Catheter mapping of retrograde atrial activation
Observations during ventricular pacing and AV nodal re-entrant paroxysmal tachycardia

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A systematic study of retrograde atrial activation sequence at commonly used electrode catheter recording sites in 8 patients without, and in 4 patients with AV nodal re-entrant paroxysmal tachycardia was made. During right ventricular pacing, the retrograde atrial activation sequence was low septal right atrium—proximal coronary sinus—distal coronary sinus—high right atrium. During the episodes of paroxysmal tachycardia, a similar pattern was observed. This information should be helpful in the understanding of abnormal activation sequences in patients with paroxysmal supraventricular tachycardia in whom retrogradely conducting anomalous pathways are suspected.

Electrophysiological studies in the catheterization laboratory have contributed significantly to the understanding of mechanisms of paroxysmal supraventricular tachycardia. One group of patients with paroxysmal tachycardia is characterized by the presence of anomalous pathways, either manifest or concealed (Durrer and Wellens, 1974; Coumel and Attuel, 1974; Spurrell, Krikler, and Sowton, 1974a; Neuss, Schlepper, and Thorman, 1975; Zipes, DeJoseph, and Rothbaum, 1974). In these patients, the anomalous pathway usually constitutes the retrograde limb of an atrioventricular circus movement during paroxysmal tachycardia. Documentation of the presence of a retrogradely conducting anomalous pathway is dependent upon demonstration of abnormal retrograde activation sequences, during both ventricular pacing and paroxysmal tachycardia (Wellens and Durrer, 1974; Svenson et al., 1974; Wellens, 1975; Denes et al., 1975; Gallagher et al., 1975). For example, retrograde activation of the atria via a left lateral Kent bundle should produce early activation of the left atrium, as recorded from a catheter placed in the distal coronary sinus.

Despite the obvious usefulness of catheter electrode atrial mapping during retrograde activation, there has been no systematic study of normal retrograde activation via the His bundle and AV node, with commonly used electrode catheter recording sites. Without a clear picture of what normal activation is like, demonstration of ‘abnormal sequences’ is of questionable meaning. In this study we have attempted to define the normal pattern of retrograde activation of the atria with commonly used catheter electrode positions in patients with and without AV nodal re-entrant paroxysmal tachycardia. This information should be extremely helpful in analysis of patients with paroxysmal tachycardia in whom retrogradely conducting anomalous pathways are suspected.

Methods

The study group consisted of 12 patients with intact AV conduction undergoing electrophysiological evaluation. Group 1 consisted of 8 patients with suspected or proven conduction disease, who were classified as having normal VA conduction. These patients fulfilled the following criteria: 1) no previous history of palpitations or paroxysmal tachycardia; 2) absence of manifest antegrade pre-excitation determined by the evaluation of standard 12-lead electrocardiograms; 3) evidence of retrograde AV nodal conduction (without evidence of retrograde
TABLE 1 Clinical, electrocardiographic, and electrophysiological findings in 12 patients

