The Diagnosis and Management of Supraventricular Tachycardia by Transesophageal Cardiac Stimulation and Recording*

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Twenty-two consecutive patients underwent esophageal stimulation and recording for the diagnosis and management of supraventricular tachycardia. In 13 of these patients, the resting electrocardiogram was normal and in nine it showed pre-excitation. Of the 13 patients with a normal resting electrocardiogram, supraventricular tachycardia was initiated in all. Seven patients had a ventricular-to-atrial interval >70 ms during supraventricular tachycardia suggesting the presence of a concealed accessory pathway, and six patients had a ventricular-to-atrial interval <70 ms during supraventricular tachycardia suggesting reentry within the atrioventricular node. Supraventricular tachycardia was initiated in four of nine patients with pre-excitation on the resting electrocardiogram and the accessory pathway was confirmed by a ventricular-to-atrial interval of >70 ms during supraventricular tachycardia in these four patients. Atrial fibrillation was initiated in eight of the nine patients with pre-excitation on the resting electrocardiogram and the shortest R-R interval during atrial fibrillation was measured. The response to therapy was assessed in seven of these nine patients by further measurement of the shortest R-R interval during atrial fibrillation following treatment. Esophageal stimulation and recording provides a simple noninvasive procedure which can be utilized as a screening technique to identify patients with intranodal reentry and those with reentry utilizing an accessory pathway. Sequential assessment of the response to therapy, especially in those patients with pre-excitation, is of considerable value in treatment.

The development of the technique of intracardiac stimulation and recording combined with programmed stimulation of the heart has resulted in a greater understanding of the mechanisms responsible for supraventricular tachycardia and has led to a more rational approach in the management of this disturbance of rhythm. It has been demonstrated that the major underlying causes of supraventricular tachycardia are reentry confined to the atrioventricular node or reentry utilizing an atrioventricular bypass tract which may be overt or concealed. Sinus nodal and atrial reentrant tachycardia are relatively uncommon.1 Life-threatening arrhythmias and sudden death have been reported to occur in patients exhibiting an accessory atrioventricular pathway.2 Those at risk of such arrhythmias demonstrate a very short anterograde effective refractory period of the accessory pathway, which may lead to a rapid ventricular response should atrial fibrillation or flutter occur.3 It is important, therefore, to distinguish between supraventricular tachycardia due to atrioventricular nodal reentry and that which arises as a result of the presence of an accessory pathway. The technique of transesophageal recording and stimulation permits noninvasive induction of supraventricular tachyarrhythmias45 and measurement of the anterograde refractory period of an accessory pathway when present.

Accordingly, a group of patients were studied utilizing this technique in order to assess its value in the investigation and treatment of supraventricular tachycardia.

**Patients and Methods**

The study group consisted of 22 consecutive patients: 14 females and eight males aged 14 to 62 years, with a mean age of 33.9 years. Twenty-one patients presented with palpitations and four patients had syncopeal episodes in addition. One patient had a syncopal episode without palpitations. Of the 22 patients, 13 had a normal resting electrocardiogram with no evidence of pre-excitation and nine patients had electrocardiographic evidence of pre-excitation. Routine physical examination, chest x-ray film and electrocardiogram were performed on all patients. Cardioactive medications were discontinued before the study in all patients for a period compatible with the requirements of their respective half-lives. The patients were studied in the fasting state with an intravenous line in situ. A Medtronic bipolar endocardial coronary sinus pacing electrode No. 6992, which has an interelectrode distance of 29 mm, was passed via the nose to the distal esophagus with the patient lying supine and in the non-sedated state. The lead was directed into the esophagus by tilting the patient's head forward and advancing it as the patient swallowed. The esophageal lead was positioned where the largest and sharpest atrial deflection was recorded. Recordings were taken in sinus rhythm at paper speeds of 50 and 100 mm/s. Incremental atrial pacing was carried out from 85 to 200 beats per minute or to the development of atrioventricular block using a

