Clinical Significance of Prolonged Sinoatrial Conduction Time

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SUMMARY Prolonged (>152 msec) calculated sinoatrial conduction times (SACT) were found in 24 of 470 patients studied by the atrial extrastimulus technique, ranging from 155 to 220 msec (180 ± 4.4; mean ± SEM). There were 18 males and six females with ages of 29 to 85 (mean 65 ± 2.6). Electrocardiographic monitoring revealed significant sinus or atrial dysrhythmias in 19 (79%) patients. Of these 19, 15 had persistent sinus bradycardia and/or sinoatrial block, three had sinus bradyarrhythmia with paroxysmal atrial tachycardia, and one had isolated atrial tachycardia. Additional electrophysiological evidence of sinus node or atrial dysfunction was present in 11 patients. Four patients needed permanent pacing during follow-up (mean follow-up period of 427 ± 39 days) because of symptomatic bradyarrhythmia. Three patients died, none suddenly.

In conclusion, prolonged calculated SACT was associated with a high incidence of electrocardiographic and electrophysiologic abnormalities of sinus node and/or atrium. Despite this, bradyarrhythmic morbidity was relatively low, suggesting that prolonged sinoatrial conduction time in the absence of symptoms is not an indication for prophylactic pacing.

IN 1973, STRAUSS AND CO-WORKERS reported a method for indirect calculation of sinoatrial conduction time, utilizing the timing of return responses following reset of the sinus node with atrial extrastimuli. They reported calculated sinoatrial conduction times of 68 to 156 msec in four patients with sinus node disease. In one additional patient, they noted total inability to reset the sinus node (zone I response), a phenomenon termed as first degree sinoatrial block with interference because of presumed perinodal refractoriness. Utilizing the method of Strauss et al., our laboratory has reported calculated sinoatrial conduction times in 36 patients without apparent sinus node disease ranging from 40 to 153 msec.

Although it has been reported that patients with sinus node dysfunction frequently have longer sinoatrial conduction times than patients without sinus node disease, the clinical, electrocardiographic and electrophysiological significance of prolonged sinoatrial conduction time in man is not known. In the present study, we have systematically examined a group of patients with prolonged sinoatrial conduction time. In addition, short-term follow-up observations of symptomatic sinus node disease were made.

Patients and Methods

Definitions

The following electrocardiographic definitions were utilized: Sinus bradycardia was defined as a mean sinus rate of less than 60 beats/min. Persistent resting sinus bradycardia described patients in whom all resting electrocardiograms revealed sinus rates less than 60 beats/min. Sinoatrial block was defined by the presence of dropped sinus P waves with a resulting asystolic pause which was a multiple of sinus cycle length. Asystolic periods greater than one and a half sinus cycle length but less than two sinus cycle lengths were also noted and were classified as sinus pauses. Atrial arrhythmias (paroxysmal atrial tachycardia and atrial fibrillation) and conduction defects were diagnosed using standard electrocardiographic criteria.

Patient Selection

Between January 1974 and January 1976, 470 patients underwent diagnostic electrophysiological studies in our laboratories, utilizing atrial pacing and atrial extrastimulus testing. Indications for study included atrioventricular (A-V) and intraventricular conduction defects, atrial or ventricular tachyarrhythmia, and suspected sinus node disease. The latter category accounted for 52 of the 470 patients studied.

Of the 470 patients, 24 (5%) had a prolonged (greater than 152 msec) calculated sinoatrial conduction time or a Zone I response (23 and one patient, respectively). These 24 included 15 with suspected sinus node disease (29% of the 52 patients with suspected sinus node disease) and nine without previously suspected sinus node disease (2% of 418 patients without suspected sinus node disease).

Clinical Methods

Initial evaluation of patients included history, physical examination, chest X-ray and serial electrocardiograms. Informed written consent was obtained prior to electrophysiological study. Electrophysiological studies were performed utilizing previously described catheter techniques.