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Clinical diagnosis</th>
<th>Electrocardiogram PR(s) QRS(s)</th>
<th>Electrophysiological findings during sinus rhythm (ms)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>HR PA AH HV FRP-AV FRP-AVN FRP-A ERPA ERPA</td>
</tr>
<tr>
<td><strong>Group 1</strong></td>
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<td></td>
</tr>
<tr>
<td>1</td>
<td>24</td>
<td>M</td>
<td>NHD</td>
<td>0.14 0.08</td>
<td>81 38 92 55 660 630 400 380</td>
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<tr>
<td>2</td>
<td>57</td>
<td>M</td>
<td>VHD</td>
<td>0.16 0.08</td>
<td>62 45 79 47 400 320 300 220</td>
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<tr>
<td>3</td>
<td>63</td>
<td>M</td>
<td>SSS</td>
<td>0.20 0.08</td>
<td>86 42 96 50 530 400 300 210</td>
</tr>
<tr>
<td>4</td>
<td>52</td>
<td>M</td>
<td>ASHD</td>
<td>0.14 0.08 (LVH)</td>
<td>92 44 89 40 390 &lt;360 350 290</td>
</tr>
<tr>
<td>5</td>
<td>34</td>
<td>F</td>
<td>Sarcoi-dosis</td>
<td>0.14 0.08</td>
<td>97 35 87 34 320 &lt;250 250 230</td>
</tr>
<tr>
<td>6</td>
<td>57</td>
<td>M</td>
<td>ASHD</td>
<td>0.16 0.08</td>
<td>53 43 120 40 450 340 340 240</td>
</tr>
<tr>
<td>7</td>
<td>64</td>
<td>M</td>
<td>ASHD</td>
<td>0.18 0.12 (RBBB &amp; LASH)</td>
<td>90 42 76 86 330 &lt;310 310 280</td>
</tr>
<tr>
<td>8</td>
<td>81</td>
<td>M</td>
<td>ASHD</td>
<td>0.24 0.12 (RBBB &amp; LASH)</td>
<td>64 59 145 65 665 450 280 230</td>
</tr>
<tr>
<td><strong>Group 2</strong></td>
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<td></td>
</tr>
<tr>
<td>9</td>
<td>25</td>
<td>F</td>
<td>NHD</td>
<td>0.14 0.08</td>
<td>91 41 58 35 545 S=545 S=250 250 180</td>
</tr>
<tr>
<td>10</td>
<td>53</td>
<td>M</td>
<td>ASHD</td>
<td>0.18 0.08 (LVH)</td>
<td>99 26 122 54 470 S=470 S=310 310 280</td>
</tr>
<tr>
<td>11</td>
<td>55</td>
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<td>ASHD</td>
<td>0.16 0.08</td>
<td>62 40 87 55 520 S=420 S=285 260 240</td>
</tr>
<tr>
<td>12</td>
<td>77</td>
<td>M</td>
<td>HCVD</td>
<td>0.16 0.08 (LVH)</td>
<td>96 50 91 49 440 S=440 S=250 250 180</td>
</tr>
</tbody>
</table>

**Abbreviations**: NHD = No organic heart disease; VHD = valvular heart disease; SSS = sick sinus syndrome; ASHD = arteriosclerotic heart disease; HCVD = hypertensive cardiovascular disease; LVH = left ventricular hypertrophy; RBBB = right bundle-branch block; LASH = left anterior superior hemiblock; FRP-AVN = AV nodal functional refractory period; ERPAVN = AV nodal effective refractory period; FRP-A = atrial functional refractory period; ERP-A = atrial effective refractory period; S = slow pathway; F = fast pathway.

anomalous pathway conduction) during right ventricular pacing, as manifested by appropriate increases in VA conduction times with increasing pacing rates (10 beats/min increments) and also during coupled ventricular stimulation. Group 2 included 4 patients with documented reproducible dual pathway AV nodal re-entrant paroxysmal supraventricular tachycardia (PSVT). These patients fulfilled criteria previously described by our laboratory (Denes et al., 1973). Clinical and electrophysiological data concerning all patients are presented in Table 1.

All patients were studied in the postabsorptive, non-sedated state. Informed consent was obtained in all patients. All drugs were discontinued 48 hours before the study. A bipolar electrode catheter was introduced percutaneously into the right femoral vein and placed across the tricuspid valve to record His bundle and low septal right atrial electrograms (Scherlag et al., 1969). A quadrupolar electrode catheter was introduced into the left femoral vein, advanced into the high lateral right atrium, and positioned at the superior vena cava right atrial junction. The two distal pairs of electrodes were used for atrial pacing and the two proximal electrodes to record high right atrial electrograms. A bipolar electrode catheter, introduced via a right antecubital vein, was positioned in the distal coronary sinus to record posterior-inferior left atrial electrograms. The position of the catheter in the coronary sinus was confirmed by its posterior location in the lateral view and by the presence of inverted P waves in leads II and III during stimulation. In addition, a 5F bipolar electrode was introduced through the same antecubital vein and positioned at the right ventricular apex for ventricular pacing. Bipolar interelectrode distance for all catheters was 1 cm.

