Comparative study of sinoatrial conduction time and sinus node recovery time¹

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Atrial stimulation studies were performed in 5 normal patients (group A) and 4 patients with electrocardiographic evidence of sinoatrial disease (group B). The technique of premature atrial stimulation was used to calculate sinoatrial conduction time. Rapid atrial pacing was applied to measure maximum sinus node recovery time. In 4 cases both stimulation methods were repeated after intravenous administration of atropine. Group A had a sinoatrial conduction time of $56 \text{ ms} \pm 11$ (SD) and a maximum sinus node recovery time of $1122 \text{ ms} \pm 158$. In 3 out of 4 patients with sinus node dysfunction a prolongation of sinoatrial conduction time could be demonstrated (145, 105, and 150 ms). In addition, one showed probable sinus node exit block after premature atrial stimulation. Sinus node recovery time was excessively prolonged in 2 (3880 and 3215 ms) and normal in the other 2 patients with sinoatrial disease (1330 and 1275 ms).

Atropine leads to a decrease of sinoatrial conduction time. Results indicate that sinus node recovery time may not be a reliable indicator of sinus node automaticity if sinoatrial conduction is disturbed. The premature atrial stimulation technique makes it possible to study the pattern of sinoatrial conduction and to evaluate its reaction to therapeutic drugs.

Analysis of atrial depolarization in the surface electrogram permits only an overall estimation of sinus node function. This involves the ability to act as an impulse generator and includes conduction of the impulse from the pacemaker site to the atrium (i.e. sinoatrial conduction).

Recently the method of rapid atrial stimulation was developed to measure sinus node recovery time which is thought to permit evaluation of sinus node automaticity (Rosen *et al.*, 1971; Mandel *et al.*, 1971). When sinus node recovery time was prolonged in patients with sinoatrial disease, this has been interpreted as a sign of a disturbed sinus node automaticity (Narula, Samet, and Javier, 1972; Mandel *et al.*, 1972). However, results obtained by this method are variable and not always conclusive (Rios *et al.*, 1972; Gupta *et al.*, 1974).

Administration of atropine has been shown to result in a decrease of sinus node recovery time (Mandel *et al.*, 1972). In other studies, however, an atropine-induced lengthening of sinus node recovery time was observed in single cases (Narula

et al., 1972; Bashour, Hemb, and Wickramesekaran, 1973).

In 1973, Strauss *et al.*, described a way of determining sinoatrial conduction time in man by use of the premature atrial stimulation technique. Thus, a differentiation between sinus node generator characteristics and sinoatrial conduction seems to be possible. In a previous publication a modification of this procedure for calculation of sinoatrial conduction time was described (Steinbeck, Körber, and Lüderitz, 1974). In the present study we report simultaneous determinations of sinoatrial conduction time and sinus node recovery time in 9 patients. Four of them were restudied after administration of atropine.

Our Results revealed a conspicuous prolongation of sinoatrial conduction time in 3 patients with sinoatrial disease. A disturbance of retrograde sinoatrial conduction during rapid atrial stimulation as well as a disturbance of antegrade sinoatrial conduction after cessation of atrial pacing may affect the apparent recovery interval of the sinus node. This reduces the significance of sinus node recovery time as an indicator of sinus node automaticity. Thus, for furthering our understanding of the underlying pathophysiological mechanisms in sinoatrial disease,

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both sinoatrial conduction time and sinus node recovery time should be measured.

Patients and methods

Nine patients were studied (see Table 1).

Group A (Cases 1–5) showed no signs of sinus node dysfunction. It included, however, one patient showing reflex slowing of sinus rate during massage of the carotid sinus (Case 2) and another with frequent premature ventricular extrasystoles (Case 5). Group B (Cases 6–9) had electrocardiographic evidence of sinoatrial disease with episodes of sinus rates less than 50/min. In addition to sinus bradycardia, supraventricular tachycardia (Cases 6 and 9), spontaneous second degree sinoatrial block (Case 7), and junctional escape rhythm after a period of atrial arrest (Case 8) were observed in these patients.

All patients gave informed, written consent for evaluation of sinus node function. They were studied in the non-sedated state and did not receive any cardioactive drugs unless stated otherwise. In all patients sinus rhythm was present during the study.

