The Evaluation of Sinoatrial Node Function in Man*

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The function of the sinoatrial node is complex. In nearly all hearts, this small bit of tissue is responsible for spontaneously generating the impulse which will be distributed to the remainder of the heart, maintaining coordinated electrical and mechanical function. In recent years, it has become clear that S-A node dysfunction is not rare, can cause disabling symptoms, and often presents difficult management problems. The challenges presented by the "Sick Sinus Syndromes" have increased our desire to know more about normal S-A node function and about function in disease states.

The intimate mechanisms of sinus node function remain a mystery despite the "prying eye" of the microelectrode and modern anatomical and chemical methods. At least the time-voltage course of spontaneous activity in the sinus node cells has been elucidated. After self-excitation, a pacemaking sinus node cell slowly depolarizes to zero potential difference with the extracellular fluid and often shows some overshoot, that is, the inside of the cell may become slightly positive relative to the extracellular potential. After reaching this peak inside-positive value of transmembrane voltage (V_m), the sinus node cell slowly repolarizes to a maximum inside-negative value—so-called maximum diastolic voltage. Then, the transmembrane voltage spontaneously begins to decrease (phase 4 depolarization) until a critical value of V_m, threshold voltage, is reached and self-excitation recurs. The rate of recurring self-excitation could theoretically be altered by changes in: 1) maximum diastolic voltage, 2) threshold voltage, and 3) rate of phase 4 depolarization. Changes in firing of a sinus node cell are most often mediated by changes in rate of phase 4 depolarization. We still do not know the precise sequence of membrane permeabilities as a function of time and voltage which are responsible for the normal automatic behavior of the S-A node.

Sinus node rate is sensitively adjusted to most suitably meet the needs of the body as a whole. These adjustments are usually mediated through autonomic reflexes which change the rate and pattern of firing on sympathetic and/or parasympathetic nerves terminating at or near the sinus node. Release of norepinephrine from sympathetic nerve terminals in the vicinity of a sinus node cell will accelerate phase 4 depolarization and the spontaneous firing rate of the S-A node while acetylcholine released from cholinergic terminals has the opposite effect.

* Supported in part by a Grant-in-Aid from the New York Heart Association and by United States Public Health Service Grants HL 12738 and HL 07541.

Presented by Dr. Bigger at the Symposium on Cardiac Arrhythmias, June 10, 1972, at Virginia Beach, Virginia.

† Recipient of a Research Career Development Award from the National Heart and Lung Institute (1-K04-HL-70204); this work was begun during his tenure as Senior Investigator, New York Heart Association.

‡ Pharmaceutical Manufacturers Association Foundation Faculty Development Awardee in Clinical Pharmacology; this work was begun during a National Heart and Lung Institute Traineeship (USPHS Grant 5-T12-HL-05864).

Once the normal automatic mechanism has generated an impulse in the S-A node itself, the impulse must be transmitted to the ordinary and specialized atrial fibers in order to effect atrial and, ultimately, total cardiac excitation. The action potential of the S-A nodal cell is of a type associated with slow conduction and vulnerability to block or fragmentation in transmission, that is, phase 0 is small in amplitude and extremely slow rising. Also, the S-A nodal pacemaker cells are small, have multiple connections with their neighbors, and are entangled in a dense connective tissue stroma. These anatomical features undoubtedly contribute to slow conduction in the sinus node. In addition, the sinus node is surrounded by a group of cells which are intermediate in time-voltage course between S-A nodal cells and atrial specialized cells. These cells, called "perinodal fibers" are not automatic under normal circumstances but do have electrophysiologic properties which promote slow conduction and block; "perinodal cells" represent a barrier to conduction into the S-A node and a region where "organization" and amplification of impulses leaving the S-A node might occur.

All of the events discussed above—impulse generation in the S-A node and its transmission to the atrium—are invisible both on the body surface electrocardiogram and in local extracellular atrial electrograms. Analysis of the behavior of the S-A node in man is even more complicated than analysis of the A-V node. We have been able to study the A-V node in man by recording local electrograms from the atrial margin of the A-V node and, on the other side, the nearby bundle of His. Programmed stimulation of the atria or ventricles and analysis of the electrical responses allow a rather complete characterization of the A-V node. The S-A node is somewhat analogous to the A-V node in that the impulse generated in the S-A node must pass through the "perinodal fiber" (analogous to the A-V node) in order to reach specialized atrial fibers (analogous to the bundle of His). However, this analogy is very incomplete in that the S-A node itself is an area of slow conduction and, in addition, spontaneously generates impulses.

