventricular escape beats with retrograde atrial activation. It also occurred commonly with retrograde Wenckebach cycles (fig. 3). During ventricular pacing at 120 beats per minute there was regular 1:1 retrograde atrial activation. The total atrial activation time was 55 msec. When the ventricular rate was increased to 145 per minute, the Wenckebach phenomenon was seen with a reciprocal beat. The retrograde atrial activation interposed between the ventricular and normal sinus beat shows a total activation time of 65 msec com-

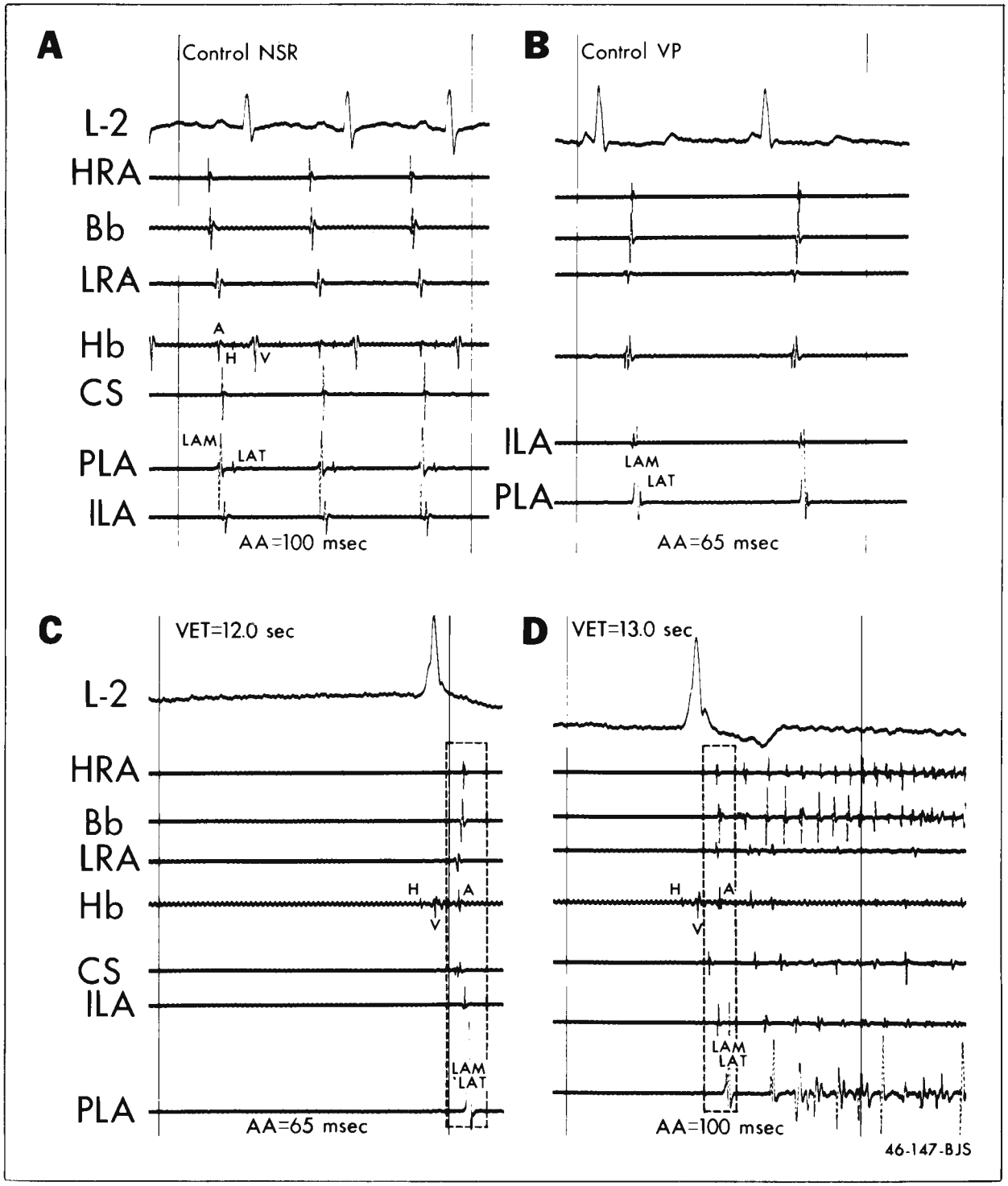

Fig. 2—The phenomenon of atrial fibrillation associated with a ventricular escape beat during vagal induced cardiac arrest. Traces from top down: Lead II (L-2) ECG; bipolar plunge wire electrograms from the high right atrium (HRA): Bachmann's bundle (Bb); low right atrium (LRA); His bundle region (Hb) showing atrial (A), His bundle (H), and ventricular (V) activation; coronary sinus ostium (CS); posterior left atrium (PLA) showing left atrium muscle (LAM), and left atrial tract (LAT) potentials (13); and from the inferior left atrium (ILA). A. Total atrial activation time (AA) measured from the beginning of the earliest activation in the