Refractory periods were determined using the extra-stimulus technique (Wit et al., 1970). Stimuli were 2 ms in duration and twice diastolic threshold in intensity, and supplied by a digital programmable pulse generator1. Intracardiac electrograms, including high right atrium (HRA) and coronary sinus (CS), as well as electrocardiographic standard leads I, II, III, and chest lead V1, were simultaneously displayed on an oscilloscope and recorded on a multichannel photographic recorder4 at paper speeds of 100 and 200 mm/s.

**Electrophysiological definitions**

Previous definitions of PA, AH (LSRA-H), and HV intervals, as well as AV nodal functional and effective refractory periods (FRP and ERP) were used (Denes et al., 1974). HRA1, LSRA1, CS1, H1, and V1 were, respectively, high right atrial, low septal right atrial, coronary sinus, His bundle, and ventricular electrograms of the sinus or driven beats (S1). HRA2, LSRA2, CS2, H2, and V2 were electrograms in response to the extrastimulus (S2).

**Procedure**

Group 1 patients 1) The antegrade sequence of atrial activation was mapped during sinus rhythm and the following intervals measured: a) P-HRA: from the beginning of the P wave in the electrocardiographic leads to the high right atrial electrogram. b) P-LSRA: from

1M. Bloom, Philadelphia, Pa.
the beginning of the P wave to the low septal right atrial
electrogram. c) P-CS: from the beginning of the P wave
to the coronary sinus electrogram. d) P-H: from the
beginning of the P wave to the His bundle electrogram.

2) The right atrium (RA) was paced at rates slightly
faster than sinus rhythm and then increased in 10
beats/min increments until AV nodal Wenckebach
periods resulted.

3) The right ventricle (RV) was paced at rates slightly
above the control sinus rate to ensure constant retrograde
conduction, and the rate was then increased in 10
beat/min increments until retrograde Wenckebach
periods were noted.

4) Ventriculoatrial conduction times (VA) were
measured from the ventricular stimulus artefact to the
first high frequency potential of the LSRA electrogram.
ΔVA time was defined as the difference in VA conduction
times obtained by pacing the RV at rates slightly
above spontaneous sinus rate and those obtained at a rate
slightly slower than that producing retrograde Wenckebach
periodicity.

5) The retrograde sequence of atrial activation was
mapped during 1:1 right-ventricular pacing and during
coupled ventricular pacing at cycle lengths of 600–900
ms. The following intervals were measured: a) S-LSRA:
from the stimulus artefact to the first high frequency
deflection of the low septal right atrial electrogram. b)
S-CS: from the stimulus artefact to the coronary sinus
electrogram. c) S-HRA: from the stimulus artefact to the
right high atrial electrogram. In two of the patients,
in addition to distal coronary sinus, we also mapped
the proximal coronary sinus. The latency between
the stimulus artefact and the ventricular electrogram was
insignificant in all cases. For purposes of analysis, the
retrograde atrial activation sequence was measured at a
paced ventricular cycle length of 600 ms (100 beats/min)
in all patients.

In group 2 patients, in addition to the above procedure,
the retrograde sequence of atrial activation was studied
during episodes of induced paroxysmal supraventricular
tachycardia, and the following intervals were measured:
a) V-LSRA: from the onset of ventricular depolarization
in the surface electrocardiogram to the first high
frequency potential of the low septal right atrial
electrogram. This interval could have a negative value in those
cases where LSRA preceded the onset of the QRS. b) V-CS:
from the onset of ventricular depolarization on the surface
electrocardiogram to the onset of ventricular
depolarization to the first high frequency
potential in the high right atrial electrogram. V-HRA:
from the onset of ventricular depolarization to the first
high frequency potential in the right high atrial
electrogram. All intervals represent the average of 10 beats.

