Atrial Excitability and Conduction During Rapid Atrial Pacing

VANCE J. PLUMB, M.D., ROBERT B. KARP, M.D., THOMAS N. JAMES, M.D., AND ALBERT L. WALDO, M.D.

SUMMARY Using temporary atrial wire electrodes placed at selected atrial sites, rapid atrial pacing at rates of up to 368 beats/min was used to study atrial excitability and conduction in seven patients who underwent open heart surgery. The threshold for atrial pacing was found to be an exponential function of pacing rate \( r = 0.55, p < 0.01 \), increasing threefold when the fastest pacing rates were compared with the slowest pacing rates \( p < 0.005 \). Atrial conduction times (measured from pacing to recording sites), prolonged during rapid atrial pacing both for studies conducted before institution of cardiopulmonary bypass \( p < 0.005 \) and for those done 7 days postoperatively \( p < 0.05 \). However, prolongation of conduction times always depended on achievement of a critically rapid pacing rate. During rapid atrial pacing, we observed a high incidence of alternans of the atrial electrogram (17 of 42 studies). Thus, human atrial excitability, conduction and electrogram morphology are not constant during pacing at rapid rates. Rather, at rapid pacing rates, there is depression of atrial excitability, prolongation of atrial conduction times and alternation in electrogram morphology. These findings have clinical relevance and theoretical implications for the understanding and treatment of rapid atrial rhythms.

LITTLE is known about atrial excitability and conduction in man or experimental animals during rapid atrial rhythms, particularly at rates greater than 200 beats/min. In our studies of rapid atrial pacing to interrupt atrial flutter in man,\(^1\) \(^2\) unexpectedly high stimulus strengths were often required to obtain atrial capture. Other investigators have observed or suggested that atrial conduction time is prolonged in experimental models of atrial flutter.\(^3\) \(^4\) It is not clear whether these incomplete observations are peculiar to atrial flutter or are characteristic of any rapid atrial rhythm. Learning more of the nature of atrial excitability and conduction during rapid atrial rhythms should provide better understanding of rhythms such as atrial flutter, atrial fibrillation, and ectopic (nonparoxysmal) atrial tachycardia. Therefore, we designed a study of atrial excitability and conduction in patients using fixed atrial wire electrodes.

**Methods**

Atrial pacing thresholds and atrial conduction times were determined 42 times in seven patients. Five patients were studied during open heart surgery and seven after open heart surgery. Six of the seven patients had aortocoronary artery saphenous vein bypass grafting and one had mitral valvuloplasty to correct mitral regurgitation due to mitral valve prolapse. The patients were 52–64 years old. Studies were conducted intraoperatively before cardiopulmonary bypass and on the seventh postoperative day. At the time of the intraoperative study, two of the patients had been receiving digoxin and two had been receiving propranolol. During induction and/or maintenance of anesthesia, three patients received scopolamine (0.4–0.5 mg) and all patients received diazepam, morphine, nitrous oxide, halothane, fentanyl and pancuronium. At the time of the postoperative study, three patients were receiving digoxin, one in combination with procainamide and one in combination with alpha-methyldopa. Four patients were taking no cardioactive drugs at the time of postoperative study. The subjects gave informed consent for all aspects of the study.

Patients who undergo open heart surgery at the University of Alabama in Birmingham routinely have a pair of Teflon-coated, stainless-steel wire electrodes placed on the right atrial epicardium near the sulcus terminalis. These wire electrodes are brought through the anterior chest wall for use in the diagnosis and treatment of postoperative arrhythmias in a manner previously described.\(^5\) All patients in this study also had another pair of wire electrodes placed on the Bachmann's bundle (the anterior interatrial myocardial band) and another pair placed on the posteroinferior left atrium near the coronary sinus. The usual technique of electrode placement was modified only in that the electrodes were placed before cardiopulmonary bypass.