Experimental and clinical observations made in the first decade of the twentieth century established that second degree S-A block could occur in animal and human hearts. In fact, second degree sinoatrial block was well established clinically from analysis of the jugular venous pulse well before this abnormality was recorded electrocardiographically. Since that time there has been a great increase in our knowledge of the electrocardiographic features of second degree S-A block. More recently, the use of electrical pacemakers as a therapeutic device has led to an increased interest in and understanding of a variety of clinical patterns of S-A nodal dysfunction. These clinical patterns include:

1. severe sinus bradycardia, not induced by drugs or inappropriately severe for the type and amount of drug administered.
2. periods of second degree S-A block, inappropriate for drug therapy.
3. long pauses in sinoatrial rhythm caused by sinus arrest, repetitive concealed sinus exit block or third degree S-A exit block.
4. chronic atrial fibrillation with a slow ventricular rate in the absence of drugs which slow A-V conduction, and inability of the heart to resume stable sinus rhythm after electrical cardioversion.
5. the tachycardia-bradycardia syndromes.

Several features of these syndromes deserve comment. The first three listed have been recognized for a long time and if accompanied by heart failure or central nervous system symptoms are often treated, and successfully, with implanted electrical pacemakers. For a long time we recognized that patients with atrial fibrillation who had a slow ventricular rate without drug treatment were prone to develop very slow ventricular rates when treated with digitalis and often had severe sinus bradycardia, sinus pauses and other rhythms of sinus dysfunction when cardioverted. Recently, we have learned that this is due to the fact that many patients with severe S-A node dysfunction also have impaired A-V conduction and sluggish ventricular pacemakers. The tachycardia-bradycardia syndromes (fig. 1) have been recognized for about twenty years, but they presented difficult, often insurmountable, management problems until combined treatment with drugs and an electrical pacemaker became available.

It is easy to recognize S-A nodal dysfunction when it presents as one of the five syndromes listed above. However, it can be difficult to know whether S-A node dysfunction is present. Two examples which present clinical difficulty are: 1) moderate sinus bradycardia which may or may not indicate intrinsic malfunction of the S-A node and portend a series of difficult rhythm problems and their

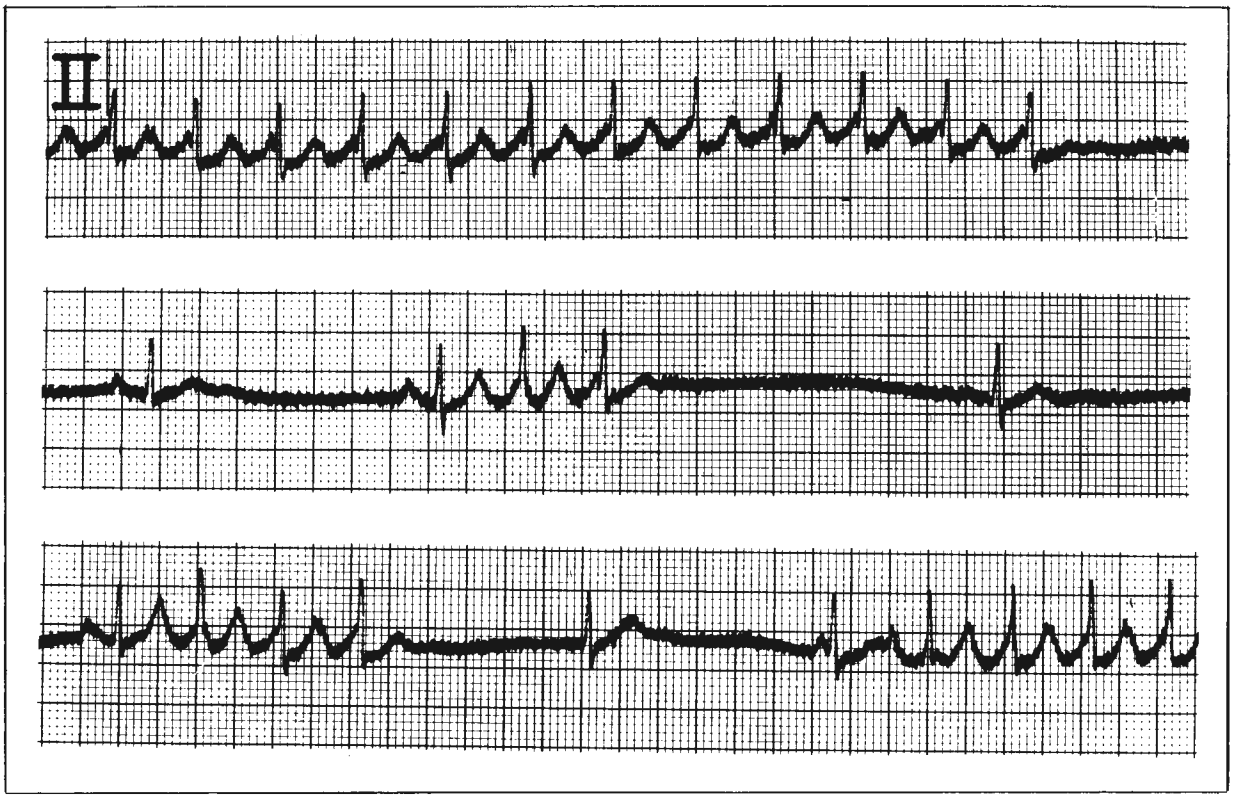

Fig. 1—The tachycardia-bradycardia syndrome. A 67-year-old man referred for evaluation of recurrent syncope, dizziness, and weakness. Lead II of his admission electrocardiogram shows a repeated change in rhythm from atrial flutter to sinus bradycardia. He was treated with a permanent transvenous pacemaker and digitalis and has been symptom-free for more than a year.

