Sinus node recovery time in the elderly

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Measurement of the sinus node recovery time has been proposed as a diagnostic tool for recognition of the sick sinus syndrome. The latter is most frequently encountered in elderly patients with hypertension, coronary heart disease, and atherosclerosis. In order to provide normal values for the sinus node recovery time in this particular population group, atrial pacing studies were carried out in 30 subjects over 50 years of age, all with peripheral vascular disease and some with angina pectoris (10), residua of infarction (6), or hypertension (7).

On stimulation, 7 patients maintained a 1:1 atrioventricular conduction up to the rate of 180/min. Second degree atrioventricular block developed in all other cases. On six occasions, Wenckebach’s periods appeared at the relatively slow pacing rate of 120/min.

The maximum postoverdrive pause ranged from 680 to 1600 ms with an average of 1100 ms ± 190 (1σ). For each pacing speed, a correlation was found between the duration of the pause and the control intrinsic cardiac rate, longer pauses being associated with longer resting PP intervals. Beyond 120/min, the duration of the pause was seen to shorten progressively as the driving rate was increased. Finally, the behaviour of the sinus node pacemaker following interruption of pacing showed individual variations. After pacing at relatively slow rates, a prompt return to near control values was consistently observed, whereas, after fast rates of driving, a phase of secondary depression developed in about one-half of the studied cases.

In recent years, the syndrome of sinus node dysfunction and sinoatrial disorders has elicited great interest (Short, 1954; Birchfield, Meneeé, and Bryant, 1957; Ferrer, 1968; Bouvain, Slama, and Temkine, 1967; Adelman and Wigle, 1969; Slama et al., 1969; Tabatznik et al., 1969; Easley and Goldstein, 1971; Rasmussen, 1971; Rubenstein et al., 1972; Wan, Lee, and Toh, 1972; Kulbertus, de Leval-Rutten, and Demoulin, 1973; Kaplan et al., 1973; Conde et al., 1973; Chokski et al., 1973). The growing recognition of the prevalence of the sick sinus syndrome, especially in elderly patients, has prompted research for practical clinical tests permitting evaluation of the sinus node function.

At present, no means are available to record selectively the sinus node potentials in man. Therefore, one has to resort to indirect methods, and it seems generally agreed that the phenomenon of postpacing depression of cardiac pacemakers may be used for this purpose (Mandel et al., 1971, 1972; Narula, Samet, and Javier, 1972). The sinus node recovery time which follows short periods of overdrive atrial pacing has been measured in normal individuals and in patients with the sick sinus syndrome; it was shown to be abnormally prolonged in some of the latter (Mandel et al., 1971, 1972; Narula et al., 1972; Kulbertus et al., 1973).

There still persists, however, some uncertainty regarding the normal limits of the sinus node recovery time. The values presently considered as normal have actually been obtained from a rather heterogeneous material with respect to age and pathology. These normal reference values need to be verified in the population group where the sick sinus syndrome is most frequently encountered, i.e. in elderly subjects, more particularly those suffering from atherosclerosis, hypertension, and coronary vascular disease.

The main purpose of this paper is to provide normal data for this particular group of patients. At the same time, some features of postoverdrive sinus node depression will be discussed in their physiological context.

Subjects and methods
The data have been gathered during a study of the validity of atrial pacing techniques for the diagnosis of latent coronary insufficiency in patients with peripheral
vascular disease. The group comprised 30 patients, 25 male and 5 female, ranging in age from 50 to 72, with an average of 62. All had evidence of peripheral vascular obstructions. Ten suffered from coronary heart disease and 7 were hypertensive. The control electrocardiogram was normal in 11 cases. It showed left ventricular hypertrophy in 3, intraventricular conduction disturbances in 6, residua of myocardial infarction in 6, and isolated changes of the repolarization phase in 4. The intraventricular conduction defects comprised 4 instances of left anterior hemiblock, one of complete left bundle-branch block, and one of left axis deviation with right bundle-branch block. The clinical history was, in all cases, devoid of any symptom that might have suggested a brady-tachycardias syndrome or transient atrioventricular conduction disturbances. The PR interval was normal in each case and no sustained bradycardia was noted. All cardiovascular drugs were stopped one week before the study.