Cardiac drugs were withheld for 48-72 hours before study. Measurements of A-H and H-V intervals were made at paper speeds of 200 mm/sec and reflect the mean of ten consecutive sinus beats. Refractory periods of the atrium and A-V node were measured as previously described by Denes and co-workers. The validity of these measurements has been supported by recent studies reported by Reddy et al.

Sinus node function was evaluated with both rapid atrial pacing and extrastimulus technique. Sinus node recovery times were measured by noting the asystolic pause after sudden cessation of atrial pacing. In each patient, three
recovery times were measured at a paced rate of 130 beats/min and averaged (normal 1680 msec or less). Sinus node responses to atrial extrastimuli (A) were categorized by noting the time of occurrence of the first spontaneous sinus beat (A) following A. Non-reset due to sinus interference was defined when A occurred at an A-A interval of more than spontaneous sinus cycle (A-A1 and A-A2 (coupling interval and return cycle interval) equaled twice A-A1. Sinus reset was defined when A-A was equal to or more than A-A and A-A was less than twice A-A1. Sinus interpolation was defined when A-A was less than A-A1 and A-A was either equal to A-A1 (complete interpolation) or more than A-A1 (incomplete interpolation). Sinus echoes were defined when A-A interval was less than A-A1, and P wave morphology of A1 approximated that of the normal P wave with a high to low sequence of atrial activation.

Each patient was categorized as to whether or not he had the above zones. In each patient, a zone, if present, was defined by its longest and shortest A-A coupling interval and in terms of its absolute duration. When considering all patients with a given zone, the zone was described in terms of its mean longest and shortest A-A coupling intervals as well as its mean absolute duration.

Sinoatrial conduction time was calculated as described by Strauss and associates. For each patient, this was obtained by measuring the difference between A-A and A-A1 interval during the zone of reset and dividing by two. A mean sinoatrial conduction time was calculated for each patient using all reset responses 92 ± 60 msec; normal mean ± 2 SD. To be included in this study of patients with prolonged sinoatrial conduction time, reset responses had to meet the following criteria: 1) Reset responses had to decrease linearly with decrease in coupling interval, parallel to line of reset (fig. 1). 2) There had to be at least ten measurable reset responses in the zone of reset (fig. 1). 3) The P wave morphology of the reset return beat (A) and its high-to-low activation sequence had to resemble that of sinus P wave (fig. 2 C, D). 4) Significant atrial ectopic activity and sinus arrhythmia (greater than 5% variation in spontaneous sinus cycle length) had to be absent (fig. 2, A-D). Three patients with apparently prolonged calculated sinoatrial conduction time failed to meet criteria 2 and/or 4 and were excluded from the present report.

**Patient Follow-up**

This study of patients with prolonged sinoatrial conduction time was conducted concomitantly with a large prospective study of patients with chronic intraventricular conduction defects. Follow-up consisted of periodic visits of patients at one to three monthly intervals to a conduction clinic. Each clinic visit included history, physical examination and electrocardiogram. Development of major symptoms (dizziness, syncope, or congestive heart failure) warranted rehospitalization of patients and in-patient electrocardiographic monitoring. Portable 24-hour outpatient tape-recorded electrocardiographic monitoring (Holter Avionics) was also utilized in 13 patients for detection of transient bradyarrhythmias. All 13 patients had dizziness or syncope. Documented symptomatic bradyarrhythmia was treated with permanent pacemaker implantation.

**Results**

**Clinical Data (table 1)**

There were 18 males and six females with ages ranging from 29 to 85 years (65 ± 2.6 years). Cardiovascular diagnoses in these patients included arteriosclerotic heart

![Figure 1](link)
disease in ten patients (42%), hypertensive cardiovascular disease in five (21%), valvular heart disease in three (12%) and no diagnosable organic heart disease in six (25%).