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current strength varying between 14 and 22 mA and a pulse duration of 9.9 ms. When this failed to initiate supraventricular tachycardia, bursts of atrial pacing at 200 to 300 beats per minute were undertaken. Incremental atrial pacing and bursts of atrial pacing were repeated following the administration of 1 mg atropine intravenously if supraventricular tachycardia was not initiated in the basal state. When supraventricular tachycardia was initiated, the cycle length of the tachycardia, the ventricular-to-atrial time (the shortest interval recorded from the onset of ventricular activation to the rapid component of the atrial electrogram recorded from the esophageal lead) and the atrial-to-ventricular time were measured. If the ventricular-to-atrial interval was >70 ms, suggesting the presence of an accessory pathway, attempts to induce atrial fibrillation by bursts of atrial pacing at 333, 750, 1,000 and 1,500 beats per minute were undertaken in those patients demonstrating pre-excitation on their resting electrocardiogram. The anterograde refractory period of the accessory pathway was measured by the shortest R-R interval during consecutive pre-excitation beats during atrial fibrillation (Fig 1-3).

The study was then repeated in eight patients following the oral administration of sotalol for 48 h or longer. If supraventricular tachycardia could be easily initiated or if the anterograde refractory period of the accessory pathway failed to lengthen in those patients with pre-excitation, the study was repeated after at least a further 48 hours. The sotalol was combined with another antiarrhythmic agent when necessary and the study was repeated at 48-h intervals or longer until failure or difficulty in initiating supraventricular tachycardia occurred combined with an increase in the anterograde refractory period of the accessory pathway in those patients with pre-excitation and a short anterograde refractory period of their accessory pathway. In two patients with Wolf-Parkinson White syndrome, the study was repeated following oral disopyramide for 48 h or longer until a therapeutic dose was reached which satisfactorily prolonged the anterograde refractory period of the accessory pathway. The response to therapy with sotalol was assessed by repeat esophageal stimulation and recording after 48 h or longer in one patient with atrioventricular node reentry and in two patients with a concealed accessory pathway. Of the remaining five patients with a concealed accessory pathway, three patients were treated with sotalol and the response to therapy was assessed clinically. Two patients did not require therapy. Of the remaining five patients with atrioventricular node reentry, one patient received digoxin, one patient received propranolol, one patient received sotalol, and two patients did not require therapy. Response to therapy was assessed clinically in these five patients.

RESULTS

Supraventricular tachycardia was initiated in the pretreatment state in 17 of 22 patients and in only one of those ten patients who were restudied following oral drug treatment. In one patient, supraventricular tachycardia could not be initiated in the pretreatment state. However, in this patient, supraventricular tachycardia was initiated following orally administered disopyramide. The cycle length of the tachycardia in the pretreatment state ranged from cycle length 370 ms to cycle length 260 ms.

The ventricular-to-atrial interval in the pretreatment state was >70 ms in all four patients with evidence of pre-excitation on the resting electrocardiogram in whom supraventricular tachycardia could be initiated, suggesting the presence of an accessory pathway. In five patients, supraventricular tachycardia could not be initiated and, therefore, the ventricular-to-atrial interval could not be measured. In the 13 patients with a normal resting electrocardiogram the ventricular-to-atrial interval was >70 ms in seven patients, suggesting the presence of a concealed accessory pathway and <70 ms in six patients, excluding participation of an atrioventricular accessory pathway in the tachycardia circuit.

Atrial fibrillation was initiated in the pretreatment state in eight of nine patients with pre-excitation on the resting electrocardiogram and in six of seven patients following treatment. Two patients did not require a repeat study.
FIGURE 2. Esophageal and electrogram tracings recorded during supraventricular tachycardia (SVT) due to reentry using the atrioventricular node as the antegrade limb of the circuit and an accessory pathway as the retrograde limb. During the supraventricular tachycardia the ventricular-to-atrial (V to A) conduction time is >70 ms.

FIGURE 3. Electrocardiogram recording following induction of atrial fibrillation by esophageal pacing at cycle length 60 ms in a patient with pre-excitation with an accessory pathway of the Kent bundle type. This is a continuous tracing showing the spontaneous reversion from atrial fibrillation to normal sinus rhythm (NSR). The arrows indicate complexes conducted exclusively through the atrioventricular node (AVN).
In those patients with pre-excitation on their resting electrocardiogram the anterograde refractory period of the accessory pathway varied between 360 to 140 ms before treatment and 300 to 270 ms following treatment. The anterograde refractory period of the accessory pathway in the pretreatment state could not be measured in one patient and following drug treatment in one patient because of failure to initiate atrial fibrillation. The patients with the anterograde refractory period of the accessory pathway of 330 and 360 ms did not have a repeat study.