Using bipolar threshold stimuli, defined as the minimal stimulus (mA) required to obtain and maintain constant atrial capture,\(^6\) each electrode site was paced in turn at a constant basic rate just faster than the spontaneous rate to measure a control conduction time to each of the other electrode sites. The pacing rate was then increased up to rates of 368 beats/min (range 237–368 beats/min) in a stepwise fashion in increments of 5–10 beats/min. At each pacing rate, pacing was performed for brief periods (at least 15 beats), the maximum pacing rate in each study being deter-
mined by clinical circumstances. For each increment in pacing rate, both the stimulus threshold and the conduction time from the pacing site to each of the other two recording sites were measured. Between each increment in pacing rate, the control pacing rate was resumed. When the strength of the pacing stimulus had to be increased to achieve capture, the new stimulus strength was maintained upon return to the basic (control) pacing rate, and the conduction times at the basic (control) rate were determined at this new stimulus strength. At the termination of the study, the threshold stimulus for pacing at the basic (control) rate was determined again to insure that the initially determined threshold had not changed.

Atrial pacing was performed using a Medtronic Model 1349A battery-powered pacemaker, which delivered a constant current stimulus of 2 msec duration. During atrial pacing, ECG leads I, II and III were recorded simultaneously with the stimulus artifact and with the bipolar atrial electrograms from each of the other two sites using an Electronics for Medicine DR-12 switched-beam oscillographic recorder. These data were also simultaneously recorded on magnetic tape (Honeywell model 5600) for later playback and analysis. ECGs were recorded between a bandpass of 0.1–500 Hz, and electrograms were recorded between a bandpass of 0.1–500 Hz or 12–500 Hz. The bipolar atrial wire electrodes were always electrically isolated from ground and from the recording device. Conduction times were measured from the stimulus artifact to the dominant deflection of the atrial electrogram of each recording site using a vernier measuring device with an accuracy of 1 msec at a paper recording speed of 100 mm/sec or using two Hewlett-Packard Model 5300A Universal interval counters. The latter were connected to two character generators which printed out the measurements in milliseconds on the Electronics for Medicine recorder. The accuracy of the printed-out measurements was randomly cross-checked using the vernier measuring device. To allow for a relatively constant electrophysiologic state to develop, the first eight paced beats were not included in any measurements.

To compare atrial conduction times at the various atrial pacing rates, the time from each stimulus site to each recording site at each of the several pacing rates was expressed as the percent change in conduction time compared with control:

\[
\text{conduction time (fast rate)} - \text{conduction time (basic rate)} \times 100 \\
\text{conduction time (basic rate)}
\]

The significance of conduction time prolongation was analyzed by applying the Fisher-Behrens test of significance for grouped data with unequal variance.* Regression equations were computed for the mean change in conduction times plotted as a function of pacing rate. The slopes of the regression equations for rates less than the critical pacing rate were then compared to the slopes for rates faster than the critical rate using the Fisher-Behrens test. The mean pacing threshold at the fastest rates was compared to the mean threshold at the basic pacing rate using the t test for paired means of unequal variance, and the natural logarithm of the fractional increase of threshold was plotted as a function of the pacing rate.

**Results**

**Pacing Threshold**

In all studies, pacing threshold significantly increased \( r = 0.55, p < 0.01 \) with increasing pacing rates (fig. 1). The threshold rise ranged from 25–925% when compared at the slowest and fastest rates (fig. 2). The average threshold at the fastest rate was three times that at the slowest rate \( p < 0.0005 \). The same magnitude of threshold increase was seen among patients studied before cardiopulmonary bypass as among those studied postoperatively. Figure 3 is an example of pacing threshold determination during atrial pacing at a rate of 330 beats/min. When an increase in pacing rate resulted in incomplete atrial capture (2:1 in this example), complete (1:1) capture could be reestablished by increasing the pacing stimulus strength.