Symptoms noted prior to study included syncope in three, dizziness in four, exertional angina in three, dyspnea on exertion in four, and palpitation in three patients. Syncope was related to sinus bradycardia in two and to ventricular tachycardia in one. Dizziness was due to atrial tachycardia in two and did not correlate with arrhythmia in two. Eleven of 24 patients (46%) were in New York Heart Association Functional class I, ten (42%) were in functional class II, and three (12%) were in class III.

**Sinus and Atrial Dysrythmia (table 2)**

Nineteen of the 24 (79%) patients had significant sinus or atrial dysrythmia detected either on resting electrocardiograms or 24 hour portable monitoring. Observed sinus rates ranged from 33 to 120 beats/min, with a mean minimum rate of 54 ± 3.0 and a mean maximum rate of 74 ± 3.9 beats/min. Ten (42%) patients (1, 3, 5, 6, 7, 10, 11, 13, 14, and 22) had persistent resting sinus bradycardia. Intermittent sinoatrial block was detected in seven (29%) patients (3, 4, 7, 12, 17, 19, and 21). Three patients (12%) had intermittent sinus pauses (6, 15, and 23). Intermittent episodes of nonsustained paroxysmal supraventricular tachycardia with sinus bradycardia were present in three (12%) patients (1, 3, and 11). In one of these patients (3) chronic atrial fibrillation developed after one month of study. Intermittent sustained paroxysmal atrial tachycardia with varying A-V block, unrelated to digitalis, was documented in one patient (24). Six patients (25%) had multiple (>5/min) premature atrial contractions on resting electrocardiograms (1, 5, 7, 8, 17, and 23).

In summary, of the 19 patients with significant sinus or

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**Table 1. Clinical Features and Sinoatrial Electrophysiological Data**

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*Cause of syncope was ventricular tachycardia.

**Figure 2. Demonstration of sinus interference and reset responses in patient 22. In each panel, simultaneous electrocardiographic leads I, II, III, V₁, high right atrial electrogram (HRA) and His bundle electrogram (HBE) are shown. Paper speed is 100 mm/sec and timelines are at one second intervals in this illustration. The extrastimulus is labeled with an arrow and the His bundle potential as H. Sinus cycle length (A₁-A₁), coupling interval (A₁-A₂), return cycle (A₂-A₁), and sinoatrial conduction time (SACT) are listed. Panel A) Sinus rhythm at a cycle length of 1170 msec. At a coupling interval of 1100 msec, A₁ occurs at an A₁-A₂ interval of 1260 msec, and A₁-A₂ is approximately equal to 2 × A₁-A₁, defining non-reset of the sinus node. Panel B) Decrease in A₁-A₂ interval to 830 msec results in lengthening of the A₁-A₂ interval to 1550 msec. The A₁-A₂ interval remains constant (2 × A₁-A₁). This defines the lower limit of zone of interference. Panel C) At A₁-A₁ of 820 msec, A₁ occurs at an A₁-A₂ interval of 2370, which is less than twice A₁-A₁ (1200 msec), and A₁-A₂ is 1550 msec is more than A₁-A₁. The sinus node is reset by A₂ defining the outer limit of the zone of reset. The sinoatrial conduction time (half the difference between A₁-A₁ and A₁-A₂) is 175 msec. Panel D) Further shortening of A₁-A₁ interval to 390 msec does not change A₁-A₂ interval suggesting unaltered sinus automaticity. The A₁-A₂ interval of 1940 msec is less than twice A₁-A₁. The sinus node is reset by A₂ defining the lower limit of zone of reset. The sinoatrial conduction time is 185 msec. The zone of non-reset in this patient ranged from 1190 to 830 msec and zone of reset was between 820 and 390 msec.
atrial dysrhythmia, 15 had sinus bradyarrhythmias alone (sinus bradycardia and/or sinoatrial block), three had both sinus bradyarrhythmia and atrial tachycardia and one had isolated atrial tachycardia.