Repeat esophageal stimulation and recording studies were performed in seven patients with pre-excitation on the resting electrocardiogram, in two patients with a concealed accessory pathway and in one patient with atrioventricular node reentry until a satisfactory response to therapy occurred. This was indicated by either a satisfactory lengthening of the anterograde refractory period of the accessory pathway combined with failure or difficulty in initiating supraventricular tachycardia in those patients with pre-excitation or failure to initiate supraventricular tachycardia following treatment in those patients with reentry using a concealed accessory pathway or atrioventricular node reentry. The patient with atrioventricular node reentry had a poor response to treatment.

Ten patients were followed up by assessment of their clinical response, six of whom were on long-term treatment and four of whom were not treated. Three patients had no further episodes of palpitations following treatment and in three patients palpitations became less frequent (Table 1).

**DISCUSSION**

The limitation of the surface electrocardiogram in the diagnosis of atrial dysrhythmia is well recognized. The technique of esophageal recording and pacing using a bipolar electrode with a wide interelectrode distance and thus lower pacing thresholds has been shown by Gallagher et al. to minimize patient discomfort during left atrial pacing. This affords a noninvasive technique for differentiating between intranodal reentry and reentry utilizing an accessory pathway and for measurement of the anterograde refractory period of the accessory pathway in those patients with overt pre-excitation. Esophageal pacing and recording is generally considered a safe procedure but instances of ventricular and diaphragmatic pacing have been reported as complications.

A study of Binkley et al. demonstrated that the esophageal recording electrode rests close to the posterior wall of the left atrium. The ability of the posterior wall of the left atrium to conduct electrical activity in conjunction with the very close proximity of the esophageal catheter to this structure makes the esophageal electrode a valid and useful technique for recording electrical activity of the left atrium. The site of maximal esophageal electrocardiograms or the patient's height may indicate the location of minimum threshold.

In this study, all patients experienced lower retrosternal discomfort which necessitated the administration of pethidine combined with diazepam intravenously. During the study this discomfort was related to the strength of the current used to stimulate the left atrium and was more marked when a current strength of >20 mA was used. The study was found to be acceptable by all patients.

In those patients with evidence of pre-excitation on their resting electrocardiogram, the presence of the accessory pathway was confirmed in five patients. In two patients in whom supraventricular tachycardia could not be initiated, the bypass tract was thought to be located between the right atrium and right ventricle, as deduced from the delta wave polarity in the surface electrocardiogram. The electrode pacing site on the left atrium was, therefore, located at a distance from the bypass tract which may explain the failure to initiate the tachycardia.

The induced episodes of supraventricular tachycardia, when not self-terminating, could be terminated in all but one patient by overdrive left atrial pacing via the esophageal electrode. This allowed repeated assessment of the ability to induce supraventricular tachycardia before and after drug treatment. The failure to initiate supraventricular tachycardia following drug therapy or the prolongation of the tachycardia cycle length in those patients in whom supraventricular tachycardia was initiated appeared to be a good indicator of response to drug therapy, since all of these patients have continued to remain asymptomatic on their drug treatment. It has been shown that one of the mechanisms underlying the development of atrial fibrillation with a very rapid ventricular response which has deteriorated into ventricular fibrillation is an initiating prolonged episode of supraventricular tachycardia, giving rise to disorganization of the atrial muscle, thus emphasizing the importance of appropriate drug therapy to prevent initiation of supraventricular tachycardia in these patients.

Seven of our patients demonstrating a normal QRS on the resting electrocardiogram showed evidence of reentry via a concealed accessory pathway during supraventricular tachycardia. However, six of our patients had evidence of atrioventricular nodal reentry. Differentiation of these two mechanisms is important when planning treatment, as many of the therapeutic agents that are useful in the treatment of supraventricular tachycardia related to atrioventricular node reentry may cause perpetuation of supraventricular arrhythmias in patients with supraventricular tachycardia, using a concealed accessory pathway as the retro-
Table 1—Characteristics of SVT and Atrial Fibrillation and Response to Treatment in 22 Patients*