The development of 2:1 atrial capture was the most frequent consequence of the rise in pacing threshold, and was seen in 42 of 50 instances. Seven patients had intermittent, irregular loss of atrial capture ("Mobitz II"). One example of "Mobitz I" loss of atrial capture was seen, manifested as atypical Wenckebach periodicity with the greatest increase in conduction time in the beat just preceding the dropped beat.

**Conduction Times**

Significant conduction time prolongation was seen with increasing pacing rate in all patients, but only after a critical pacing rate was achieved. Figure 4

![Figure 1. Comparison of the increase in threshold (plotted as the natural logarithm) and the atrial pacing rate for all studies. The solid line indicates the regression line and the dotted line the standard error.](image-url)
shows atrial conduction time determination and illustrates the prolongation of conduction times noted during critically rapid pacing (see also figure 3). Table 1 is a list of the full range of pacing rates and corresponding conduction times for the entire study from which figure 4 is taken. Figure 5 is from a representative study in which the percent change in conduction time from the pacing site on Bachmann's bundle to the recording site on the posterior left atrium is plotted over the full range of pacing rates. Note that as the pacing rate increases, conduction times are relatively constant until a critical rate is reached, at which point conduction times prolong ($p < 0.01$). As the pacing rate increases, so does the pacing threshold, from 8 to 23 mA.

When the pacing threshold increased from 7 to 15 mA at a pacing rate of 300 beats/min, the increased stimulus strength was associated with a decrease in the control conduction times at the basic pacing rate of 103 beats/min (table 1). Because pacing threshold increased as rate increased, conduction times were remeasured at the basic rate for each increase in stimulus strength. As stimulus strength increased, conduction times at the basic rate shortened (fig. 6).

**Figure 2.** Pacing threshold at the slowest pacing rate (range 102–125 beats/min) compared with the pacing threshold at the fastest pacing rate (range 273–345 beats/min) for each study.

**Figure 3.** ECG lead I recorded simultaneously with bipolar atrial electrograms ($A_{EG}$) recorded from the sulcus terminalis (ST) and Bachmann's bundle (BB) during pacing from the posteroinferior left atrium. At the previous pacing rate of 320 beats/min, the atria were completely captured at a stimulus strength of 12 mA. However, when the pacing rate was abruptly increased from the control rate to 330 beats/min, there was only 2:1 atrial capture. Illustrated are the last several beats of 2:1 atrial capture as the stimulus strength was being increased to 14 mA, at which point 1:1 atrial capture was obtained. Paper recording speed is 50 mm/sec. Time lines are at 1-second intervals. S = stimulus artifact.
Maximal suprathreshold pacing (determined by the pacing threshold at the fastest pacing rate) shortened the conduction times during pacing at the basic rate by an average of 6% (range 2–8%) from values observed when pacing at basic rate using threshold stimuli.

In the one case where Bachmann's bundle was paced before cardiopulmonary bypass, conduction