high right atrium to the end of the latest activation, LAT in the posterior left atrium, is 100 msec. B. During the ventricular pacing (VP) with retrograde atrial activation the total atrial activation time equals 65 msec. C. With vagal stimulation induced cardiac asystole and a ventricular escape time (VET) of 12 secs, the ventricular escape beat produces normal retrograde atrial activation with a total atrial activation time of 65 msec. No atrial arrhythmia ensues. D. With a ventricular escape time of 13 secs, a ventricular escape beat with the same configuration as seen in C produces a dispersion of retrograde atrial activation. Compare the first sequence of atrial activation in panel D (within the dotted rectangle) with the first sequence of atrial activation seen in panel C. Total atrial activation equals 100 msec in panel D for the first atrial sequence as compared to the normal 65 msec seen in panels B and C. Note the occurrence of atrial fibrillation subsequent to the dispersion of atrial activation in panel D. Interval between time lines equals 1 second.

pared to normal retrograde activation time of 55 msec, probably indicative of some dispersion of atrial activation associated with the stress of A-V nodal conduction. When the rate was increased to 150 per minute a short Wenckebach cycle was seen with a reciprocal beat. Only the last beat of the Wenckebach cycle shows dispersion of atrial activation with a total time of 75 msec compared to the normal 55 msec. Also note that there is a direct relationship between the degree of dispersion and the degree of retrograde A-V nodal delay. When the H-A time was 165 msec, AA was 65 msec, whereas when the H-A time was 195 msec, the AA was 75 msec. With 2:1 conduction no dispersion of atrial activation was seen.

In figure 4, we have presented a schematized version of what may be taking place during retrograde atrial activation when dispersion of atrial activation occurs. In panel A, the normal pattern of retrograde atrial activation is shown as recently demonstrated by Spach *et al.* (10) in the dog and rabbit A-V nodal preparation. These authors found that activation coming out of the A-V node rather uniformly excited atrial musculature at the A-V nodal-atrial border. In panel B, on the other hand, we have postulated that during stress of A-V nodal conduction during which there is "functional longitudinal dissociation" of activation through the A-V node, impulses may asynchronously exit from one portion of the A-V node into adjacent atrial tissue; whereas at other sites within the A-V node, marked slowing or block of retrograde impulses occur. Due to this asynchronous exit from the A-V node, atrial activation may proceed either back into the A-V