For comparison, the frequency of atrial dysrhythmias was investigated in 37 patients with suspected sinus node disease (sinus bradycardia, S-A block and/or sinus arrest) and normal calculated sinoatrial conduction time. Ten of these (27%) had significant atrial arrhythmias: five with paroxysmal atrial flutter or fibrillation and five with atrial tachycardia. In addition, three (8%) other patients had multiple premature atrial contractions on resting electrocardiograms.

Other Electrocardiographic Observations

P-R intervals ranged from 0.13 to 0.34 sec (mean, 0.21 ± 0.01) and were prolonged (greater than 0.20 sec) in 12 of 24 (50%) patients. Intraventricular conduction defects were present in ten patients (42%); three with left anterior hemiblock, four with right bundle branch block and left anterior hemiblock and three with left bundle branch block. Multiple premature ventricular contractions were present in four patients and two of these had intermittent ventricular tachycardia.

Sinoatrial Function (table 1)

By definition (study design), calculated sinoatrial conduction times were prolonged in the 23 patients with a zone of reset and ranged from 155 to 220 msec with a mean ± SEM of 180 ± 4.4 msec. In an additional patient, only Zone 1 responses were present throughout the scanned sinus cycle suggesting prolonged sinoatrial conduction time.1 The zones of sinus responses to atrial extrastimuli were as follows: A zone of non-reset due to interference (Zone I) was defined in all patients during late atrial diastole (figs. 1 and 2). The mean zone of interference was between 975 (outer limit) and 574 msec (inner limit) with an absolute duration of 401 msec at a mean sinus cycle length of 975 msec (figs. 1, 2 A and B). This zone accounted for the last 41% of the total sinus cycle length.

Sinus reset was defined in all patients except the one with a Zone I response (in whom the zone of interference accounted for 100% of scanned sinus cycle length). The mean zone of reset in the 23 patients was between 570 (outer limit) and 352 msec (inner limit), with an absolute duration of 218 msec at a mean sinus cycle length of 962 msec, accounting for middle 22% of the sinus cycle length (figs. 1, 2 C and D). A zone of interpolation was defined in two patients and ranged from 315 (outer limit) and 285 msec (inner limit) with an absolute duration of 30 msec at a mean sinus cycle length of 885 msec and accounted for 3% of scanned cycle length. None of the patients had sinus echo responses during atrial extrastimuli testing.

Sinus node recovery times were measured in all patients. These ranged from 820 to 2740 msec with a mean of 1421 ± 98 msec and were prolonged (>1680 msec) in five (20%). Atrial effective and functional refractory periods were measured during sinus rhythm. Atrial effective refractory period ranged from 190 to 530 msec with a mean of 298 ± 15.7 msec and was prolonged (>350 msec) in four (16%). The atrial functional refractory period ranged from 280 to 550 msec, with a mean of 352 ± 15.4 msec, and was prolonged (>375 msec) in eight (33%).

In summary, in addition to prolonged sinoatrial conduction time, 11 (46%) patients had electrophysiological evidence of sinus node or atrial dysfunction. Three patients had evidence of sinus node dysfunction alone, six had evidence of isolated atrial dysfunction, and two had both sinus and atrial dysfunction.

A-V Nodal and Intraventricular Conduction

A-H interval ranged from 54 to 225 msec, with a mean of 118 ± 9.7 msec, and was prolonged (>130 msec) in six (25%).1 A-V nodal effective refractory period could be measured in eight patients and ranged from 270 to 510 msec, with a mean of 419 ± 10.5 msec and was prolonged (>400 msec) in four (16%). Atrioventricular nodal functional refractory period, measured in all patients, ranged from 370 to 580 msec with a mean of 492 ± 11 msec and was prolonged (>500 msec) in 12 (50%). H-V interval (HisPurkinje conduction) ranged from 31 to 75 msec with a mean of 47 ± 2.6 msec and was prolonged in (>55 msec) in five patients (21%).9

In summary, 12 patients (50%) had electrophysiological evidence of A-V nodal dysfunction and/or abnormalities of intraventricular conduction.