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>ECG</th>
<th>CL (ms)</th>
<th>SVT V-A Interval (ms)</th>
<th>Atrial Fibrillation Shortest R-R Interval (ms)</th>
<th>Mechanism of SVT</th>
<th>Treatment</th>
<th>Outcome</th>
<th>Duration of Follow-Up (mon)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F</td>
<td>WPW</td>
<td>280-310</td>
<td>No SVT</td>
<td>170 ... 235 ... 280</td>
<td>Reentry using manifest AP</td>
<td>Sotalol, 160 mg twice daily</td>
<td>Palpitations × 1</td>
<td>24</td>
</tr>
<tr>
<td>2</td>
<td>F</td>
<td>WPW</td>
<td>300-320</td>
<td>No SVT</td>
<td>130 ... 240 ... 300</td>
<td>Reentry using manifest AP</td>
<td>Sotalol, 160 mg twice daily Quinidine Durules, 200 mg twice daily</td>
<td>Asymptomatic</td>
<td>27</td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>WPW</td>
<td>310</td>
<td>No SVT</td>
<td>160 ... No A fib ... 300</td>
<td>Reentry using manifest AP</td>
<td>Sotalol, 80 mg twice daily</td>
<td>DNR</td>
<td>...</td>
</tr>
<tr>
<td>4</td>
<td>F</td>
<td>WPW</td>
<td>No SVT</td>
<td>85</td>
<td>250 ... No A fib ...</td>
<td>Reentry using manifest AP</td>
<td>Sotalol, 160 mg twice daily</td>
<td>Asymptomatic</td>
<td>20</td>
</tr>
<tr>
<td>5</td>
<td>M</td>
<td>WPW</td>
<td>330-340</td>
<td>No SVT</td>
<td>150 ... 120 ... 140 ... 300</td>
<td>Reentry using manifest AP</td>
<td>Disopyramide retard, 375 mg twice daily</td>
<td>Palpitations × 1</td>
<td>17</td>
</tr>
<tr>
<td>6</td>
<td>M</td>
<td>WPW</td>
<td>No SVT</td>
<td>360</td>
<td>120 ... 230 ... 270</td>
<td>Reentry using manifest AP</td>
<td>Disopyramide retard, 500 mg twice daily</td>
<td>Asymptomatic</td>
<td>13</td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>WPW</td>
<td>No SVT</td>
<td>170</td>
<td>280 ...</td>
<td>Reentry using manifest AP</td>
<td>Disopyramide retard, 500 mg twice daily</td>
<td>Asymptomatic</td>
<td>26</td>
</tr>
<tr>
<td>8</td>
<td>M</td>
<td>WPW</td>
<td>No SVT</td>
<td>...</td>
<td>330 ...</td>
<td>Reentry using concealed AP</td>
<td>Sotalol, 120 mg twice daily</td>
<td>Asymptomatic</td>
<td>16</td>
</tr>
<tr>
<td>9</td>
<td>F</td>
<td>WPW</td>
<td>...</td>
<td>360</td>
<td>...</td>
<td>Reentry using concealed AP</td>
<td>Sotalol, 160 mg twice daily</td>
<td>Asymptomatic</td>
<td>18</td>
</tr>
<tr>
<td>10</td>
<td>F</td>
<td>N</td>
<td>270</td>
<td>No SVT</td>
<td>85 ...</td>
<td>Reentry using concealed AP</td>
<td>Sotalol, 160 mg at night</td>
<td>Palpitations less frequent</td>
<td>13</td>
</tr>
<tr>
<td>11</td>
<td>F</td>
<td>N</td>
<td>310</td>
<td>310</td>
<td>110 ...</td>
<td>Reentry using concealed AP</td>
<td>Sotalol, 80 mg mane</td>
<td>Asymptomatic</td>
<td>7</td>
</tr>
<tr>
<td>12</td>
<td>F</td>
<td>N</td>
<td>320</td>
<td>130</td>
<td>...</td>
<td>Reentry using concealed AP</td>
<td>Sotalol, 40 mg twice daily</td>
<td>Palpitations decreased</td>
<td>8</td>
</tr>
<tr>
<td>13</td>
<td>F</td>
<td>N</td>
<td>330</td>
<td></td>
<td>165 ...</td>
<td>Reentry using concealed AP</td>
<td>Sotalol, 40 mg twice daily</td>
<td>Asymptomatic</td>
<td>7</td>
</tr>
<tr>
<td>14</td>
<td>F</td>
<td>N</td>
<td>280</td>
<td></td>
<td>120 ...</td>
<td>Reentry using concealed AP</td>
<td>Sotalol, 40 mg twice daily</td>
<td>Palpitations decreased</td>
<td>8</td>
</tr>
<tr>
<td>15</td>
<td>M</td>
<td>N</td>
<td>290</td>
<td></td>
<td>190 ...</td>
<td>Reentry using concealed AP</td>
<td>Sotalol, 40 mg twice daily</td>
<td>Palpitations decreased</td>
<td>8</td>
</tr>
<tr>
<td>16</td>
<td>F</td>
<td>N</td>
<td>370</td>
<td></td>
<td>80 ...</td>
<td>Reentry using concealed AP</td>
<td>Sotalol, 40 mg twice daily</td>
<td>Palpitations decreased</td>
<td>8</td>
</tr>
<tr>
<td>17</td>
<td>F</td>
<td>N</td>
<td>320-370</td>
<td>No SVT</td>
<td>60 ...</td>
<td>Reentry in AVN</td>
<td>Sotalol, 160 mg twice daily</td>
<td>Palpitations + SVT</td>
<td>29</td>
</tr>
<tr>
<td>18</td>
<td>F</td>
<td>N</td>
<td>260-275</td>
<td>A/V SIM</td>
<td>...</td>
<td>Reentry in AVN</td>
<td>Propranolol, 20 mg twice daily</td>
<td>Asymptomatic</td>
<td>5</td>
</tr>
<tr>
<td>19</td>
<td>F</td>
<td>N</td>
<td>320</td>
<td></td>
<td>60 ...</td>
<td>Reentry in AVN</td>
<td>Sotalol, 80 mg twice daily</td>
<td>Asymptomatic</td>
<td>17</td>
</tr>
<tr>
<td>20</td>
<td>M</td>
<td>N</td>
<td>310</td>
<td></td>
<td>55 ...</td>
<td>Reentry in AVN</td>
<td>Digoxin, 0.25 mg daily</td>
<td>Palpitations less frequent</td>
<td>5</td>
</tr>
<tr>
<td>21</td>
<td>M</td>
<td>N</td>
<td>320-330</td>
<td></td>
<td>...</td>
<td>Reentry in AVN</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>22</td>
<td>F</td>
<td>N</td>
<td>270-320</td>
<td></td>
<td>...</td>
<td>Reentry in AVN</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
</tbody>
</table>