sequelae and 2) drug-induced sinus bradycardia or S-A block which improves to normal or near-normal when the inducing drug is removed. We need clinical applicable tests to evaluate patients who demonstrate such events. Ideally, these tests would separate patients with intrinsic S-A nodal dysfunction who require careful follow-up observation and have a high probability of need for early therapeutic intervention from those who merely have a slow sinus rate or those in whom a combination of extrinsic factors caused a temporary impairment in function of an essentially normal S-A node. Recently, two techniques have been used in the attempt to evaluate sinus node function in man: 1) rapid atrial pacing and 2) premature atrial stimulation. We will discuss briefly the use of these techniques in analyzing S-A nodal function.

Rapid Atrial Pacing. Out of a group of patients with syncopal attacks who presented to the National Heart Hospital in England, four were noted to have periods of sinus bradycardia alternating with periods of atrial tachyarrhythmias. In these patients the sinus rate usually ranged between 22 and 50 per minute. The episodes of atrial tachyarrhythmias were of variable duration, and in one patient, syncope associated with the termination of the tachyarrhythmia was documented. The episodes of syncope in these patients were due to a long period of cardiac standstill that followed the sudden termination of the atrial tachyarrhythmia (fig. 1). The extra long pauses that followed the termination of the tachyarrhythmia were a manifestation of depressed sinus node automaticity. That this was the case is suggested by the effects of quinidine hydrochloride on the sinus rate, that is, atrial standstill was observed in all four patients. Recent experimental studies reporting on the sinus node response to atrial pacing have obtained data that is somewhat analogous to the clinical observations on the sinus node response following sudden termination of an atrial tachyarrhythmia. These reports also have speculated on the ability of this technique to

determine sinus node automaticity in patients with and without evidence of sinus node dysfunction. The technique consists of pacing the atria at rates ranging between a rate slightly in excess of the spontaneous sinus rate and 170 per minute. The duration of the pacing period has ranged from 15 seconds to 5 minutes in different studies. The sinus escape interval is determined by measuring the interval between the last paced P wave and the first spontaneously occurring sinus P wave. The sinus escape interval is dependent upon the rate of atrial pacing and the spontaneous sinus heart rate prior to pacing. The longest sinus escape interval usually occurs at a rate near 130 per minute (fig. 2) and, in general, the slower the spontaneous heart rate, the longer the sinus escape interval after atrial pacing is discontinued. Varying the duration of atrial pacing from 15 to 180 seconds has little effect on the sinus escape interval, so that pacing for one minute is sufficient when measuring the sinus escape interval.

Rapid atrial pacing is most useful in evaluating a patient with syncope and sinus bradycardia. If

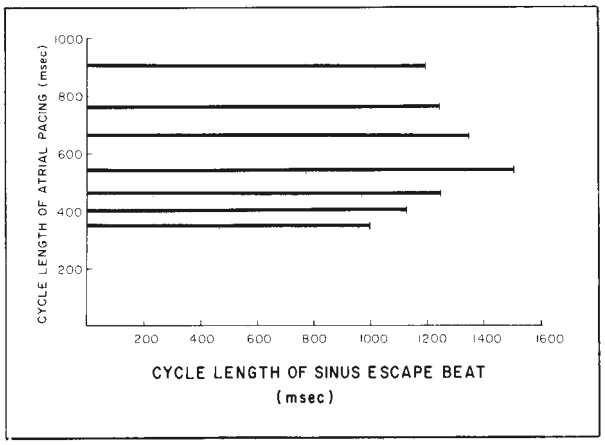

Fig. 2—The effect of atrial pacing on sinus escape interval. The patient's mean spontaneous sinus cycle length was 1050 msec (57/min). The right atrium was paced at each cycle length for 60 seconds and the interval between the last paced P wave and the first sinus P wave measured (plotted on the abscissa). As the paced atrial cycle length shortened, the sinus escape interval lengthened to a maximum of 1510 msec at a pacing cycle length of 540 msec (110/min). Thereafter, the cycle length of the sinus escape beat paradoxically decreased as the pacing rate increased. This finding suggests that, due to entrance block, the sinus node is actually being discharged more slowly at faster atrial pacing rates so that the decreasing cycle length of the sinus escape beat reflects the slower rate of sinus node discharge. The progressive shortening of the sinus escape interval as the rate of atrial pacing increases above 110/min probably reflects increasing degrees of sinoatrial entrance block.

prolonged sinus pauses are demonstrated, this condition suggests that sinus malfunction is responsible for the patient's syncope and pacemaker therapy is recommended. Also, one should not cardiovert patients with atrial fibrillation who have a history of either clinical sinus node dysfunction or a prolonged sinus escape interval unless a ventricular pacemaker is in place.