A quadripolar electrode catheter was inserted via the basilic vein and the tip placed at the lateral wall of the right atrium. The distal pair of electrodes was used for stimulation. From the proximal pair of electrodes a high right atrial electrogram was simultaneously recorded with a standard electrocardiogram. For rapid atrial stimulation and premature atrial stimulation the Tektronix Pulse Generator System Series 2600 was used. Rectangular stimuli of twice diastolic threshold and 2 ms in duration were passed through an isolation transformer (Tektronix Type 2620). For calculation of sinoatrial conduction time, single premature stimuli were delivered with a variable time delay after the last spontaneous atrial depolarization. The bipolar atrial electrogram was used as trigger signal, and the entire atrial cycle was scanned.

Definitions and measurements

Applying the premature atrial stimulation technique, the following time intervals were measured.

- 1) The interval between the last two spontaneous atrial depolarizations preceding the stimulation impulse (a_1 – a_2).
- 2) The interval between the last atrial depolarization and the stimulus-induced atrial excitation (so-called curtailed cycle a_2 – a_3).
- 3) The interval between the stimulus-induced atrial excitation and the next spontaneous atrial depolarization (so-called postextrasystolic cycle a_3 – a_4).

Premature beats elicited late in the spontaneous atrial cycle are followed by a fully compensatory pause (no pacemaker reset: a_2 – a_3 plus a_3 – a_4 is twice the a_1 – a_2 interval). Earlier premature beats are followed by a non-compensatory pause (pacemaker reset: a_2 – a_3 plus a_3 – a_4 is shorter than twice the a_1 – a_2 interval (see Wenckebach, 1903; Eccles and Hoff, 1934; Bonke, Bouman, and van Rijn, 1969; Strauss *et al.*, 1973)).

For calculation of sinoatrial conduction time we determined the postextrasystolic interval (a_3 – a_4) which

was exactly at the borderline between fully compensatory and non-compensatory (Steinbeck *et al.*, 1974). The a_2 – a_3 interval at this borderline represents the shortest premature beat interval whose retrograde excitation front does not reach the sinus node, whereas earlier premature beats reset the pacemaker and may depress it (Bonke *et al.*, 1969; Klein, Singer, and Hoffman, 1973).

Therefore, the postextrasystolic interval a_3 – a_4 at this borderline, minus the spontaneous atrial cycle a_1 – a_2 , is equivalent to the sum of conduction time from atrium to the sinus node plus from sinus node to atrium. Half of the total sum of conduction time gives the sinoatrial conduction time.

The sinus node recovery time is defined as the time interval between the last paced atrial activation and the first spontaneous beat of sinus origin after cessation of rapid atrial stimulation. Sinus node recovery time was measured after stimulation with frequencies above sinus rate. The pacing rate was then increased by increments of 10 or 20 beats/min. Each pacing period lasted one minute. The maximum sinus node recovery time is the longest time interval seen at any of the stimulation rates.

Results

The results of atrial stimulation studies are listed in Table 1.

Sinoatrial conduction time

Group A patients had sinoatrial conduction times of $56 \text{ ms} \pm 11$ (mean $\pm$ SD), which are considered normal (Steinbeck *et al.*, 1974). Of 4 group B patients, 3 had a conspicuous prolongation of sinoatrial conduction time.

The results of premature atrial stimulation in Case 6 are shown in Fig. 1. When single beats are elicited after an interval equal to between 70 and 100 per cent of the preceding atrial cycle a_1 – a_2 , the sum of the a_2 – a_3 and a_3 – a_4 interval is about twice the a_1 – a_2 cycle. Thus, these data agree with the theoretical line indicating fully compensatory pauses. If the curtailed cycles are shortened further, they are followed by non-compensatory pauses, as indicated by the data below the theoretical line of the compensatory pause. From this a sinoatrial conduction time of 145 ms is calculated.

Beats introduced early in atrial diastole are followed by abruptly shortened postextrasystolic intervals. The postextrasystolic atrial depolarization starts at the moment where it would have been expected if the premature beat had not occurred. The sum of the curtailed cycle a_2 – a_3 and postextrasystolic cycle a_3 – a_4 is equal to the preceding a_1 – a_2 interval indicated by the data close to the theoretical broken line (Fig. 1). This type of response is highly suggestive of sinus node entrance block of the premature beat.