Results

Antegrade sequence of atrial activation
Individual values, as well as mean values for
P-HRA, P-LSRA, P-CS, and P-H for both groups
1 and 2 are presented in Table 2. In every patient,
the sequence of antegrade atrial activation was as follows:
HRA, LSRA, and then CS.

Retrograde atrial activation sequences
During right ventricular pacing with 1:1 VA
conduction, a consistent pattern of retrograde atrial
activation was observed in both groups (Fig. 1,
Table 3). In all patients except one, the sequence of
retrograde atrial activation was as follows: low
septal right atrium, coronary sinus, and high right
atrium. In the one exception (Case 12), high right
atrium slightly preceded coronary sinus. In the 2
patients in whom proximal coronary sinus was also
mapped, this followed low septal right atrium by 10

<table>
<thead>
<tr>
<th>Case No.</th>
<th>P-HRA</th>
<th>P-LSRA</th>
<th>P-CS</th>
<th>P-H</th>
<th>W (beats/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
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<td>12</td>
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<td>70</td>
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<td>13</td>
<td>45</td>
<td>80</td>
<td>124</td>
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<td>15</td>
<td>59</td>
<td>94</td>
<td>204</td>
<td>120</td>
</tr>
<tr>
<td>Mean ± SEM</td>
<td>15 ± 2</td>
<td>42 ± 2·8</td>
<td>81 ± 3·5</td>
<td>140 ± 10·4</td>
<td>155 ± 13·8</td>
</tr>
</tbody>
</table>

| Group 2  |       |        |      |     |               |
| 9        | 10    | 41     | 57   | 99  | 180           |
| 10       | 5     | 26     | 89   | 148 | 160           |
| 11       | 19    | 40     | 98   | 127 | 170           |
| 12       | 15    | 50     | 80   | 141 | 190           |
| Mean ± SEM | 12 ± 3 | 39 ± 5 | 81 ± 8·8 | 128 ± 11 | 175 ± 6·4 |

Abbreviations: See text for definition of P-HRA, P-LSRA, P-CS, and P-H. W—Paced rate producing AV nodal Wenckebach periods.
FIG. 1 Case 4. Retrograde atrial activation sequence during right ventricular pacing at cycle length of 600 ms (100 beats/min). Shown are leads I, II, III, and V1. HRA = high right atrial electrogram; CS = coronary sinus electrogram; LSRA = low septal right atrial electrogram; S = stimulus artefact. In this and subsequent illustrations, paper speed was 100 mm/s, and time lines are at 1 s.

FIG. 2 Case 6. The retrograde atrial activation sequence during right ventricular pacing at increasing rates is shown. The VA conduction time is represented on the ordinate in ms. The cycle length of ventricular pacing is expressed on the abscissa. Note that the sequence of activation LSRA-CS-HRA does not change with increasing pacing rates.

FIG. 3 Case 6. Coupled right ventricular pacing at cycle length of 850 ms. Note that at $V_1V_2$ of 290 ms, the retrograde atrial activation sequence is identical to the basic driving rate.
Catheter mapping of retrograde atrial activation

Cycle length 850 ms
- A1-A2
- V2-HRA
- V2-CS
- V2-LSRA

FIG. 4 Case 6. Coupled right ventricular pacing at cycle length of 850 ms. On the left (ordinate), A1-A2 is plotted against V1V2. On the right (ordinate), V2-A2 is plotted against V1V2. Note that the retrograde atrial activation sequence remains similar, despite appropriate increases in V2-A2.

and 15 ms, and preceded distal coronary sinus by 40 and 30 ms, respectively.

The retrograde atrial activation sequence did not change with ventricular pacing at increasing rates. An example is shown in Fig. 2. VA conduction times were plotted against right ventricular pacing rates (expressed in cycle length). The right ventricle was paced at 70 beats/min, and then in 10 beats/min increments until retrograde Wenckebach resulted at a rate of 170 beats/min. The VA time remained unchanged until 670 ms (rate of 90/min) and then increased progressively up to the point of retrograde Wenckebach. The sequence of retrograde atrial activation remains unaltered at all pacing rates.