The normal sinus escape interval is not well-established, although values below 1.4 seconds have been called normal. However, the sinus escape interval is dependent on the basic sinus cycle length. Thus, in a young athlete, neither a heart rate of 43 per minute nor a sinus escape interval greater than 1.4 seconds need necessarily indicate sinus node dysfunction. Second, as was pointed out earlier, sinus node automaticity is regulated by autonomic nervous system tone. Thus, when sympathetic nervous system activity is increased, sinus node automaticity is enhanced. Since the sinus escape interval is determined largely by sinus node automaticity, a patient with sinus node dysfunction might not show a prolonged sinus node escape interval when his sympathetic nervous system activity is enhanced.

It must be emphasized that the ability of rapid atrial pacing to adequately assess sinus node automaticity is dependent upon 1:1 conduction from the atrium to the sinus node without excessive conduction delay between the atrium and sinus node. Should conduction from the atrium to the sinus node fail during atrial pacing, then the sinus node must be depolarized at a rate that is, in fact, much slower than the rate of atrial pacing. This entrance block could explain why the sinus node escape interval at 150 per minute was shorter than the escape interval at 130 per minute (fig. 2).

In patients with diseased sinoatrial nodes and sinoatrial junctional tissue, conduction from the sinus node to the atrium and from the atrium to the sinus node may be prolonged (fig. 6). In this circumstance, atrial pacing may fail to adequately assess sinus node automaticity since conduction from the atrium to the sinus node may become less than 1:1 even at very low pacing rates.

It is of interest that prolonged secondary sinus pauses are seen after discontinuing atrial pacing at 170 per minute (fig. 3). Secondary sinus pauses can recur many times during the first 20 seconds following termination of rapid atrial pacing. The duration of the secondary sinus pauses can even be greater than the duration of the sinus escape interval. This