In Case 7 a sinoatrial conduction time of 105 ms

TABLE I Results of atrial stimulation studies in 9 patients

Case no.	Age (yr) and sex	Arrhythmias	Sinus rate (per min)	Sinoatrial conduction time (ms)	Maximum sinus node recovery time (ms)
Group A					
1	27 F	—	96	60	1050
2	50 M	Carotid sinus hypersensitivity	96	40	895
3	51 M	—	65	50	1205
4	33 F	—	64	60	1310
5	66 F	Premature ventricular extrasystoles	85	70	1150
Group B					
6	57 M	Sinus bradycardia, supraventricular tachycardia	62	145	1330
7	61 F	Sinus bradycardia, sinoatrial block	57	105 Sinoatrial exit block	3880
8	69 F	Sinus bradycardia, junctional escape rhythm	60	150	3215
9	50 F	Sinus bradycardia, premature ventricular extrasystoles, supraventricular tachycardia	69	*	1275

* Exact calculation of sinoatrial conduction time was not possible because of sinus arrhythmia.

was determined (Fig. 2). Moreover, some impulses are followed by postextrasystolic intervals exceeding the other intervals by one basic cycle length. The escape beat was always of sinus origin and the following atrial cycle lengths were in the range of prepacing values. This indicates that an intermittent sinoatrial exit block after single premature beats is present as well as a prolongation of sinoatrial conduction time.

Case 8 had the longest sinoatrial conduction time (150 ms) seen in any patients.

In Case 9 a considerable sinus arrhythmia did not allow an exact determination of the borderline between compensatory and non-compensatory pauses or, therefore, calculation of sinoatrial conduction time.

Sinus node recovery time

Values of maximum sinus node recovery time in group A are normal (Table 1) (Narula *et al.*, 1972). From Case 4, 10 successive atrial cycle lengths after discontinuation of rapid atrial pacing at a frequency of 70, 90, 110 and 150 per minute are plotted in Fig. 3A. The interval from the last paced beat to the first beat of sinus origin is always the longest interval. Thereafter the cycle lengths gradually return to normal prepacing values, with some slightly decreased intervals (after pacing

at 90 and 110/min). All other patients of group A showed a similar pattern of successive atrial cycle lengths after rapid atrial stimulation.

Two patients of group B showed a normal maximum sinus node recovery time (Cases 6 and 9); the other 2 patients had a conspicuous prolongation of maximum sinus node recovery time (Cases 7 and 8) (see Table 1).

From Case 8, 10 successive atrial cycle lengths after cessation of rapid atrial stimulation are plotted in Fig. 3B. The first 3 atrial cycles after pacing at 90/min are prolonged by one basic cycle length. The next 3 intervals are abruptly shortened, being identical to normal prepacing atrial cycles. The seventh a-a interval is also twice the basic cycle length, the following cycles being only slightly longer than normal. From this it can be concluded that the first three and the seventh beat-to-beat interval after cessation of atrial pacing at 90/min are determined by a sinoatrial exit block with 2:1 atrial response, in addition to depression of the sinus node pacemaker. Sinoatrial exit block with 3:1 atrial response is superimposed on the value of 'sinus node recovery time' after cessation of pacing at 110/min. An intermittent sinoatrial exit block is present after cessation of atrial pacing at 140/min, not during the first beats but in the fourth and fifth atrial cycles. This is indicated by a sudden doubling of the atrial cycle length.

FIG. 1 Postextrasystolic cycle a_3-a_4 as a function of the curtailed cycle a_2-a_3 (Case 6). Both values are related to the preceding a_1-a_2 interval: a_1-a_2 interval = $963 \text{ ms} \pm 54$ ($n=19$). Transition from compensatory to non-compensatory a_3-a_4 interval occurs at a curtailed cycle of about 70 per cent of the a_1-a_2 interval. Postextrasystolic interval a_3-a_4 at this borderline ($=130\%$), minus the spontaneous atrial cycle a_1-a_2 ($=100\%$), represents conduction time into and out of the sinus node: sinoatrial conduction time = 145 ms . Early premature beats are followed by abruptly shortened postextrasystolic intervals. This is highly suggestive of sinus node entrance block of the impulse, because a_3 demonstrated no increase in latency, the configuration of the postextrasystolic depolarizations was unchanged, and the subsequent sinus rate was not altered (Goldreyer and Damato, 1971).

Similarly abrupt changes of atrial cycle length were observed in Case 7 after cessation of rapid atrial stimulation as in the patient above. In this patient also a sinoatrial exit block was assumed to be present after premature atrial stimulation. These abrupt changes did not occur in Cases 6 and 9 or in any of the group A patients.

