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.
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
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 dura-
tion 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 spontan-
eously 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 discon-
tinued. 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
prolonged sinus pauses are demonstrated, this con-
dition 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 pro-
longed 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 inter-
vall is dependent on the basic sinus cycle length. Thus,
in a young athlete, neither a heart rate of 43 per min-
ute nor a sinus escape interval greater than 1.4 sec-
onds need necessarily indicate sinus node dysfunction.
Second, as was pointed out earlier, sinus node auto-
maticity is regulated by autonomic nervous system
tone. Thus, when sympathetic nervous system activ-
ity 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 auto-
maticity is dependent upon 1:1 conduction from the
atrium to the sinus node without excessive con-
duction 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 es-
cape 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 follow-
ning 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

\textbf{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 para-
doxically decreased as the pacing rate increased. This find-
ing 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 re-
fects increasing degrees of sinoatrial entrance block.
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 ($A_2$) 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...
the $A_3A_1$ 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—

$$ (A_2A_3)-(A_1A_1) = (A_2$-$SAN_2) + (SAN_3$-$A_3). $$

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 $A_3$, 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 (\( A_2\text{-SAN}_2 \)) and out of (\( \text{SAN}_3-A_2 \)) the S-A node. In this example with a cycle length of 1,000 msec, the total conduction time—\( (A_2\text{-SAN}_2) + (\text{SAN}_3-A_2) \)—would be 250 msec \((1.25 - 1.00) \times 1000\). 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—\( (A_2A_3 - A_1A_1) \times 1000 = (1.6 - 1.0) \times 1000 = 600 \) msec. If the conduction time in and out were equal then conduction time from sinus node to atrium would take 300 msec. Even though \( (\text{SAN}_1-A_1) \) and \( (\text{SAN}_3-A_2) \) are very probably not equal under such circumstances, \( (\text{SAN}_1-A_1) \) 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 \( \text{SAN}_1-A_1 \) 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—\( (A_2A_3 - A_1A_1) \times 1000 = (1.7 - 1.0) \times 1000 = 700 \) 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 (\( A_2\text{-SAN}_2 \) greatly prolonged); even if \( \text{SAN}_2\text{-SAN}_3 \) is constant, \( A_2A_3 \) will be prolonged in direct proportion to the increase in \( A_2\text{-SAN}_2 \). 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, \( \text{SAN}_2\text{-SAN}_3 \) may prolong. Even if \( A_2\text{-SAN}_2 \) and \( \text{SAN}_3-A_2 \) 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.