### Table 1. Conduction Times—Representative Study (Pacing Site—Sulcus Terminalis)

<table>
<thead>
<tr>
<th>Pacing rate (beats/min)</th>
<th>Stimulus (mA)</th>
<th>CT to BB (msec ± sd)</th>
<th>CT to PL (msec ± sd)</th>
</tr>
</thead>
<tbody>
<tr>
<td>103</td>
<td>7</td>
<td>65 ± 0.4</td>
<td>68 ± 0.7</td>
</tr>
<tr>
<td>111</td>
<td>7</td>
<td>67 ± 0.7</td>
<td>68 ± 1.0</td>
</tr>
<tr>
<td>120</td>
<td>7</td>
<td>68 ± 0.6</td>
<td>68 ± 1.1</td>
</tr>
<tr>
<td>129</td>
<td>7</td>
<td>68 ± 0.7</td>
<td>69 ± 1.8</td>
</tr>
<tr>
<td>141</td>
<td>7</td>
<td>69 ± 0.6</td>
<td>71 ± 3.0</td>
</tr>
<tr>
<td>150</td>
<td>7</td>
<td>69 ± 0.5</td>
<td>70 ± 1.8</td>
</tr>
<tr>
<td>160</td>
<td>7</td>
<td>70 ± 0.7</td>
<td>69 ± 0.8</td>
</tr>
<tr>
<td>170</td>
<td>7</td>
<td>70 ± 0.8</td>
<td>70 ± 0.8</td>
</tr>
<tr>
<td>180</td>
<td>7</td>
<td>69 ± 1.1</td>
<td>70 ± 1.9</td>
</tr>
<tr>
<td>192</td>
<td>7</td>
<td>69 ± 0.9</td>
<td>69 ± 0.5</td>
</tr>
<tr>
<td>200</td>
<td>7</td>
<td>69 ± 0.8</td>
<td>70 ± 1.4</td>
</tr>
<tr>
<td>214</td>
<td>7</td>
<td>69 ± 0.6</td>
<td>71 ± 1.0</td>
</tr>
<tr>
<td>225</td>
<td>7</td>
<td>69 ± 1.0</td>
<td>70 ± 1.6</td>
</tr>
<tr>
<td>235</td>
<td>7</td>
<td>69 ± 0.6</td>
<td>71 ± 1.0</td>
</tr>
<tr>
<td>240</td>
<td>7</td>
<td>70 ± 1.1</td>
<td>73 ± 1.7</td>
</tr>
<tr>
<td>252</td>
<td>7</td>
<td>69 ± 1.1</td>
<td>74 ± 1.6</td>
</tr>
<tr>
<td>267</td>
<td>7</td>
<td>70 ± 1.2</td>
<td>76 ± 2.0</td>
</tr>
<tr>
<td>273</td>
<td>7</td>
<td>74 ± 3.0</td>
<td>77 ± 1.7</td>
</tr>
<tr>
<td>285</td>
<td>7</td>
<td>73 ± 1.7</td>
<td>80 ± 1.9</td>
</tr>
<tr>
<td>300</td>
<td>15</td>
<td>76 ± 3.3</td>
<td>90 ± 1.8</td>
</tr>
<tr>
<td>103</td>
<td>15</td>
<td>60 ± 0.4</td>
<td>60 ± 0.8</td>
</tr>
<tr>
<td>311</td>
<td>15</td>
<td>72 ± 1.2</td>
<td>82 ± 1.7</td>
</tr>
</tbody>
</table>

Abbreviations: CT = conduction time; BB = Bachmann's bundle; PLA = posteroinferior left atrium.

**Figure 4.** ECG lead I recorded simultaneously with unfiltered bipolar atrial electrograms (AEG) from Bachmann's bundle (BB) and the posteroinferior left atrium (PLA) during pacing from the sulcus terminalis. Conduction times from the stimulus site to each recording site are shown (in msec) both at a pacing rate of 285 beats/min and at the basic pacing rate of 103 beats/min. Paper recording speed is 100 mm/sec. Time lines are at 1-second intervals. S = stimulus artifact.

**Figure 5.** The percent change (± sd) in conduction time and the pacing threshold are plotted as a function of pacing rate for a representative study. Note the constancy of conduction times until the pacing rate reached the critical rate of 286 beats/min, when conduction times prolonged markedly.
DEPENDING OF ATRIAL CONDUCTION
TIME ON STIMULUS STRENGTH
(REPRESENTATIVE STUDY)

FIGURE 6. Comparison of the percent change in conduction
time (± sd) at a constant pacing rate as the stimulus
strength is increased. Threshold for this pacing rate was 8
mA.

times to both distal recording sites decreased by
7-20% at intermediate pacing rates and increased at
very rapid pacing rates (fig. 7). This was the only
time we saw a significant conduction time decrease.

Studies Before Cardiopulmonary Bypass

Eleven of 13 studies showed significant conduction
time prolongation with increasing pacing rate. Con-
duction time prolongation in every study depended
on achieving a critically rapid pacing rate. The mag-
nitude of this critical pacing rate varied among
patients, and in individual patients varied among the
various pacing sites (table 2).