Follow-up Data (table 3)

Follow-up periods ranged from 28 to 668 days with a mean of 427 ± 39 days. Permanent pacemakers were implanted in four patients who developed symptomatic bradyarrhythmia. Patient 1 was an 85-year-old female with arteriosclerotic heart diseasse, congestive heart failure, and sinus brady-
cardia admitted with a history of syncope. Electrophysiological studies revealed a calculated sinoatrial conduction time of 160 msec and prolonged sinus node recovery time. Twenty-four hour portable electrocardiographic monitoring revealed sinus rate between 40-55 beats/min while awake and 33-50 beats/min during sleep. In addition, intermittent episodes of paroxysmal atrial tachycardia with sinus bradycardia were noted. The patient was treated with a permanent pacemaker implantation on the tenth day for recurrent syncope.

Patient 3 was an 80-year-old male with arteriosclerotic heart disease, sinus bradycardia, and sinoatrial block who was admitted for congestive heart failure. Initial electrophysiological studies revealed a calculated sinoatrial conduction time of 220 msec and normal sinus recovery time. Portable electrocardiographic monitoring revealed intermittent paroxysmal atrial tachycardia. Digitalis therapy for paroxysmal atrial tachycardia and heart failure was accompanied by development of persistent atrial fibrillation with slow ventricular response. A permanent pacemaker was inserted on the 36th day of study. The patient subsequently died of heart failure on 137th day of follow-up.

Patient 4 was a 59-year-old male admitted with recurrent syncope and a normal physical examination and resting electrocardiogram. Electrophysiological studies revealed a calculated sinoatrial conduction time of 155 msec and normal sinus recovery time. A permanent pacemaker was implanted on the 14th day for recurrent syncope and documentation of frequent episodes of sinoatrial block.

Patient 11 was a 62-year-old female with arteriosclerotic heart disease, sinus bradycardia, and congestive heart failure. Electrophysiological studies revealed calculated sinoatrial conduction time of 205 msec and normal sinus recovery time. Portable electrocardiographic monitoring revealed persistent sinus bradycardia with intermittent paroxysmal supraventricular tachycardia. Patient was treated with permanent pacemaker implantation on 25th day for congestive failure and bradycardia.

Three of the patients (12%) died during follow-up (pts 3, 16, and 23) on days 137, 28, and 350, respectively. The cause of death was congestive heart failure in two and a cerebrovascular accident in the other. None died suddenly. Postmortem examinations were not obtained.

In summary, permanent pacemakers were needed in only four of 24 patients with prolonged calculated sinoatrial conduction time, despite the frequent presence of electrocardiographic abnormalities of sinus node function. The short term mortality of 12% seemed appropriate, taking into account ages and presence of organic heart disease in this group. The mortality did not appear to be directly related to sinus node dysfunction.

### Discussion

Sinus node function can be evaluated only by indirect means. In 1973, Strauss and co-workers, described a method of indirectly calculating sinoatrial conduction time in man utilizing atrial extrastimulus technique. They calculated sinoatrial conduction with impulses resetting the sinus node. The degree to which the return cycles exceeded the spontaneous sinus cycle reflected the conduction time of the atrial impulses into and then out of the sinus node. Sinoatrial conduction time was calculated to be half the difference between return and spontaneous sinus cycles.

Calculated sinoatrial conduction time in their four patients with sinus node disease ranged from 68 to 156 msec.

Recently, several workers have reported ranges of normal values for calculated sinoatrial conduction time in patients without apparent sinus node dysfunction. Calculated sinoatrial conduction times ranged from 40-70 msec, with a mean ± 2 SD of 56 ± 22 msec, in five patients reported by Steinbeck et al., from 39.5 to 97.5 msec, with a mean ± 2 SD of 70 ± 30 msec, in 18 patients reported by Masini et al; from 28.5 to 115.5 msec, with a mean ± 2 SD of 84.5 ± 26 msec, in 20 patients reported by Engel and co-workers, and from 40 to 153 msec, with a mean ± 2 SD of 92 ± 60 msec in 36 patients reported from our laboratory. In the present report, we have used our own upper limit of normal (152 msec). We recognize that is somewhat longer than other laboratories. Because of this, some workers might consider the patients in the present series as having moderate or marked sinoatrial conduction time prolongation.