The retrograde sequence of atrial activation was also analysed during right ventricular coupled stimulation. Fig. 3 and 4 are representative examples. Activation sequences remained unchanged, despite appropriate increases in V2-A2.

Retrograde sequence of atrial activation during paroxysmal supraventricular tachycardia

The low septal right atrium was activated earliest during induced episodes of paroxysmal supraventricular tachycardia in all patients (Fig. 5 and Table 4). In 3 of the 4 patients, coronary sinus electrograms preceded high right atrial electrograms. In one patient (Case 12), the high right atrial electrogram preceded the coronary sinus electrogram by 2 ms.

Discussion

The human heart is frequently capable of retrograde conduction. This ability has been demonstrated during spontaneous junctional (Puech and Grolleau, 1972; Damato and Lau, 1969) and ventricular rhythms (Winternitz and Langendorf, 1944; Kistin and Landowne, 1951; Kistin, 1961), and during electrophysiological studies using ventricular pacing (Damato, Lau, and Bobb, 1970; Goldreyer and Bigger, 1970). The reported frequency of the low septal right atrium (LSRA) precedes coronary sinus (CS) and high right atrium (HRA).

FIG. 5 Case 10. Retrograde atrial activation sequence during induced AV nodal re-entrant PSVT. Note that low septal right atrium (LSRA) precedes coronary sinus (CS) and high right atrium (HRA).
TABLE 3 Retrograde conduction times (in ms) during ventricular pacing at cycle length of 600 ms

<table>
<thead>
<tr>
<th>Case No.</th>
<th>S-LSRA</th>
<th>S-CS</th>
<th>S-HRA</th>
<th>Δ VA</th>
<th>W (beats/min)</th>
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<td>Group 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>1</td>
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<td>8</td>
<td>220</td>
<td>270</td>
<td>283</td>
<td>25</td>
<td>140</td>
</tr>
<tr>
<td>Mean ± SEM</td>
<td>167 ± 15.5</td>
<td>196 ± 17.9</td>
<td>216 ± 17.8</td>
<td>32 ± 8.4</td>
<td>160 ± 6</td>
</tr>
</tbody>
</table>

Group 2

|        |        |      |       |      |              |
| 9        | 135    | 155  | 195   | 25   | 190          |
| 10       | 220    | 232  | 252   | 45   | 150          |
| 11       | 160    | 172  | 192   | 35   | 150          |
| 12       | 175    | 203  | 201   | 50   | 170          |
| Mean ± SEM | 172.5 ± 18 | 190.5 ± 17 | 210 ± 14 | 39 ± 5.5 | 165 ± 9.6 |

**Abbreviations**: See text for definitions of S-LSRA, S-CS, S-HRA, and Δ VA. W = paced rate producing retrograde AV nodal Wenckebach periods.