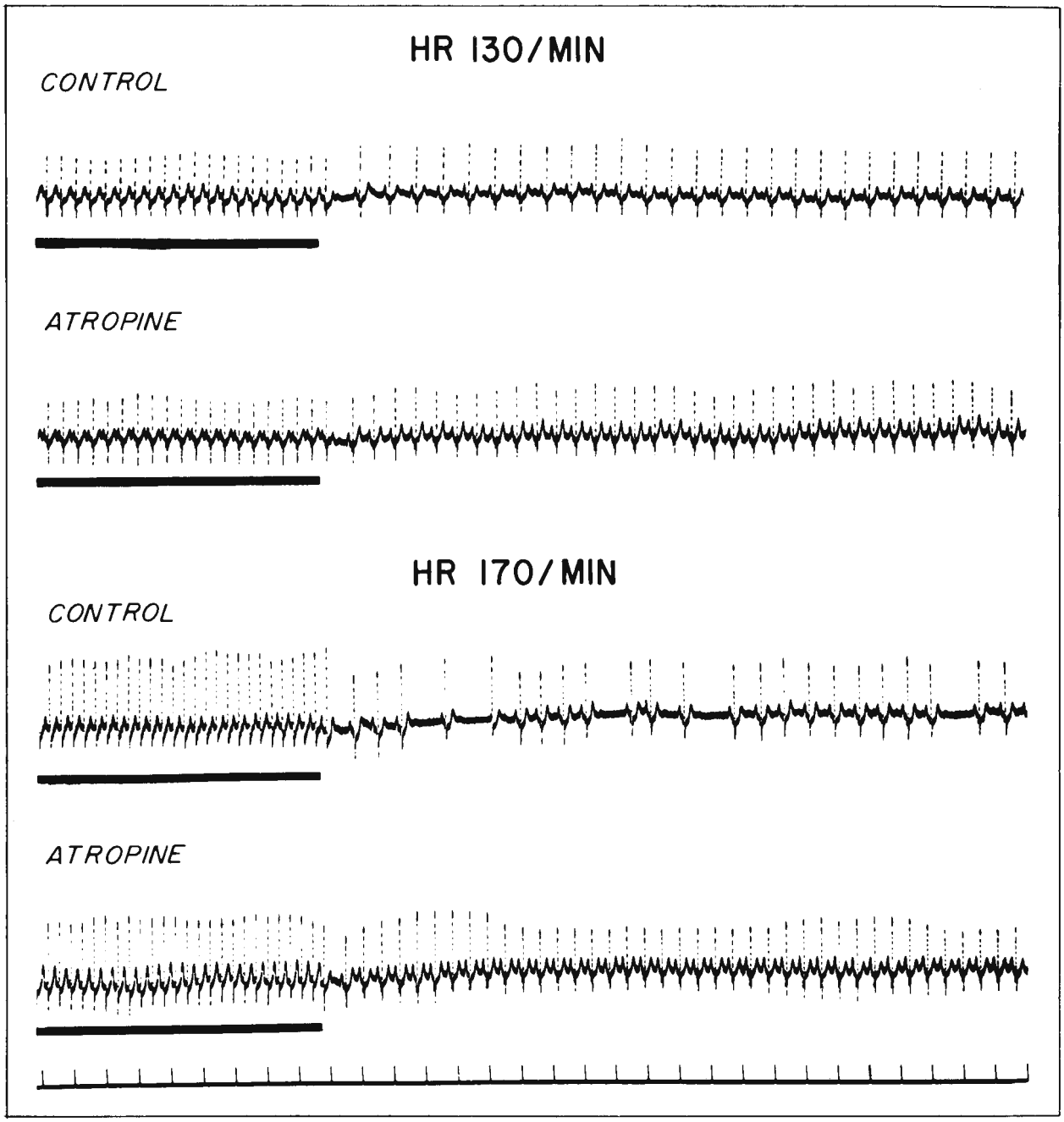

Fig. 3—Secondary sinus pauses occurring during the measurement of sinus escape interval. A 53-year-old woman with angina pectoris, and normal coronary arteriograms was referred for evaluation of sinus bradycardia. In this patient, the longest sinus escape interval was seen after an atrial pacing rate of 130/min. At a pacing rate of 170/min, the sinus escape beat interval was shorter than at 130/min, and prolonged secondary sinus pauses were noted following termination of rapid atrial pacing. These secondary sinus pauses may reflect pacemaker malfunction or advanced degrees of sinoatrial exit block. Atropine caused shortening of the sinus escape interval at all paced rates and abolished secondary sinus pauses. See text for discussion.

pattern of beating is compatible with repeated short periods of sinoatrial exit block (repetitive concealed exit block). Secondary sinus pauses seen after discontinuing atrial pacing are abolished by atropine, suggesting that they may be encouraged by cholinergic influences (fig. 3).

In patients with classic features of the "Sick Sinus Syndrome," sinus escape intervals as long as 5 seconds have been seen. Two mechanisms can be postulated to explain these extraordinarily long pauses. The first is that in the depressed sinus node, automaticity is particularly sensitive to overdrive suppression, and the long escape intervals reflect an extreme degree of sinus node pacemaker depression. The second possibility is that sinoatrial exit block is just more pronounced in these cases of advanced sinus node dysfunction than in milder forms (fig. 3). It is certainly reasonable that in cases of "Sick Sinus Syndrome" both mechanisms might operate together to produce the extremely long sinus pauses.

The episodes of sinoatrial entrance and exit block and concealed conduction at the junction between the sinus node and atrium are analogous to the better-known phenomena of A-V and V-A conduction block and concealed conduction in the *A-V junction.*

Premature Atrial Stimulation. A second technique which has been employed recently in evaluating S-A node function is that of premature atrial stimulation (PAS). We have performed PAS to evaluate S-A node function in the following manner. Two pairs of catheter electrodes are placed in the upper right atrium, near the junction of the superior vena cava and atrium. If A-V conduction is also to be evaluated, a third pair of electrodes is positioned over the bundle of His and used for recording. The high right atrial electrogram is used to trigger a counter during spontaneous rhythm so that a premature stimulus (S_2) can be delivered to the atrium during every seventh or eighth spontaneous cycle. A programmable stimulator is used so that the stimulus can be moved throughout the entire atrial cycle to elicit atrial premature depolarizations (APD or A_2). The following intervals are measured: 1) *the spontaneous sinus cycle* (A_1A_1), that is, the interval between the two spontaneous atrial depolarizations immediately preceding A_2, 2) *the test cycle* (A_1A_2), the interval between the atrial premature depolarization (A_2) and the immediately preceding spontaneous atrial depolarization (A_1), and 3) *the return cycle* (A_2A_3), that is, the interval between A_2 and the subsequent spontaneous atrial depolarization (A_3). In order to check the stability of atrial cycle length and evaluate the feasibility of normalizing, we measure the spontaneous atrial cycle immediately following the return cycle (A_3A_1). In order to compare results from different patients with a wide variety of different heart rates, we analyze the response to a series of stimuli which scan the atrial cycle by plotting the *normalized return cycle* (A_2A_3 per A_1A_1) as a function of the *normalized test cycle* (A_1A_2 per A_1A_1). Figure 4 shows such a plot. For purposes of discussion a typical plot can be divided into three zones.

Zone I. A_2's elicited late in atrial diastole are followed by a return cycle which is fully compensatory, that is, the sum of the test and return cycles approximately equals two spontaneous sinus cycles—$A_1A_2 + A_2A_3 = 2(A_1A_1)$. Typically, this response is seen in the terminal quarter of the spontaneous sinus cycle (0.75 to 1.00 of the cycle). Our postulated mechanism for this behavior is shown in figure 5A. The A_2 elicited by the electrical stimulus propagates toward the sinus node and collides with the emerging impulse which has spontaneously arisen in the sinus node. Since spontaneous activity in the sinus node has not been disturbed, the next spontaneous impulse arises in the sinus node and activates the atrium at the expected time.