Figure 8 shows the grouped data on conduction
time changes for patients studied before cardio-
pulmonary bypass (13 studies in five patients)
alalyzed by the linear regression method for pacing
rates less than 240 beats/min and faster than 240
beats/min. There was a clear difference (p < 0.005) in
the slope of the slower vs the faster pacing rates, repre-
senting conduction time prolongation at the faster
rates. The choice of 240 beats/min as the critical pac-
ing rate for the group is arbitrary, with individual
variation in the critical pacing rate. For example, the
circled data points show a study in which conduction
times began to prolong at a pacing rate of only 159
beats/min (from a patient with coronary artery dis-
ease).

Postoperative Studies

Conduction time prolongation at increasing pacing
rates showed greater variability in the postoperative
studies than in the studies performed before cardio-
pulmonary bypass. All patients still showed conduction
time prolongation with increasing pacing rates,
but not invariably with each combination of pacing
and recording site. In only two patients did conduction
time prolong regardless of pacing site or recording
site. In two cases conduction times prolonged to one
distant site but not to the other site being
simultaneously recorded. In fact, in individual
patients, the incidence of postoperative studies show-
ing conduction time prolongation ranged from
25-100% (mean 77%). Table 3 shows for all post-

<table>
<thead>
<tr>
<th>Pt</th>
<th>Pacing site</th>
<th>Recording site</th>
<th>Critical rate (beats/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>ST</td>
<td>BB</td>
<td>251</td>
</tr>
<tr>
<td>2</td>
<td>ST</td>
<td>PLA</td>
<td>275</td>
</tr>
<tr>
<td>3</td>
<td>PLA</td>
<td>BB</td>
<td>221</td>
</tr>
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<td>4</td>
<td>ST</td>
<td>BB</td>
<td>216</td>
</tr>
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<td>5</td>
<td>ST</td>
<td>PLA</td>
<td>159</td>
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<tr>
<td>6</td>
<td>ST</td>
<td>BB</td>
<td>290</td>
</tr>
<tr>
<td>7</td>
<td>PLA</td>
<td>ST</td>
<td>275</td>
</tr>
<tr>
<td>8</td>
<td>PLA</td>
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<td>9</td>
<td>PLA</td>
<td>ST</td>
<td>203</td>
</tr>
<tr>
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<td>ST</td>
<td>312</td>
</tr>
<tr>
<td>11</td>
<td>BB</td>
<td>PLA</td>
<td>312</td>
</tr>
</tbody>
</table>

Mean 253.5

Abbreviations: ST = sulcus terminalis; BB = Bach-
mann's bundle; PLA = posteroinferior left atrium.

TABLE 2. Critical Pacing Rate—Prebypass Studies

<table>
<thead>
<tr>
<th>Pacing site</th>
<th>Recording site</th>
<th>Study duration (months)</th>
</tr>
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<tr>
<td>ST</td>
<td>BB</td>
<td>100</td>
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<tr>
<td>ST</td>
<td>PLA</td>
<td>66.7</td>
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<tr>
<td>PLA</td>
<td>ST</td>
<td>80</td>
</tr>
<tr>
<td>PLA</td>
<td>BB</td>
<td>80</td>
</tr>
<tr>
<td>BB</td>
<td>ST</td>
<td>100</td>
</tr>
</tbody>
</table>

Abbreviations: ST = sulcus terminalis; BB = Bach-
mann's bundle; PLA = posteroinferior left atrium; CT =
conduction time.

TABLE 3. Postoperative Pacing Studies

operative studies the incidence of conduction time prolongation to each of the other two sites when each site was paced. The grouped data on conduction time changes for all postoperative studies (29 studies in seven patients) are shown in figure 9. Again, the linear regression equation slopes are significantly different for the fast pacing rates compared with the slow pacing rates (p < 0.05), indicating conduction time prolongation at the fastest rates that occurred in 22 of the 29 postoperative studies. The circled data points show a study where conduction time prolongation began at a pacing rate of only 140 beats/min (from the patient with mitral valve prolapse), illustrating the individual variability of this phenomenon and the somewhat arbitrary nature of the choice of 240 beats/min as the critical pacing rate for the group. Table 4 shows the critical pacing rate for each postoperative study.