Atropine Two patients from each group were restudied 5 minutes after intravenous administration of 1 mg atropine (Table 2).

Sinoatrial conduction time was decreased in Cases 1, 3, and 6 (Fig. 4). The transition from compensatory to non-compensatory postextrasystolic pauses takes place at an a_2-a_3 interval of 85 per cent of the a_1-a_2 cycle. From this a sinoatrial conduction time of 55 ms is calculated. Further shortening of the curtailed cycle within the range of non-compensatory pause results in a continuous

FIG. 2 Co-ordinates as in Fig. 1 (Case 7). a_1-a_2 interval = $1050 \text{ ms} \pm 50$ ($n=46$). The borderline between compensatory and non-compensatory intervals a_3-a_4 is at about 80 per cent of a_1-a_2 : sinoatrial conduction time = 105 ms . Single premature beats are followed by prolonged postextrasystolic intervals. For further explanation see text.

prolongation of the postextrasystolic cycle. A sinus node entrance block was not observed after early premature beats. In Case 9 sinus rhythm was regular after atropine, and a sinoatrial conduction time of 45 ms could be determined.

Atropine diminished maximum sinus node recovery time in both patients in each group (Table 2). 'Corrected maximum sinus node recovery time' (i.e. the maximum 'sinus node recovery time' in excess of the preceding sinus cycle length) was reduced by 325 ms in Case 1, and by 238 ms in Case 9; it diminished only slightly in Case 3 (by 19 ms), and in Case 6 (by 23 ms).

The values of sinus node recovery time from Case 3 are shown after various stimulation frequencies before and after atropine (Fig. 5A). A positive relation between sinus node recovery time and pacing rate is not apparent; maximum sinus node recovery time is achieved at $130/\text{min}$ before and after drug administration. Atropine decreased sinus node recovery time at any pacing rate at which it was tested. Similar results were obtained in Case 1, except that atropine raised the pacing rate giving rise to maximum sinus node recovery time from 120 to $150/\text{min}$.

Fig. 5B gives the results of Case 6 (group B). Maximum sinus node recovery time is observed after atrial pacing at a frequency of $80/\text{min}$. Periods of more frequent stimulation up to $130/\text{min}$ are followed by a shorter sinus node recovery

FIG. 3 *A*) Ten successive atrial cycle lengths after rapid atrial pacing at 70, 90, 110, and 150/min (Case 4). Average sinus cycle before stimulation: 940 ms (see text). *B*) (Case 8). Stimulation rates: 80, 90, 110, and 140/min. Average sinus cycle before stimulation: 995 ms. After the pacing periods of 90, 110, and 140/min considerable changes in atrial cycle lengths are observed, which differ exactly by one basic a-a interval. For further discussion, see text.

TABLE 2 Atrial stimulation after 1 mg intravenous atropine

Case no.	Sinus rate /min	Sinoatrial conduction time (ms)	Maximum sinus node recovery time (ms)
1	120	25	600
3	95	35	895
6	83	55	1070
9	83	45	900

time. The pacing rate could not be raised above 130/min because of the onset of angina pectoris. After administration of atropine the pacing rate causing maximum sinus node recovery time is increased to 120/min. After stimulation at 130/min, sinus node recovery time is longer after administration of atropine than before.

Discussion

It is the purpose of this study to demonstrate the advantage of simultaneous measurement of sino-

FIG. 4 Same patient as in Fig. 1 after admission of atropine 1 mg intravenously. Co-ordinates as in Fig. 1. a_1-a_2 interval = $726 \text{ ms} \pm 14$ ($n=32$). Atropine leads to a decrease of sinoatrial conduction time (55 ms).

atrial conduction time and sinus node recovery time for the detailed analysis of the pathophysiological conditions underlying sinoatrial disease.

In 1973, Strauss *et al.*, applying the premature atrial stimulation technique, first reported a way of determining sinoatrial conduction time in man.