Zone II. Typically, A_2's elicited in the middle half of atrial diastole (0.25-0.30 to 0.75 of the cycle) show a very different pattern. Despite the decreasing A_1A_2 per A_1A_1, the A_2A_3 per A_1A_1 cycle remains approximately constant. The A_2A_3 interval is less than compensatory but greater than

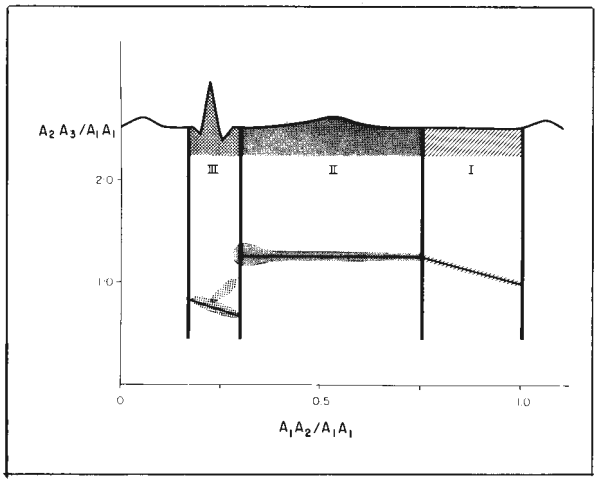

Fig. 4—The three zones of the atrial cycle revealed by premature atrial stimulation. Every seventh spontaneous atrial cycle, a stimulus (S_2) evoked a premature atrial depolarization. Here, the normalized return cycle (A_2A_3/A_1A_1) is plotted as a function of the normalized test cycle (A_1A_2/A_1A_1). The atrial zones are referenced to the electrocardiogram above. See text for detailed explanation.

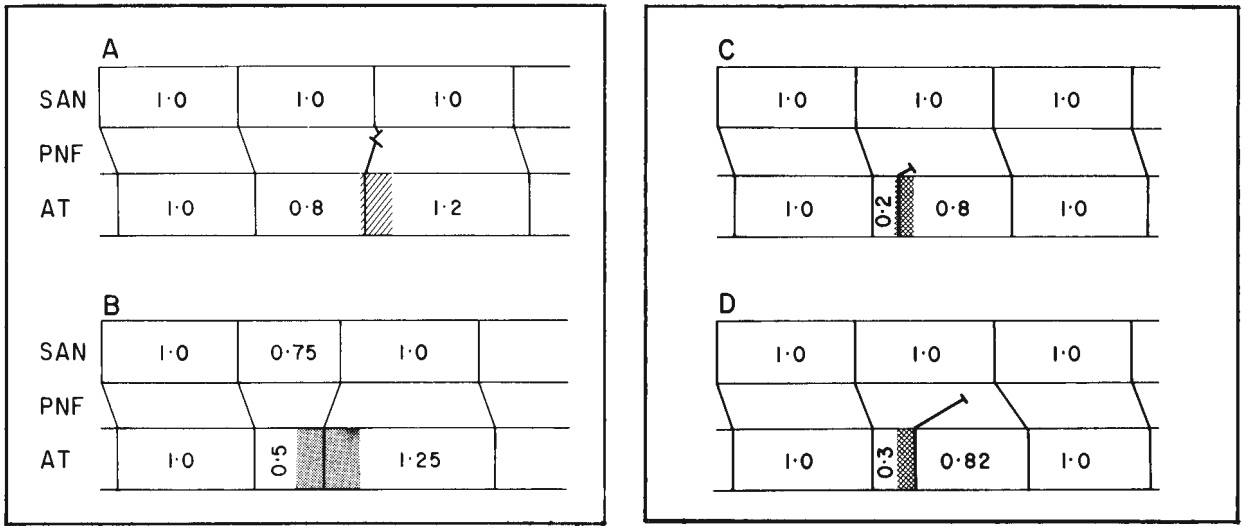

Fig. 5—Ladder diagrams depicting the events responsible for the response in Zones I, II, and III. SAN = sinoatrial node, PNF = peri(sinus)nodal fibers, AT = atrium.

A. Events in Zone I: non-reset of the sinus node

B. Events in Zone II: reset of the sinus node

C. Events in Zone III: non-reset of sinus node—interpolated APD

D. Events in Zone III: non-reset of sinus node—interpolated APD with concealed atrial-sinoatrial conduction causing delay of conduction of the subsequent sinus impulse to the atrium.

the A_1A_1 interval and is constant (fig. 4). Figure 5B shows the mechanism we postulate for this phenomena observed in Zone II. The S_2 evokes an A_2 which propagates across the junction between the S-A node and atrium to discharge the S-A node pacemaker before it spontaneously excites itself, that is, the S-A node pacemaker is reset. The pacemaker repolarizes and immediately begins to depolarize spontaneously; when threshold is reached, another S-A nodal action potential results, and this impulse propagates to the atrium producing A_3. Thus, three events contribute to the duration of the A_2A_3 interval: 1) the conduction time from atrium to S-A node pacemaker site (A_2-SAN_2), 2) the time to the next spontaneous S-A nodal action potential (SAN_2-SAN_3), and 3) the conduction time from the S-A nodal pacemaker to the atrium (SAN_3-A_3). The A_2A_3 interval remains almost constant throughout Zone II, indicating that the sum of these three events remains almost constant. If the spontaneous S-A node cycle following reset (SAN_2-SAN_3) is equal to the basic sinus cycle length (SAN_1-SAN_1), then the difference between A_2A_3 and the spontaneous atrial cycle (A_1A_1) represents the sum of conduction into and out of the S-A node—$(A_2A_3)-(A_1A_1) = (A_2\text{-}SAN_2) + (SAN_3\text{-}A_3)$.