The procedure was explained to the patients and informed consent was obtained in each case. The subjects were all studied in the postabsorptive state and received no premedication. A bipolar pacing catheter was passed transcutaneously in the right femoral vein and positioned along the high lateral wall of the right atrium. Pacing at various rates was performed with a battery-powered R wave coupled pulse generator (Medtronic Model 5837). In order to avoid resetting of the sinoatrial pacemaker discharge or development of transient atrial flutter or fibrillation (Lange, 1965), pacing was started at a frequency exceeding the resting intrinsic cardiac cycle of 20 beats/min. The patient was paced at increasing atrial rates. The rates used were generally 100, 120, 130, 140, 150, 160, and frequently 180/min. The periods of pacing lasted 2 minutes and were separated by intervals of 3 minutes. The scalar X, Y, and Z leads were recorded using the McFee-Parungao system and a Hewlett-Packard 1520 A vectorcardiograph. The signals were recorded on a magnetic tape (Hewlett-Packard instrumentation Recorder 3960 A) and later reproduced on a 3-channel electrocardiograph apparatus (Hewlett-Packard Model 1513 A).

The postoverdrive sinus pause was measured from the last spike of stimulation to the beginning of the first P wave seen after turning off the pacemaker (Fig. 1). Whenever the asystolic pause ended with a junctional or ventricular escape beat, its value was discarded for the calculation of the mean sinus node recovery time. Finally, the PP intervals were measured during the first few beats after the end of pacing.

**Results**

Before describing the influence of pacing on the sinus node function, it is noteworthy that only 7 out of about 200 postoverdrive pauses ended with a junctional or ventricular escape beat. Arrhythmias which were not present in the control tracing sometimes occurred during the postoverdrive period. Atrial pacing was thus followed twice by a transient episode of paroxysmal atrial tachycardia and, once, by a bout of atrial chaotic rhythm. Ventricular ectopic contractions were observed in 8 patients and atrial premature beats in 10 others, one of whom showed a period of atrial bigeminal rhythm.

The maximum postoverdrive pause ranged from 680 to 1600 msec, with an average of 1100 ms ± 190 (10). These results can also be expressed in terms of corrected sinus node recovery time, i.e. the recovery interval in excess of the control sinus cycle (Narula et al., 1972). This parameter ranged from 110 to 680 ms with an average of 295 ms ± 119 (10).

For each paced rate, a significant linear correlation was found between the duration of the pause and the control PP interval measured from the last 10 beats before pacing, longer pauses being associated with slower control rates. This is illustrated by the set of diagrams showed in Fig. 2. A similar linear correlation was disclosed between the maximum pause measured in each patient and the control PP interval (P < 0.001).

The response to various pacing rates is studied in Fig. 3. Statistically, no significant difference could be disclosed between the duration of the pause measured after pacing at 100 and 120/min (Student's t test for paired observations). On the other hand, a progressive linear decrease of the pause duration was noted as the atrial pacing rate was increased from 120 to 160/min.

The behaviour of the first 20 PP intervals following cessation of pacing was also studied, after various rates of stimulation, in 16 instances. Some individual variations were observed. In 7 patients, interruption of pacing was followed, whatever the rate of overdrive, by a prompt return to near control values. In the 9 others, the response

**FIG. 1** Example of the sinus pause observed after a 2-minute overdriving of the atrium at 150/min. The sinus node recovery time is measured from the last spike of stimulation to the first P wave following interruption of pacing.
FIG. 2  Relation between the resting PP interval and the duration of the pause observed after pacing at 100/min, 120/min, 140/min, and 160/min. The equations of the regression lines are as follows:

- at 100/min: $y = 236 + 0.929x; r = 0.62; P < 0.01$
- at 120/min: $y = 156 + 1.089x; r = 0.70; P < 0.001$
- at 140/min: $y = 123 + 1.080x; r = 0.55; P < 0.01$
- at 160/min: $y = 211 + 0.844x; r = 0.575; P < 0.01$

FIG. 3  Relation between pause duration and rate of pacing. The data presented in this figure are limited to 21 patients whose sinus node recovery time could be accurately measured at each pacing rate. The other patients were excluded because of the occurrence of escape beats or rhythm disturbances in some of the postoverdrive periods.