The indirect calculation of sinoatrial conduction time in man necessitates several assumptions: 1) there is no change in sinus automaticity or shift of sinus pacemaker with atrial extrastimuli; 2) all extrastimuli during the zone of reset are able to penetrate the sinus node and reset it; and 3) the antegrade and retrograde conduction times through the perinodal fibers are of equal magnitude.

In a recent experimental study, Miller and Strauss reported a poor correlation between direct and indirect measurements of sinoatrial conduction time in rabbit hearts. Shortening of sinus node action potentials by extrastimuli elicited in late atrial diastole (which were unable to capture and reset the sinus node) resulted in return cycles (A2-A3) which were less than compensatory (pseudo-reset). This caused underestimation of true sinoatrial conduction time.
when calculated indirectly. Bonke and associates demonstrated in the isolated right atrium of the rabbit that premature atrial extrasystoles elicited early in the atrial cycle caused pacemaker shifts within the sinus node. The curves depicting the atrial return cycle response to extrastimuli in man differ significantly from those obtained by Miller and Strauss in their animal studies. Some of the characteristics of human return cycle responses include: \(^1\) 1) A clearcut transition from non-reset to reset responses with extrastimuli, as opposed to the long transition periods in animals (fig. 1); 2) Constant A\(_2\)-A\(_3\) intervals despite progressive shortening of the A\(_1\)-A\(_3\) interval during the zone of reset in contrast to the variable patterns of return cycles in experimental animals (fig. 1); and 3) The occurrence of A\(_1\)-A\(_3\) responses that parallel the line of reset (line of zero sinoatrial conduction time) (fig. 1), suggesting unaltered sinus automaticity in man as opposed to nonuniformity of these responses in animal experiments. Moreover, in man the P wave morphology of return beat (A\(_3\)) during reset approximates that of the normal sinus beat, suggesting absence of major pacemaker shifts in the sinus node (fig. 2 C, D).

It has recently been reported that sinoatrial conduction times in patients with sinus node dysfunction are significantly longer than in those with apparently normal sinus node function. Steinbeck et al. reported that sinoatrial conduction time ranged from 105 to 150 msec, with a mean of 133 msec, in three patients with sinus node dysfunction. Masini et al. reported seven patients with sinus node dysfunction in whom sinoatrial conduction times ranged from 75.5 to 148.5 msec with a mean of 126 msec. Similar results were reported by Engel and co-workers. In the present report, 29% of patients with suspected sinus node disease had prolonged sinoatrial conduction time, as opposed to 2% of patients without suspected sinus node disease.

In our series, prolonged sinoatrial conduction time was associated with a high incidence of electrocardiographic abnormalities of sinus node function. Sinus bradycardia and/or sinoatrial block was a frequent finding (19 of 24 patients). Only 13 of 24 patients underwent 24 hours tape recorded electrocardiographic monitoring for detection of bradyarrhythmia. Application of this technique to more of our patients might have uncovered additional electrocardiographic sinus node abnormalities. Atrial tachyarrhythmia was also common, although not significantly more frequent than in patients with sinus node dysfunction without prolonged sinoatrial conduction time.

We noted a poor correlation between prolonged sinoatrial conduction time and sinus node recovery time. Sinus node recovery times were prolonged in only five of the 24 patients with prolonged sinoatrial conduction times. It should be emphasized that sinus node recovery time and calculated sinoatrial conduction time identify two different aspects of sinus node function. The recovery time is a measure of automaticity while sinoatrial conduction time is a measure of perinodal conduction. Among other factors, one of the determinants of sinus recovery time is the ability of driven atrial impulses to penetrate the sinus node. Increased refractoriness and/or depressed perinodal conduction might allow fewer driven atrial impulses to penetrate the sinus node (sinus entrance block) resulting in pseudo-shortening of recovery time.

