INTRODUCTION

Recommendations regarding the implantation of a permanent pacemaker in adults with atrioventricular (AV) block have been published by the American Heart Association and the American College of Cardiology.

The class I indication means that there is evidence or a general consensus that the procedure is beneficial, useful, or effective. Patients with grade 2 and 3 AV block with symptoms attributable to bradycardia and experiencing syncope, transient nausea, and confusion to cerebral hypotension belong to this category.1

These symptoms usually resolve with pacemaker implantation, but there are patients who experience symptoms after placement of a pacemaker. Our aim is to study the cause of syncope and presyncope after the pacemaker implantation.
pacemaker placement.

STUDY POPULATION

Between March 1999 and February 2000, 638 patients who had permanent pacemakers implanted by AV block were seen at the Ignacio Chávez National Institute of Cardiology pacemaker clinic for follow-up. Four women with syncope or presyncope between 26 and 71 years of age were studied. None of the patients had structural cardiac damage. The symptoms were observed between 1 month and 16 years after pacemaker implantation (Table 1).

METHODS

Complete clinical workup was obtained for all patients, including neurological evaluation, 24-hour Holter monitoring, stress test, and telemetry to analyze the stimulation system. A tilt table test was performed on a motorized table with footrest. During the test, an electrocardiogram was monitored continuously and arterial pressure was measured. The tilt was 70º for 30 minutes. When syncope did not occur, 5 mg of sublingual isosorbide dinitrate was administered and the same tilt was maintained for 15 minutes or until syncope occurred.2-4 The test was considered positive if presyncope or syncope with systemic hypertension (systolic <90 mm Hg or a reduction =30% of baseline) or bradycardia (frequency <50 beats/min or a reduction =20% from baseline) was present.

RESULTS

Neurological evaluation, 24-hour Holter and the stress test were normal or negative. Telemetry corroborated adequate function of the various components of the stimulation system.

In the 4 patients, the baseline tilt table test was negative and the isosorbide test was positive in 3 patients and negative in 1. Table 2 shows the baseline cardiac frequency, type of rhythm, and maximum cardiac frequency observed; cardiac frequency and arterial pressure at the moment at which the test was considered positive, as well as the type of pacemaker and the discharge frequency of each apparatus. Of note, patient 2 had an increase in sinus frequency from 76 to 134 beats/min before showing symptoms, which was observed with a sinus rhythm of 84 beats/min and an arterial pressure of 60/0 mm Hg; the pacemaker discharge program was 60 beats/min. In patient 3, symptoms were observed at 10 minutes, with an arterial pressure of 60/30 mm Hg and a decrease in the sinus frequency of the atrial rhythm from 82 beats/min to less than 50 beats/min, which required stimulation by the pacemaker programmed to discharge at that frequency. In patient 4, symptomatology appeared at 8 minutes, with a decrease in cardiac frequency from 85 to 75 beats/min and an arterial pressure of 80/50 mm Hg; the pacemaker, with a stimulation frequency programmed at 60 beats/min, did not discharge.

DISCUSSION

There are patients with AV block whose symptoms recur after electrical stimulation.5 The most frequent cause of persistent symptoms in patients with chronic AV block after implantation of a permanent pacemaker is dysfunction of the stimulation system.6 Other

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Table 1. Clinical characteristics

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Diagnosis</th>
<th>Year of symptoms</th>
<th>S1</th>
<th>PS1</th>
<th>S2</th>
<th>PS2</th>
<th>Year of ITT</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>71</td>
<td>CAVB without structural damage</td>
<td>S 1997</td>
<td>1 year</td>
<td>5</td>
<td>0</td>
<td>2</td>
<td>24</td>
</tr>
<tr>
<td>2</td>
<td>63</td>
<td>CAVB without structural damage</td>
<td>PS 1998</td>
<td>1 month</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>3</td>
<td>49</td>
<td>CAVB without structural damage</td>
<td>S y PS 1990</td>
<td>4 years</td>
<td>&gt;10</td>
<td>&gt;10</td>
<td>0</td>
<td>&gt;10</td>
</tr>
<tr>
<td>4</td>
<td>26</td>
<td>CAVB without structural damage</td>
<td>S 1984</td>
<td>16 years</td>
<td>4</td>
<td>0</td>
<td>1</td>
<td>10</td>
</tr>
</tbody>
</table>

AVB indicates Complete AV block; S, syncope; PS, presyncope; S1, number of syncopal episodes before pacemaker implant; PS1, number of presyncopal episodes prior to pacemaker implant; S2, number of syncopal episodes after pacemaker implant; PS2, number of presyncopal episodes after pacemaker implant; ITT, tilt table test.

Table 2. Tilt table test results

<table>
<thead>
<tr>
<th>Patient</th>
<th>Mode</th>
<th>Programmed CF</th>
<th>Baseline CF and type of rhythm</th>
<th>Moment of positivity</th>
<th>Minimum CF</th>
<th>Minimum CP</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>VVI</td>
<td>60</td>
<td>VVI 60/min</td>
<td>Negative test</td>
<td>Negative test</td>
<td>Negative test</td>
</tr>
<tr>
<td>2</td>
<td>DDDR</td>
<td>60</td>
<td>Sinus 76/min</td>
<td>Negative test</td>
<td>RS 81</td>
<td>60/0</td>
</tr>
<tr>
<td>3</td>
<td>DDD</td>
<td>50</td>
<td>VDD 60/min</td>
<td>10</td>
<td>DDD 50/min</td>
<td>60/30</td>
</tr>
<tr>
<td>4</td>
<td>DDD</td>
<td>60</td>
<td>VDD 60/min</td>
<td>8</td>
<td>VDD 75/min</td>
<td>80/40</td>
</tr>
</tbody>
</table>

*Moment syncope occurred

CF indicates cardiac frequency; PA, systemic arterial pressure.
possibilities are the so-called «pacemaker syndrome,» arrhythmias, or neurological problems. After discounting these causes of syncope, symptoms may be secondary to reflexive type disturbances or autonomic changes.9,10

Of the 4 patients studied, a diagnosis of postural orthostatic tachycardia was established in patient 2, as cardiac frequency increased by more than 30 beats/min with a 70º tilt. Syncope was not observed in this patient.11-13

The other 2 patients with a positive test had neurally mediated syncope (also called vasovagal or neurocardiogenic).

Per Sutton classification,14 patient 3 had mixed syncope, as the arterial hypotension was concomitant with bradycardia, causing the pacemaker to kick in at 50 beats/min. On the other hand, patient 4 had vasodepressor syncope, and cardiac frequency decreased to less than 10% of baseline at the time of hypotension. The finding of orthostatic intolerance in this patient 16 years after pacemaker implant indicates that it is unlikely that syncope observed prior to the implant was due to orthostatic intolerance, and supports the idea that this type of patient can suffer syncope caused by various mechanisms.

In the patient with a negative tilt table test, it was not possible to determine why the syncope persisted after placement of the pacemaker despite performance of tests which included an electrophysiological study.15

In this limited series, we found examples of different types of orthostatic intolerance. The appearance or persistence of syncope or presyncope in patients with complete AV block and an implanted pacemaker require taking a careful clinical history and complete physical examination including a neurological examination, review of the pacemaker with telemetry, a Holter study, and a tilt table test. Establishing a correct diagnosis allows selection of adequate treatment for the patient.

REFERENCES