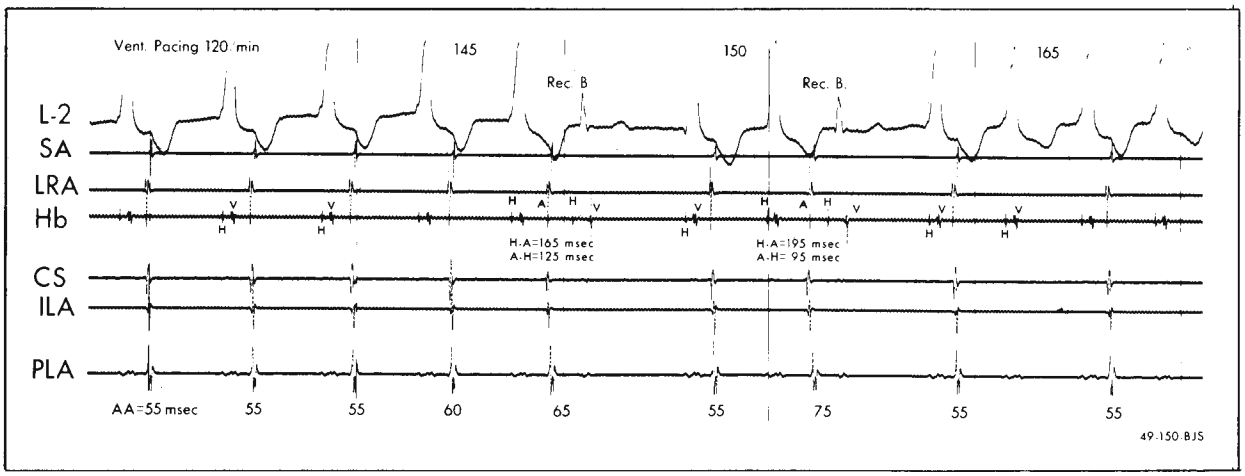

Fig. 3—The occurrence of atrial dispersion during retrograde Wenckebach cycles. Traces from above: Lead II (L-2) ECG: bipolar plunge wire electrograms from the sinus node area (SA); the low right atrium (LRA); the area of the His bundle showing only His bundle activity (H) and ventricular activation (V); coronary sinus ostium (CS); the inferior left atrium (ILA); and the posterior left atrium (PLA). During ventricular pacing at 120 per minute with 1:1 retrograde conduction, atrial activation time (AA) equals 55 msec. As the rate is increased to 145 per minute, a Wenckebach cycle occurs ending in a reciprocal beat (Rec. B). Note that the total atrial activation time increases from 60 to 65 msec just prior to the reciprocal beat and coincident with the retrograde Wenckebach cycle. With a ventricular pacing rate of 150 per minute, another Wenckebach cycle occurs and now the beat prior to the reciprocal beat shows dispersion of atrial activation, 75 msec. With ventricular pacing at 165 per minute there is 2:1 retrograde conduction with no change in the atrial activation pattern or total atrial activation time from the normal.

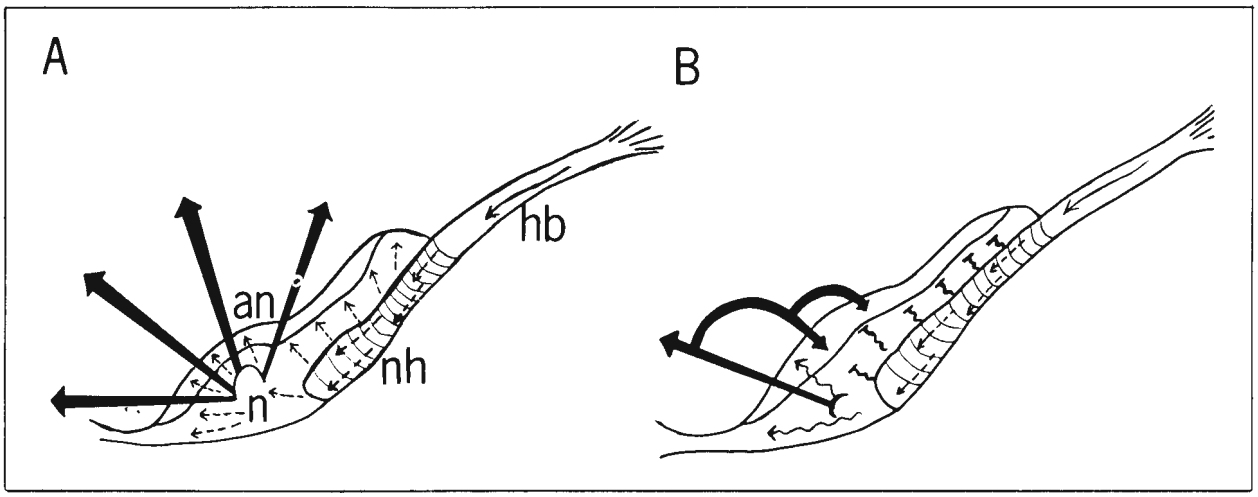

Fig. 4—A schematic version of retrograde atrial activation. A. Indicates activation proceeding from the His bundle retrograde through the various regions of the A-V node: nh, n, an. (After Spach *et al.*) (10). Activation spreads uniformly into the atrium. B. Retrograde activation with asynchronous exit of impulses from the A-V node leading to the possibility of reentry into the node or the atrium. See text for discussion.

node and then to the ventricle to produce a reciprocal beat, or activation may proceed in an asynchronous manner so that local reentry circuits may develop at one or more sites to produce atrial arrhythmias. In any event, dispersion of atrial activation would be a consequence of this asynchronous exit from the A-V node.