We used a modification of this procedure, which

FIG. 5 A) Sinus node recovery time (ms) after rapid atrial stimulation with various frequencies before and after atropine 1 mg intravenously (Case 3). Sinus rate is 65/min, after atropine 95/min. After pacing had been stopped at 80 and 100/min (before atropine) a premature atrial extrasystole occurred first so that sinus node recovery time could not be measured. B) Coordinates as in Fig. 5A (Case 6). Sinus rate is 62/min, after atropine 83/min. For further explanations see text.

avoids the possibility of sinus node depression after premature beats influencing calculation of sinoatrial conduction time (Steinbeck *et al.*, 1974). On the other hand, this method of calculation may lead to an underestimate of the true sinoatrial conduction time (Miller and Strauss, 1974). Anterograde and retrograde sinoatrial conduction times may not be identical. Sinoatrial conduction time is, however, half of the sum of conduction time from the site of electrodes used for atrial bipolar electrogram into the sinus node and back from the sinus node to the recording electrodes. Nevertheless, the value of $56 \text{ ms} \pm 11$ in 5 patients without evidence of sinoatrial disease agrees with experimental results derived from transmembrane action potential measurements in a spontaneously beating preparation of the isolated right atrium of the rabbit (West, 1955; Hoffman and Cranefield, 1960; Sano and Yamagishi, 1965; Strauss and Bigger, 1972).

The method of rapid atrial stimulation has been applied to measure sinus node recovery time, which is thought to permit evaluation of sinus node automaticity (Rosen *et al.*, 1971; Mandel *et al.*, 1971). However, two conditions have to be fulfilled if observations made with overdrive suppression are to provide a valid measure of sinus node automaticity.

- 1) Successful retrograde sinoatrial conduction of the impulse delivered to the right atrium.
- 2) Successful antegrade sinoatrial conduction immediately after the period of atrial pacing (Strauss and Bigger, 1973).

Our study of 5 patients without any signs of

abnormal sinus node function (group A) revealed a normal sinoatrial conduction time as well as a normal sinus node recovery time.

Different results were obtained in group B. Four patients with electrocardiographic evidence of sinoatrial disease were analysed. Two (Cases 7 and 8) showed an excessively prolonged sinus node recovery time. With the results of premature atrial stimulation in these patients in mind, we think it unlikely that prolongation of sinus node recovery time was brought about by severe sinus node depression alone. Both patients showed a very prolonged sinoatrial conduction time. In addition, one of them showed intermittent sinoatrial exit block after single premature beats as a sign of a more severe impairment of sinoatrial conduction. After cessation of rapid atrial pacing, abrupt changes of atrial cycle length from one beat to another were observed, in both patients, differing by one basic interval. This pattern of abruptly changing successive atrial cycle lengths after the period of atrial pacing was never seen in the 5 patients with normal sinoatrial conduction time. We, therefore, suppose that impairment of antegrade sinoatrial conduction, after cessation of rapid atrial stimulation, resulting in sinoatrial exit block of various degrees, was superimposed on sinus node depression in the 2 patients with apparently very prolonged sinus node recovery times (Cases 7 and 8).

Case 6 with evidence of sinoatrial disease demonstrated a prolonged sinoatrial conduction time in the presence of a normal maximum sinus node recovery time. Atropine, 1 mg intravenously, reduced sino-

atrial conduction time as well as maximum sinus node recovery time. After atropine, however, the pacing rate causing maximum sinus node recovery time was raised from 80 to 120/min. At a stimulation frequency of 130/min, the recovery time was longer after atropine than before (see Fig. 5B).

On the basis of the premature atrial stimulation studies (Fig. 1 and 4) we interpret the results of rapid atrial stimulation in this patient as follows: Sinoatrial conduction is grossly impaired. At low frequencies a maximum of impulses is conducted from the atrium to the sinus node and causes maximum sinus node recovery time. At higher stimulation rates it is assumed that impairment of sinoatrial conduction increases; as a consequence, fewer impulses penetrate to discharge the sinus node pacemaker thus leading to an apparently paradoxical shortening of sinus node recovery time. This explains the normal value of maximum sinus node recovery time which would probably be prolonged in the presence of an adequate retrograde sinoatrial conduction. Atropine is thought to improve retrograde sinoatrial conduction. As a result, maximum sinus node recovery time is observed after a considerably increased pacing frequency, and sinus node recovery time at 130/min is longer after atropine than before.

Our results indicate that sinus node recovery time may not be a reliable indicator of sinus node automaticity if sinoatrial conduction is disturbed. It seems that sinus node entrance block during rapid atrial stimulation leads to a shortening, and sinus node exit block after rapid atrial pacing, to a prolongation of the sinus node recovery time. These mechanisms offer an explanation for the observation that patients with sinoatrial disease may sometimes show greatly prolonged or normal sinus node recovery times besides moderate prolongation of sinus node recovery time after rapid atrial stimulation. Results may also be obscured by the occurrence of sinus nodal re-entry beats after atrial pacing (Narula, 1975).