**Figure 8.** Rate dependence of conduction time for all precardiopulmonary bypass studies. Rates less than and equal to 240 beats/min are compared with rates greater than 240 beats/min.

**Figure 9.** Rate dependence of conduction time for all postoperative studies. Rates less than and equal to 240 beats/min are compared with rates greater than 240 beats/min.
TABLE 4. Critical Pacing Rate—Postoperative Studies

<table>
<thead>
<tr>
<th>Pt</th>
<th>Pacing site</th>
<th>Recording site</th>
<th>Critical rate (beats/min)</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>ST</td>
<td>BB</td>
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<td>ST</td>
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<td>BB</td>
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</tr>
<tr>
<td>7</td>
<td>PLA</td>
<td>ST</td>
<td>290</td>
</tr>
</tbody>
</table>

Mean 240.3

Abbreviations: ST = sulcus terminalis; BB = Bachmann's bundle; PLA = posteroinferior left atrium.

Electrical Alternans of the Atrial Electrogram

During rapid atrial pacing, electrical alternans appeared in the atrial electrogram in 17 of 42 studies (fig. 10). Electrical alternans is also present in figure 4 in the atrial electrogram recorded from the posterior left atrium. Figure 10 represents an example of alternation of the conduction time intervals that accompanied the electrical alternans in six of 17 studies. When the slower basic pacing rate was resumed, the alternans was abolished (fig. 10). Eight times, the electrical alternans occurred in only one of the two simultaneous atrial electrograms recorded from different atrial sites (figs. 4 and 10).

Discussion

Atrial Excitability

The demonstration by our study that atrial excitability becomes depressed as the atrial pacing rate is increased has not, to our knowledge, been described in man. However, stimulus thresholds are known to increase with increasing pacing rate in isolated guinea pig ventricles and isolated rat atria. It has been assumed that the rise in threshold is due to the stimuli falling in the relative refractory period of the muscle immediately below the stimulating electrodes. A similar explanation seems reasonable for our studies.

The design of our study did not permit us to detect any supernormal phase of excitability such as has been reported for canine atrium, for the canine Bachmann's bundle, and for isolated specialized atrial fibers. In one of our experiments, conduction times decreased by 7-20% while pacing Bachmann's bundle at intermediate rates (fig. 7), a finding that may indicate a supernormal phase of conduction.

The increase in atrial pacing threshold as the pacing rate is increased has clear clinical implications and may help explain certain problems in the clinical use of rapid atrial pacing to interrupt type I atrial flutter. The pacing threshold may be quite high at pacing rates approaching and exceeding the atrial rate during type I atrial flutter (240-340 beats/min). The successful interruption of type I atrial flutter by rapid atrial pacing typically requires pacing rates substantially in excess of the flutter rate, so further increases in threshold should be anticipated. In fact, the pacing threshold at very rapid pacing rates may sometimes exceed 20 mA, the maximal current output of most commercially available pacemakers. It is reasonable to suspect that some reported failures of pacing to interrupt classic (type I) atrial flutter may have been caused by using subthreshold stimuli.

Conduction Time Prolongation

We found that as the atrial pacing rate is increased, atrial conduction time almost always prolongs, but only after a critical rate is reached. Others have shown that atrial conduction might prolong at rapid atrial rates. Atrial conduction time has been reported to increase in dogs as the rate of atrial stimulation was increased beyond a critical rate. Parallel decreases in conduction velocity and the rate of rise of the action potential have been reported during rapid stimulation above a critical rate in isolated rabbit atria. Similar findings have been reported without changes in resting (diastolic) transmembrane potential. In isolated rabbit atria, the conduction velocity during induced circus movement tachycardia was shown to be much slower than conduction in the same tissue paced at slower rates. Slow atrial conduction has been demonstrated in a variety of models of canine atrial flutter.