The equation of the regression line is:

$$y = 1.329 - 2.73x; r = -0.23; P < 0.02.$$
was different after slow and fast rates of stimulation: a progressive return to resting values was noted after pacing at relatively slow rates (100 or 120/min); on the other hand, after pacing at higher rates (above 150/min) the very first PP intervals after the pause were generally shorter than during the control period; a stepwise lengthening of the cycle length then followed, leading in 7 instances to a phase of secondary depression lasting for about 10 beats (Fig. 4).

The response of the atrioventricular junction was finally studied. It was confirmed that the interval between the stimulation spike and the beginning of the following ventricular complex progressively lengthened when the pacing rate was increased (Damato et al., 1969). Seven patients maintained a 1:1 atrioventricular conduction up to the rate of 180/minute. Twenty-two others developed a second degree atrioventricular block with Wenckebach's periodicity. The pacing rates at which the 1:1 atrioventricular conduction was lost were distributed as follows: 120/min (6 cases); 130/min (1 case); 140/min (5 cases); 150/min (5 cases); 160/min (1 case); and 180/min (4 cases). In the last patient a type II second degree atrioventricular block appeared when the atrial stimulation rate reached 160/min.

**Discussion**

These results will be discussed from both the electrophysiological and clinical points of view.

**a) Electrophysiological aspects**

The phenomenon of overdrive suppression has been demonstrated by in vitro and in vivo experiments in animals (Gaskell, 1882; Amory and West, 1962; Lange, 1965; Lu, Lange, and Brooks, 1965; Brooks and Lu, 1972); it has also been well documented in man (Mandel et al., 1971, 1972; Narula et al., 1972). The precise mechanisms underlying the suppression of spontaneous impulse formation immediately upon cessation of a superimposed drive are still debated. Pharmacological observations demonstrating that suppression is enhanced by neostigmine and reduced by atropine or hemicholinium suggest that artificial driving may result in a local release of acetylcholine (Amory and West, 1962; Vincenzi and West, 1963; Lange, 1965). As atropine does not suppress completely the postoverdrive depression, however, it seems logical to assume that another mechanism is involved in the after-effect of overdrive (Lange, 1965). The hypotheses of changes in potassium flux (Scher et al., 1959; Lu et al., 1965) or of activation of an electrogenic sodium pump (Vassalle, 1970) have been considered. The data obtained in the present study cannot contribute to the understanding of this phenomenon.

Our findings demonstrate that, for the various pacing rates, there is a linear correlation between the intrinsic cardiac rate in sinus rhythm and the duration of the postoverdrive pause. Mandel et al. (1971) also observed a linear relation between the control sinus rate and the maximum pause in their patients.

**FIG. 4 Study of the subsequent PP intervals following interruption of pacing.**

C.R. = control rate; St: stimulation.

Three examples are shown. In A, a prompt return to control values is seen after cessation of pacing. In B and C, the same occurs after slow rates of pacing. However, when the rate of overdrive is 160/min, the first PP interval after the pause is shorter than the control value. This is followed, in B, by a progressive return to the resting rate and, in C, by a phase of secondary depression.
The experimental findings in animals generally indicate that, within limits, the initial suppression increases with the rate of overdrive (Lu et al., 1965; Brooks and Lu, 1972). Previous observations in man also showed a stepwise lengthening of the pause as the pacing rate was increased (Mandel et al., 1971). There was, however, in Mandel’s series (1971) a sharp shortening of the pause when the rate of overdrive was set at 150 beats/min. As regards this phenomenon, Lu et al. (1965) stated that ‘whatever the depressing influence may be, there is an optimum rate of drive for its accumulation’. Our results, obtained in elderly subjects, indicate that in this age group, the optimum rate is low, since, beyond 120/min, the sinus node recovery time progressively and steadily decreases as the rate of atrial stimulation is increased.