*WPW = Wolff-Parkinson-White syndrome; N = normal; SVT = supraventricular tachycardia; V-A = ventricular to atrial; AP = accessory pathway; A/V SIM = atrium and ventricle depolarized simultaneously; AVN = atrioventricular node; DNR = did not return.
grade limb of the circuit.\textsuperscript{18}

In patients with Wolff-Parkinson-White syndrome, electively induced atrial fibrillation and directly measuring the shortest and mean R-R interval provides the most accurate and relevant information in assessing the life-threatening potential of atrial fibrillation and is the method of choice for evaluating the problem.\textsuperscript{13-15} Several studies have shown that most episodes of ventricular fibrillation that occur in patients with Wolff-Parkinson-White syndrome have occurred in patients in whom the shortest R-R interval between pre-excitation beats during atrial fibrillation is less than 220 ms.\textsuperscript{13,14,18} In our study group, two patients had a very short anterograde refractory period of the accessory pathway of 140 and 170 ms. In the remaining patients in whom atrial fibrillation was successfully induced in the pretreatment state, the anterograde refractory period of the accessory pathway was >220 ms. Failure to initiate atrial fibrillation in the pretreatment state in patient 3 was due to repeated induction of supraventricular tachycardia at all cycle lengths used.

Esophageal stimulation and recording provides a relatively simple noninvasive procedure that can be utilized as a screening technique to identify patients with intranodal reentry and those with reentry utilizing an accessory pathway. In those patients with Wolff-Parkinson-White syndrome, it provides a method of sequential assessment of the response of the accessory pathway to drug therapy and, therefore, allows us to optimize treatment in these patients.

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