Zone III. In some human hearts, the A_2A_3 interval remains constant until S_2 becomes so premature that no A_2 can be elicited (atrial refractory period is encountered). In others, a third zone may be encountered in which several phenomena may occur. The position of this zone varies from about 0.18-0.23 to 0.25-0.35 of the cycle. When the A_1A_2 interval is shortened to 0.3 of the A_1A_1 interval, the A_2A_3 interval may suddenly shorten from values of about 1.25 to about 0.70 of the A_1A_1 interval. This would indicate a true interpolated A_2, entirely analogous to the rarely observed phenomenon of spontaneous interpolated APD. Such an event indicates (fig. 5C) that the A_2 blocks in tissues around the S-A node; S-A discharge occurs on time and conducts normally to the atrium to produce A_3. For the SAN_3-A_3 conduction time to remain normal, the perinodal tissues must recover from the refractoriness engendered by the blocked A_2 before the SAN_3 impulse arrives. If the perisinus node zone is still refractory when SAN_3 propagates through this region on its way to the atrium, SAN_3-A_3 and A_2A_3 will be prolonged; A_2A_3 might be 0.8 to 0.9 of A_1A_1 at $A_1A_2 = 0.3$ rather than 0.7, the value expected if SAN_3-A_3 remains equal to SAN_1-A_1 (fig. 5D).

In general, the response in patients with normal

or near-normal sinoatrial conduction is much like that shown in figure 4. However, many other patterns are being encountered as sinus node function is evaluated in this way. It is possible to obtain useful information with premature atrial stimulation which is not obtainable in any other way. For example, analysis of the response to premature atrial stimulation can reveal *first degree* S-A block in man. Figure 6 diagrammatically shows normal and two degrees of conduction impairment between the S-A node and atrium. The normal case is illustrated by line A. The transition between Zones I and II comes when A_2A_3 is 1.25 of A_1A_1 and A_2A_3 remains constant as A_1A_2 is shortened further. As mentioned above, neglecting changes in spontaneous S-A node cycle length, the difference between A_2A_3 and A_1A_1 represents the sum of conduction into (A_2-SAN_2) and out of (SAN_3-A_3) the S-A node. In this example with a cycle length of 1,000 msec, the total conduction time—(A_2-SAN_2) + (SAN_3-A_3)—would be 250 msec ([1.25 − 1.00] = × 1000). Line B in figure 6 shows the transition between Zones I and II at 0.4 the spontaneous atrial cycle length. In this instance, total conduction time would be 600 msec—

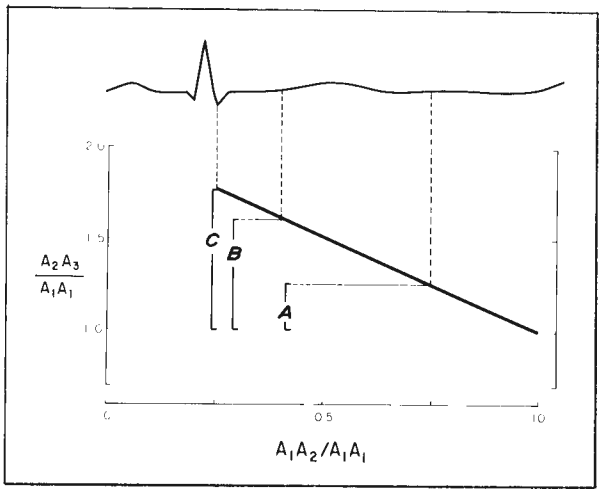

Fig. 6—The diagnosis of first-degree sinoatrial block in man by premature atrial stimulation. The abscissa is the normalized test cycle (A_1A_2/A_1A_1), the ordinate the normalized return cycle (A_2A_3/A_1A_1). The heavy diagonal line represents responses without reset (Zone I). Line A shows the transition from Zone I to Zone II in the usual portion of the cycle. Line B shows transition from Zone I to Zone II at 0.4 of the cycle indicating marked sinoatrial conduction delay. In C, a transition from Zone I to Zone II was never obtained, indicating either extreme sinoatrial conduction delay or, much less likely, unidirectional S-A block throughout the cycle.