Our experiments measured the conduction time between two atrial sites, which is a function not only of conduction velocity, but also of the length of the pathway of conduction, the stimulus strength and stimulus latency. Stimulus latency would be expected to show approximately equal degrees of conduction prolongation to all recording sites during stimulation at a fixed site and would not be expected to be a function of rate. The four studies in which conduction times prolonged to one distant site but not to another simultaneously recorded site provide indirect evidence against stimulus latency as an explanation of our results.

We presume that the decrease in conduction times...
at the basic pacing rate as the stimulus strength was increased represents the activation of an increasingly large area of atrial mass at the site of stimulation.\textsuperscript{23} The nonuniform decrease in conduction time at the basic rate as suprathreshold pacing stimuli were used may be related to variation in the absolute magnitude of the increase in stimulus strength, to variation in bipolar interelectrode distance, and to variation in the nature of the myocardial tissues\textsuperscript{24} in contact with the epicardial wires.

A lengthening of the pathway of impulse conduction would also be manifest as conduction time prolongation. Since atrial refractoriness is known to be inhomogenous,\textsuperscript{3, 6, 20, 26} an increase in rate might render parts of the prior shortest path refractory, so that conduction via a new pathway would be longer. The bipolar atrial electrogram morphology changes we observed, however, were largely a change in the amplitude of the atrial electrogram with preservation of the form recorded simultaneously at two widely spaced atrial sites, suggesting almost identical directions of atrial depolarization.

As the pacing rate increases, the stimulus may encroach upon phase 3 of the action potential, decreasing the takeoff potential of the propagated impulse compared with pacing at slower rates and resulting in a slowing of conduction velocity.\textsuperscript{27, 28} Unfortunately, in vivo atrial refractoriness has not been defined in man for rates in excess of 150 beats/min.\textsuperscript{29} At even faster rates the action potential duration and atrial refractoriness may continue to shorten.\textsuperscript{28} However, the tail of atrial refractoriness after 70% of repolarization may be very important in considering the relationship of pacing rate and atrial conduction to atrial refractoriness, and encroachment of the stimulus on repolarization may be a cause for slowing of conduction velocity and prolongation of conduction times.

Rapid atrial pacing at a critically rapid rate might be associated with a relative depolarization of transmembrane resting potential apart from repolarization (phase 3) considerations. In fact, rate-dependent rises in extracellular potassium concentration, which decrease the resting transmembrane potential of human atrium,\textsuperscript{30} have been reported during rapid stimulation of isolated rabbit atria,\textsuperscript{31} and would be expected to decrease conduction velocity at rapid pacing rates, but do not explain why prolongation of conduction time depended on achievement of a critical pacing rate.

Whatever the mechanism of prolongation of atrial conduction times with faster rates, our studies suggest a fundamental difference from the mechanism of the rise in atrial pacing threshold with increasing rate. First, the rate dependence of threshold was seen in each study, in contrast to the conduction time prolongation during rapid pacing which, while noted in the majority of studies, was not always seen. Second, the rises of threshold and increases in conduction times did not occur concurrently (fig. 5). Third,
the increase in threshold did not appear to depend on achievement of a critical pacing rate, whereas prolongation of conduction did.

Electrical Alternans

Atrial electrical alternans as demonstrated in our study during pacing at very rapid rates has also been demonstrated during rapid atrial pacing in the dog, during canine atrial arrhythmias induced by prior atrial electrical stimulation, during acetylcholine-induced atrial arrhythmias, during spontaneous canine atrial flutter, and in spontaneous human atrial flutter. Its mechanism has been attributed to local change in conduction and to the stimulation rates approaching the maximum frequency permitted by the limiting refractory period of the tissue involved. These observations suggest that the presence of electrical alternans is a function of rapid rate and is independent of the underlying mechanism of the rhythm.