The premature atrial stimulation technique provides a means of studying the pattern of sinoatrial conduction and evaluating its reaction to drugs.

References

- Bashour, T., Hemb, R., and Wickramasekaran, R. (1973). An unusual effect of atropine on overdrive suppression. *Circulation*, **48**, 911.
- Bonke, F. I. M., Bouman, L. N., and van Rijn, H. E. (1969). Change of cardiac rhythm in the rabbit after an atrial premature beat. *Circulation Research*, **24**, 533.
- Eccles, J. C., and Hoff, H. E. (1934). The rhythm of the heart beat. II - Disturbances of rhythm produced by late premature beats. *Proceedings of the Royal Society, London, Series B*, **115**, 327.
- Goldreyer, B. N., and Damato, A. N. (1971). Sinoatrial-node entrance block. *Circulation*, **44**, 789.
- Gupta, P. K., Lichstein, E., Chadda, K. D., and Badui, E. (1974). Appraisal of sinus nodal recovery time in patients with sick sinus syndrome. *American Journal of Cardiology*, **34**, 265.
- Hoffman, B. F., and Cranefield, P. F. (1960). *Electrophysiology of the Heart*. McGraw-Hill, New York.
- Klein, H. O., Singer, D. H., and Hoffman, B. F. (1973). Effects of atrial premature systoles on sinus rhythm in the rabbit. *Circulation Research*, **32**, 480.
- Mandel, W. J., Hayakawa, H., Allen, H. N., Danzig, R., and Kermaier, A. I. (1972). Assessment of sinus node function in patients with the sick sinus syndrome. *Circulation*, **46**, 761.
- Mandel, W. J., Hayakawa, H., Danzig, R., and Marcus, H. S. (1971). Evaluation of sino-atrial node function in man by overdrive suppression. *Circulation*, **44**, 59.
- Miller, H. C., and Strauss, H. C. (1974). Measurement of sinoatrial conduction time by premature atrial stimulation in the rabbit. *Circulation Research*, **35**, 935.
- Narula, O. S. (1975). Sinus node re-entry. *Circulation*, **50**, 1114.
- Narula, O. S., Samet, P., and Javier, R. P. (1972). Significance of the sinus-node recovery time. *Circulation*, **45**, 140.
- Rios, J. C., Bashour, T., Cheng, T. O., and Motomiya, T. (1972). Atrial pacing in sick sinus node syndrome (abstract). *Circulation*, **45** and **46**, Suppl. II, 211.
- Rosen, K. M., Loeb, H. S., Sinno, M. Z., Rahimtoola, S. H., and Gunnar, R. M. (1971). Cardiac conduction in patients with symptomatic sinus node disease. *Circulation*, **43**, 836.
- Sano, T., and Yamagishi, S. (1965). Spread of excitation from the sinus node. *Circulation Research*, **16**, 423.
- Steinbeck, G., Körber, H.-J., and Lüderitz, B. (1974). Die Bestimmung der sinuatrialen Leitungszeit beim Menschen durch gekoppelte atriale Einzelstimulation. *Klinische Wochenschrift*, **52**, 1151.
- Strauss, H. C., and Bigger, J. T., Jr. (1972). Electrophysiological properties of the rabbit sinoatrial perinodal fibers. *Circulation Research*, **31**, 490.
- Strauss, H. C., and Bigger, J. T., Jr. (1973). The authors reply. An unusual effect of atropine on overdrive suppression. *Circulation*, **48**, 911.
- Strauss, H. C., Saroff, A. L., Bigger, J. T., Jr., and Giardina, E. G. V. (1973). Premature atrial stimulation as a key to the understanding of sinoatrial conduction in man. *Circulation*, **47**, 86.
- Wenckebach, K. F. (1903). Ueber die Dauer der compensatorischen Pause nach Reizung der Vorkammer Säugethierherzens. *Archiv für Physiologie (Leipzig)*, p. 57.
- West, T. C. (1955). Ultramicroelectrode recording from the cardiac pacemaker. *Journal of Pharmacology and Experimental Therapeutics*, **115**, 283.
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