In order to relate this phenomenon of dispersion of atrial activation with the occurrence of reentry atrial tachycardias, we attempted to induce echo beats in the dog atrium with the delivery of premature stimuli to the atrium during sinus rhythm (fig. 5). To enhance the occurrence of echo beats, we also delivered local subthreshold epicardial stimulation to a site in the low right atrium near the coronary sinus ostium so that parasympathetic nerve elements supplying the A-V node were preferentially affected (8). In this way, exacerbation of A-V conduction delay was obtained. In panel A, normal retrograde atrial activation time was 55 msec; in panel B, a premature atrial stimulus was delivered to the high right atrium during sinus rhythm and induced an echo beat. Note the stimulus artifacts seen on the coronary sinus (CS) trace. These are due to the subthreshold stimuli applied to epicardial parasympathetic nerve elements (local vagal stimulation). The echo beat has a general appearance of retrograde atrial activation with activity in the low right atrium occurring before activation of the high right atrium or the posterior left atrium. Note also that the total atrial activation is slightly prolonged during this echo beat as compared to the normal retrograde atrial activation pattern, 85 msec as compared to 55 msec. In panel C, with the same coupling interval, an atrial echo beat was induced by an atrial premature systole, however, it was followed by another reentry beat. Note that there was a greater degree of dispersion of atrial activation, 95 msec, in the echo beat seen in panel C as compared to the echo beat in panel B, 85 msec. The reentrant beat also showed the general appearance of retrograde activation; however, the degree of atrial dispersion is now only 85 msec and reentrant rhythm spontaneously terminated. Also note that the degree of dispersion of atrial activation can appear on the surface ECG as well although the interposition of P wave, ST, and T waves may obscure this aspect. Thus, it can be seen that with a high degree of dispersion, the P wave is broadened with 95 msec of atrial dispersion as compared to 85 msec. The P wave polarity remains the same.

Dr. Alfred Pick was kind enough to bring to our attention some suggestive evidence that atrial dispersion does occur in patients under conditions of compromised A-V conduction. In figure 6, taken from Katz and Pick (4), it can be seen that during retrograde Wenckebach cycles not only does the