($A_2A_3 - A_1A_1$) × 1000 = (1.6 − 1.0) × 1000 = 600 msec. If the conduction time in and out were equal then conduction time from sinus node to atrium would take 300 msec. Even though (SAN_1-A_1) and (SAN_3-A_3) are very probably not equal under such circumstances, (SAN_1-A_1) must be prolonged because A_1A_2 had to be shortened to 0.4 of the spontaneous cycle length before reset occurred, that is, before Zone II was encountered. This indicates that when the A_1A_2 interval was longer than 0.4, A_2 collided with the emerging sinus impulse; the fact that reset only occurs when A_2 is introduced early in the cycle surely indicates that first degree sinoatrial block is present (that SAN_1-A_1 is prolonged). Line C of figure 6 shows an even more extreme case of sinoatrial block. Throughout the entire cycle where responses could be elicited, A_1A_2 per A_1A_1 from 1.0 to 0.3, A_2A_3 was fully compensatory. This pattern of response indicates that A_2 never reached the S-A node pacemaker to reset it; therefore, the conduction time between the S-A node and atrium must be very long or unidirectional block must be present. The total conduction time is 700 msec—($A_2A_3 - A_1A_1$) × 1000 = (1.7 − 1.0) × 1000 = 700 msec. Thus, premature atrial stimulation can be used to detect first degree sinoatrial block in man, a feat not possible with any other technique.

Also, a variety of behavior has been observed in the early part of Zone II. When A_2 is placed early in Zone II, for example, 0.30-0.40 of A_1A_1, A_2A_3 may depart from its usual constant value. If the perinodal tissues are more refractory than usual, an A_2 in this portion of the cycle may conduct into the S-A node with great delay (A_2-SAN_2 greatly prolonged); even if SAN_2-SAN_3 is constant, A_2A_3 will be prolonged in direct proportion to the increase in A_2-SAN_2. If the S-A node pacemaker is unstable, then the spontaneous cycle length of the sinoatrial node pacemaker may not recover immediately after being reset by A_2, that is, SAN_2-SAN_3 may prolong. Even if A_2-SAN_2 and SAN_3-A_3 are not increased, A_2A_3 will prolong. An increase in A_2A_3 caused either by changes in conduction or automaticity would cause early Zone II responses to curve upward and be readily apparent in a plot of A_2A_3 vs. A_1A_2. Finally, in Zone III, the effects of concealed conduction can be so marked as to cause A_2A_3 to vary from 0.7 to values exceeding 1.0.

Thus, it is apparent that there is a great deal that can be learned from analyzing the responses to atrial pacing or premature atrial stimulation. The

final role for these techniques in evaluating S-A node function will not be settled until they have been applied to a large group of patients and careful follow-up of these patients has been continued for a period of time sufficient to determine the prognostic value of the tests. However, we have every reason to hope that these tests which permit new insights into S-A node function will ultimately improve our ability to predict the course of patients with S-A node dysfunction. If they do, such tests will greatly improve our therapeutic management of the "Sick Sinus Syndromes."

PANEL DISCUSSION

Dr. Baird: Dr. Bigger, is atrial pacing of any value in stressing ventricular conduction in patients with bilateral bundle branch block?

Dr. Bigger: Do you mean rapid atrial pacing?

Dr. Baird: Yes.

Dr. Bigger: Very little, I think. In such cases, rapid atrial pacing usually produces block at the A-V node. It is more than useful to use premature atrial stimulation, a technique which often permits one to demonstrate transmission through the A-V node but block in the bundle branches, if a His bundle recording is also made. This is a better technique for demonstrating bundle branch disease. In A-V nodal disease, of course, you may get second degree A-V block at an unduly low rate during fixed rate atrial pacing.

Dr. Scherlag: As I think most people know, there are patients who show periods of complete heart block and then periods of sinus rhythm with normal conduction. Doctor (Onkar) Narula has alluded to the fact that these patients, after complete heart block and subsequent sinus rhythm, sometimes will show 1:1 conduction up to rates of 150 or 180 per minute without showing any evidence of fatigue in A-V conduction. My indication from his data is that atrial pacing is not a good way of assessing conduction defects in the His-Purkinje system, even with atropine. Doctor Narula feels that the use of premature beats might be a useful tool in assessing critical A-V conduction delays, particularly those in the His-Purkinje system, but I do not think the definitive data are as yet available.

Dr. Bigger: Premature atrial stimulation is also a better test to assess A-V conduction when marked left axis right bundle branch block is present, since you can actually measure the functional refractory period of the posterior division of the left bundle with premature atrial stimulation. Prediction of future functional performance is what we all would like, but I am afraid that is the pot of gold at the end of the rainbow. As we have emphasized so many times, the heart under test conditions may not relate to the heart under conditions not related to the test or predict the future. Much remains to be learned about the prognostic significance of functional testing.

Dr. Moe: Years ago I used to demonstrate this phenomenon to students in the open chest dog as an attempt, in effect, to estimate refractory period of the sinus node. If you deliver a premature atrial stimulus at a time when the S-A node is refractory, the node will not discharge. Thus, the next expected sinus beat arrives almost on schedule and is, therefore, an almost interpolated atrial beat. Later premature beats will, of course, discharge the pacemaker although with a delay attributable to delayed conduction. The sum of the test cycle plus the "return" cycle, when plotted against the duration of the test cycle, will exhibit a sharp break. I wonder if you ever saw this break in the human heart.

Dr. Bigger: Yes, we have seen that quite often.