1967; Kistin, 1961; Lister et al., 1967; Narula, 1974; Goldreyer and Bigger, 1970). The sequence of retrograde atrial activation in man has received less attention. Kraus, Yahini, and Neufeld (1966) using indirect pressure recordings from both atria, showed that the left atrium was activated first in one patient with premature ventricular contractions and intact VA conduction. Massumi et al. (1969) studied 18 patients with intact VA conduction during right ventricular pacing. In 11 patients, the right atrium was activated an average of 15 ms before the left atrium; in 6 patients, both atria depolarized almost simultaneously, and in one, the left atrium preceded the right atrium. During left ventricular pacing in 2 out of 3 patients, the left atrium was activated first. In their study (Massumi et al., 1969), a bipolar electrode catheter was used to explore the right atrium and an oesophageal bipolar electrode the left atrium. The location of the recording intracardiac electrodes was not precisely delineated. More recently, Svenson et al. (1974) reported on one patient without pre-excitation: during RV pacing, the low septal right atrial electrogram preceded low lateral right atrium and lateral left atrium (recorded from the distal coronary sinus). This observation is in agreement with our findings, that in both groups of patients, low septal right atrial activation always preceded distal coronary sinus and high right atrial activation. The demonstration of early activation of the low septal right atrium is consistent with previous experimental observation in animals. Thus, Spach et al. (1971), studying the retrograde excitation sequence in isolated hearts of dogs and rabbits, found that initial atrial activation occurred in an area over the posteroinferior region of the AV node, and that from there, the wave front spread over the lower septum. Mignone and Wallace (1966), by mapping the endocardial surface of the right atrium in dogs with a bipolar probe electrode, found that during ventricular pacing with retrograde transmission, the earliest atrial activity was recorded near the ostium of the coronary sinus and approximately 1 cm above the AV ring; 3 to 12 ms later, atrial tissue at the region where a chronically His electrode was implanted was activated (LSRA). In dogs, Damato et al. (1970) studied the retrograde atrial mapping during RV or His bundle pacing, and found that the lower portion of the atrial septum was activated first, followed next by Bachman's bundle, the coronary sinus, and then the sinus node.

TABLE 4 Retrograde atrial activation sequence during PSVT (in ms)

<table>
<thead>
<tr>
<th>Case No.</th>
<th>V-LSRA</th>
<th>V-CS</th>
<th>V-HRA</th>
</tr>
</thead>
<tbody>
<tr>
<td>9</td>
<td>-5</td>
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</tr>
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<td>25</td>
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</tr>
<tr>
<td>12</td>
<td>75</td>
<td>98</td>
<td>96</td>
</tr>
<tr>
<td>Mean ± SEM</td>
<td>36 ± 20.5</td>
<td>63 ± 17</td>
<td>78 ± 12.1</td>
</tr>
</tbody>
</table>

**Abbreviations**: See text for definitions of V-LSRA, V-CS, and V-HRA.

**Retrograde atrial activation during AV nodal re-entrant PSVT**

Current evidence suggests that the underlying mechanism of most paroxysmal supraventricular tachycardia is an intra-AV nodal circus movement caused by functional longitudinal dissociation
tachycardia, (Denes H1-H2) (A1-A2, tachycardia (Denes pvtcatachycardia:paroxysmal animal studies suggest entrance et reported activated be each retrograde during atria was In that of episodes Clinical implications anomalous connexion finding sinu, and and pacing of this In Though pathway. grams of the above suggests the presence of an anomalous connexion between ventricles and atria. For example, early activation of the coronary sinus suggests the presence of a left-sided anomalous pathway. Though low lateral right atrial electrograms were not recorded during this study, it seems reasonable to postulate that early activation of the low lateral right atrium (relative to the low septal right atrium) would reflect the presence of a lateral right-sided extranodal pathway.

In a patient with recurrent paroxysmal supraventricular tachycardia, it seems crucial to record retrograde activation sequences during induced episodes of tachycardia. The presence of an abnormal retrograde activation sequence suggests the participation of a retrogradely conducting anomalous pathway in an AV re-entrant circus movement. In a patient with manifest antegrade pre-excitation, the circus movement would generally be consistent with the electrocardiographically predicted site of anomalous pathway, e.g. early activation of coronary sinus in a patient with type A pre-excitation. The finding of early activation of the atrium contralateral to the predicted site of pre-excitation, e.g. early activation of the coronary sinus in a patient with type B pre-excitation, could suggest the presence of a second anomalous pathway used for retrograde conduction (Spurrell, Kriessler, and Sowton, 1974b; Denes et al., 1975). The presence of an abnormal retrograde activation sequence in a patient without manifest antegrade pre-excitation suggests the presence of a concealed (unidirectionally conducting) Kent bundle (Spurrell et al., 1974a; Zipes et al., 1974a).

These considerations seem vital when considering a patient for anomalous pathway ablation, because of recurrent paroxysmal supraventricular tachycardia.

References
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