There was a relatively high incidence of prolonged atrial refractory periods in our patients with a prolonged sinoatrial conduction time, suggesting an association of atrial, perinodal, and sinus nodal disease. Since the sinus node and perinodal tissues are right atrial structures, it was not surprising to find frequent atrial dysfunction in this group. The presence of atrial disease could relate to the development of atrial dysrhythmias (paroxysmal atrial tachycardia and atrial fibrillation) in some of our patients. We would postulate that many of our patients had atrial degenerative disease with secondary involvement of the sinus node.

There was frequent evidence of both electrocardiographic and electrophysiologic abnormalities of conduction through the A-V node and intraventricular conduction system in patients of the present series. These observations are consistent with those of previous workers, who reported a high incidence of A-V conduction defects in patients with sinus node disease. The clinical course of our patients with prolonged sinoatrial conduction time was relatively benign. Sinocept occurred in only three of our 24 patients. The incidence of congestive heart failure was also low and more than 80% of patients were in cardiac functional class I or II. Permanent pacemakers were required in only four patients for symptomatic bradyarrhythmia, despite a high incidence of electrocardiographic evidence of sinus node dysfunction. The decision for permanent pacemaker therapy was a clinical decision, based upon documentation of symptomatic bradyarrhythmias.

Our short-term results suggest that prolonged sinoatrial conduction time with or without electrocardiographic evidence of sinoatrial dysfunction, in the absence of symptoms, is not an indication for prophylactic pacemaker insertion. Additional prospective studies utilizing measurement of sinoatrial conduction time, as well as other clinical and electrophysiologic parameters of sinus node function, will be necessary before it is known whether a group of patients in need of prophylactic pacing can be detected. It is our opinion at present that the decision for pacing patients with suspected sinus node dysfunction should be made on the basis of clinical symptoms.

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Effects of Procainamide and Quinidine Sulfate in the Wolff-Parkinson-White Syndrome

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SUMMARY Thirty-three patients with Wolff-Parkinson-White syndrome were studied electrophysiologically before and after administration of intravenous procainamide and oral quinidine sulfate. Procainamide prolonged the shortest R-R (SRR) interval between two consecutive pre-excited beats during atrial fibrillation 20-70 msec in 15 of 21 patients with no change observed in 6 of 21 patients. Quinidine sulfate prolonged the SRR 20-170 msec in all 16.

In 14 of 18 patients where procainamide and quinidine were comparable, quinidine prolonged the SRR 30–100 msec more than procainamide.

RECENT DEVELOPMENTS in methods of preoperative and operative technique have established the potential role of surgical intervention for treatment of selected cases of the pre-excitation syndrome refractory to medical management. The selection of optimal medical therapy has also been given a more rational basis by combining electrophysiologic studies with pharmacologic trials. The antegrade and retrograde effective refractory periods of normal and accessory pathways may have prognostic importance when used to assess the potential value of various pharmacologic agents. However, these measurements cannot always be made because of atrial and ventricular muscle refractoriness. Additional information is provided by elective induction of atrial fibrillation before and after drug administration. Several authors have described an approximately linear relationship between the antegrade effective refractory period (AERP) of the accessory pathway (AP) and the maximal ventricular response during atrial fibrillation. The purpose of this study was to evaluate the usefulness of both refractory period determination and elective induction of atrial fibrillation in assessing the effect of antiarrhythmic therapy in patients with pre-excitation.

Materials and Methods
Thirty-three patients (25 male, 8 female) with Wolff-Parkinson-White syndrome were selected for drug study because the short ERP of their accessory pathway (AP) per-
Clinical significance of prolonged sinoatrial conduction time.
R C Dhingra, F Amat-y-Leon, C Wyndham, P C Deedwania, D Wu, P Denes and K M Rosen

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