The factors that determine the pause duration remain conjectural. It is clearly demonstrated that besides acetylcholine, catecholamines are liberated during cardiac drive by artificial pacemakers (Amory and West, 1962; Vincenzi and West, 1963; Lange, 1965; Brooks and Lu, 1972). The overall response to atrial overdrive is, therefore, determined by the release of two mediators with opposite influence. Their relative importance might change with the rate of atrial pacing. Haemodynamic alterations, possibly resulting in a reflex discharge, may also be induced by pacing (Schwartz, Zimmermann and Thormann, 1974); their influence on sinoatrial node suppression must be considered though this has been thought to be minimal (Mandel et al., 1971). Finally, it is known that nodal cells fail to follow an increasing rate much earlier than the adjacent atrial cells (Brooks and Lu, 1972). Furthermore, studies of conduction within the node reveal the possibility of block or decreasing propagation of impulse in this tissue (Lu et al., 1965). According to Strauss et al. (1973), a second degree sinoatrial entrance block may occur at fast rates of pacing and lead to a paradoxical effect: faster atrial stimulation rates causing slower discharge of the node, and, consequently, decreasing sinus depression. It thus appears that several factors are involved in the determination of the postpacing pause duration. All these factors may also influence the behaviour of the sinus node pacemaker during the short period that follows interruption of overdrive. In this respect, let us just mention that the phenomenon of secondary depression which was seen in some of our patients after fast rates of pacing was also observed, though less frequently, by Mandel et al. (1971). This feature deserves further attention.

A final comment should be made about the escape phenomenon. While cessation of the activity of a natural dominant pacemaker normally allows subsidiary pacemakers to emerge, the postoverdrive pause rarely ends with an escape beat. This particular behaviour is probably because, besides having the faster intrinsic rate, the sinoatrial node is also the pacemaker which is the least depressed by overdrive and which recovers the most promptly after interruption of pacing (Lange, 1965; Lu et al., 1965; Brooks and Lu, 1972).

b) Clinical implications

The phenomenon of postpacing depression of cardiac pacemakers has been used to assess the sinus node function and thus to provide a diagnostic tool for recognition of the sick sinus syndrome (Mandel et al., 1971, 1972; Narula et al., 1972). The sinus node recovery time has been determined by different investigators (<1400 ms, Rosen et al., 1971; 1040 ms ± 56, Mandel et al., 1971; 1073 ms ± 67, Mandel et al., 1972; 958 ms ± 149, Engel and Schaal, 1973; corrected sinus recovery time: <525 ms, Narula et al., 1972). The purpose of the present study was to find out whether these previously reported values could be reliably applied in the group of elderly patients with atherosclerosis, coronary insufficiency, or hypertension among whom most of the cases of sinoatrial disease are recruited. In fact, the average values that we have measured are very close to those obtained by others. As a rule of thumb, it may be stated that whenever the sinus node recovery time is longer than 1500 ms, the sinus node function is likely to be abnormal. The overdrive suppression of sinus node function observed in patients with the sick sinus syndrome may far exceed this mean average maximal value (Mandel et al., 1971, 1972; Narula et al., 1972; Rosen et al., 1971; Engel and Schaal, 1973; Kulbertus et al., 1973). However, this is not a uniform finding. Subjects with well-documented sinoatrial disease may display rather short pauses after atrial pacing. It is possible that in some of these cases, sinoatrial node entrance block prevents the sinus pacemaker from being discharged during atrial stimulation. In fact, if the measurements are repeated after injection of atropine, the entrance block may disappear and the phenomenon of prolonged postoverdrive sinus node suppression may be disclosed (Bashour et al., 1973). Measuring the sinus node recovery time both in basal conditions and after atropine should, therefore, become a common practice.

A comment should finally be made regarding the use of atrial pacing as a method of investigating the functional capacity of the atrioventricular junction. Narula and Samet (1971) stressed that atrial pacing does not yield reliable information in that respect.
Our data fully support their views. They show that second degree atrioventricular block may, in fact, develop at rather low atrial rates (120/min) in elderly subjects without any signs or symptoms of atrioventricular or intraventricular conduction disturbances (which admittedly does not exclude latent pathology). On the other hand, like Narula and Samet (1971), we have seen patients develop transient or permanent complete heart block shortly after an atrial pacing study where they had maintained a 1:1 atrioventricular conduction up to cardiac rates higher than 150/min (Kulbertus et al., 1973). Atrial pacing studies alone can be of little help when one has to make the decision of inserting a pacemaker in patients suspected of transient atrioventricular block.

References


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