Theoretical Implications

Although the mechanism of rapid atrial arrhythmias such as ectopic atrial tachycardia, atrial flutter and atrial fibrillation is uncertain, it has been proposed that conditions of slowed conduction and diminished excitability are necessary for their perpetuation. The present study suggests that since slowed conduction and diminished excitability are an intrinsic property of rapid atrial rates, rapid atrial rhythms of whatever mechanism might tend to be self-sustaining.

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The Nature of Atriosinus Conduction During Rapid Atrial Pacing in the Rabbit Heart

CHARLES R. KERR, M.D., AND HAROLD C. STRAUSS, M.D., C.M.

SUMMARY During clinical electrophysiologic investigations, the sinus node recovery time (SNRT) lengthens as the atrial pacing rate increases; after reaching a maximum value, SNRT shortens with further increases in pacing rate. This phenomenon has been ascribed to the onset of atriosinus entrance block. In this study we investigated the nature of atriosinus conduction during rapid atrial pacing and related changes in SNRT to changes in conduction. In eight rabbit sinus node preparations, the cristal terminalis was paced for 1 minute at cycle lengths ranging from 400 to 100 msec, while cristal terminalis electrograms were recorded by surface electrode and sinus node transmembrane action potentials were recorded by microelectrode. In all experiments SNRT prolonged progressively as the cycle length shortened until 2:1 atriosinus block occurred; at that point SNRT shortened. After correction for spontaneous cycle length, SNRT was inversely related to the cycle length of action potentials recorded from the pacemaker site in the sinus node (r = -0.84), indicating that SNRT depends on the number of impulses reaching the pacemaker site. In four experiments, the microelectrode was moved toward the cristal terminalis in intervals of 50-100 μ, repeating the pacing sequence at each site. The site of atriosinus block could be identified as the point at which action potential amplitude fell rapidly. With progressively shorter cycle lengths, the site of block moved progressively farther from the pacemaker site in the sinus node. Therefore, the shortening of SNRT with rapid pacing may be explained by the presence of atriosinus block and by a reduction in the number of impulses that reach the pacemaker site in the sinus node.

RAPID ATRIAL PACING is frequently used to assess sinus node automaticity in patients suspected of having sinus node dysfunction. The duration of the recovery cycle after a train of rapid atrial pacing is called the sinus node recovery time (SNRT), and its prolongation has been widely accepted as evidence of sinus node dysfunction.1-4 In general, as the rate of atrial pacing is increased, the duration of the recovery cycle increases until a maximum degree of suppression occurs; beyond this point the recovery cycle shortens as the atrial pacing rate continues to be increased. In man, maximum suppression normally occurs at 118–130 beats/min.4, 5, 6 The decrease in SNRT at faster rates may be the result of block of impulses before they can reach and depolarize the sinus node. Such a reduction in the rate of sinus node discharge would cause a reduction in sinus node suppression.4, 7-10 In cases of sinus node dysfunction, atriosinus entrance block may occur at lower pacing rates, so maximum sinus node recovery time may occur at pacing rates lower than 118 beats/min.5, 6, 9, 11, 12

Although the shortening of the SNRT at fast pacing rates has been well documented and the presence of atriosinus block has been postulated to explain this phenomenon, in vitro studies are required to corroborate these findings. In this study we used an isolated sinus node preparation from the rabbit to characterize atriosinus conduction during rapid atrial pacing. In particular, we examined the nature and site of block of retrogradely conducted impulses and the relationship between the rate of pacing and SNRT.

From the Division of Cardiology, Department of Medicine, and the Department of Pharmacology, Duke University Medical Center, Durham, North Carolina.

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Dr. Kerr is a Fellow of the Medical Research Council of Canada.

Address for correspondence: Charles R. Kerr, M.D., Division of Cardiology, Vancouver General Hospital, 2775 Heather Street, Vancouver, B.C. V5Z 3J5.

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