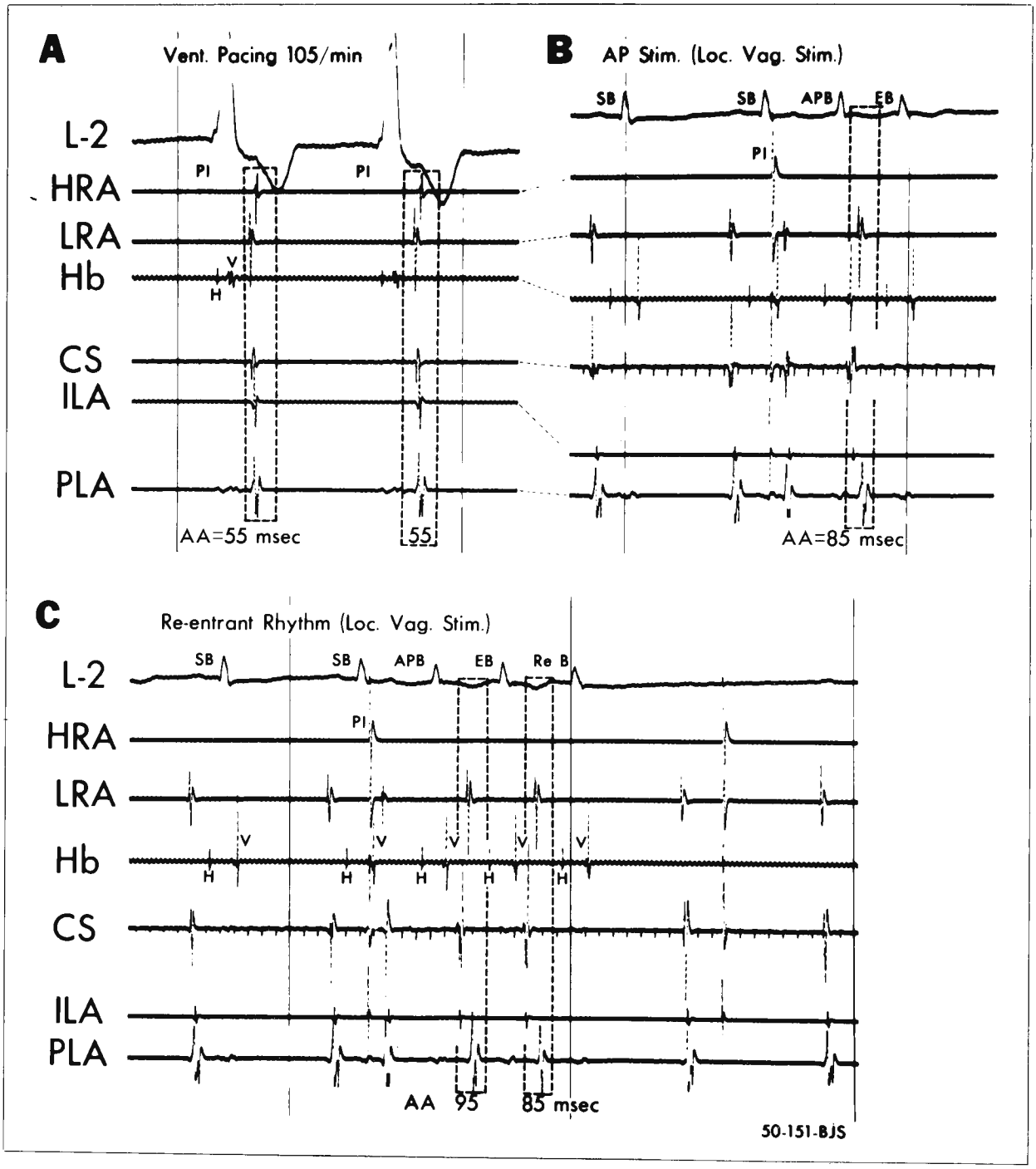

Fig. 5—Comparison between normal retrograde activation in the atrium and dispersion of atrial activation. Traces from above: Lead II (L-2) of the ECG; bipolar plunge wire electrograms from the high right atrium (HRA); the low right atrium (LRA); the His bundle region (Hb); the coronary sinus ostium (CS); the inferior left atrium (ILA); and the posterior left atrium (PLA). A. During ventricular pacing at 105 beats per minute retrograde conduction to the atrium indicates early activation of the low right atrium and coronary sinus regions with activity proceeding to the high right atrium and posterior left atrium. The total atrial activation time (AA) in each beat is 55 msec. PI = pacer impulse. B. Atrial premature stimuli (AP Stim.) was applied to the heart after two simultaneous sinus beats (SB). The atrial premature beat (APB) is followed by an echo beat (EB). Note the inverted P wave in lead II as

well as the sequence of atrial activation (within the dotted rectangle) conforming to the general retrograde atrial activation pattern as seen in panel A. However, atrial activation time was 85 msec, a 30 msec increase from that seen during normal retrograde atrial activation. The small stimulus artifacts seen on the coronary sinus trace indicate the application of subthreshold epicardial stimulation to parasympathetic nerve elements supplying the A-V node (Loc.Vag. Stim.) (5). C. A short burst of reentrant rhythm is shown with the introduction of an atrial premature stimulus which induces an atrial premature beat followed by an echo beat and another spontaneous beat labelled REB or reentrant beat. The last two beats of this short run both show characteristics of retrograde atrial activation; however, the echo beat now shows an atrial activation time of 95 msec followed by a reentrant beat with atrial activation time of 85 msec. See text for discussion.

R-P interval lengthen, but also with this lengthening there is a slight but appreciable broadening and deeping of the retrograde P wave with the greatest effect occurring prior to the reciprocal beat.

In summary, we have found a close association between the stress of A-V nodal conduction and the occurrence of atrial dispersion or aberration. We could not explain atrial dispersion on the basis of incomplete recovery of atrial activity, slowing of atrial conduction, or the occurrence of atrial fusion. We postulate that the phenomenon of dispersion of atrial activation is related to the functional dissociation within the A-V node seen during situations which stress A-V conduction. This phenomenon may represent a mechanism whereby a second area of "turnaround" other than the A-V node is involved in reentry atrial tachyarrhythmias. Indeed, such a mechanism could provide a physiological basis for the concept of the atrial bridge being involved in supraventricular tachycardias and reciprocal beating.

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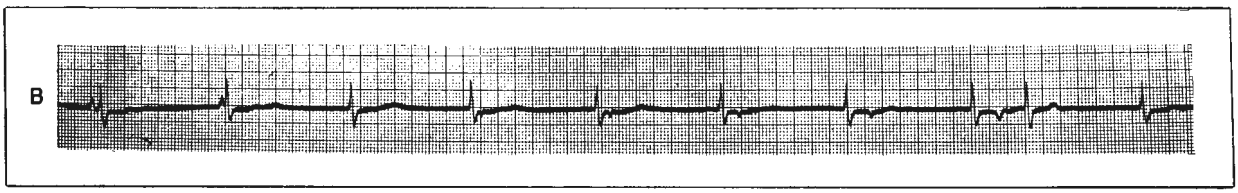

Fig. 6—An electrocardiograph tracing taken from a patient showing a retrograde Wenckebach cycle ending in a reciprocal beat. Note that with the progressive lengthening of the R-P interval, there is a broadening and deepening of the P wave (Reproduced with permission from Katz, L. N. and Pick, A. *Clinical Electrocardiography*, Part I. "The Arrhythmias." Lea and Febiger, Philadelphia, 1958, Chapt. 9, fig. 67).

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