Identification of Concealed Posteroseptal Kent Pathways by Comparison of Ventriculoatrial Intervals From Apical and Posterobasal Right Ventricular Sites

Jesús D. Martínez-Alday, MD; Jesús Almendral, MD; Angel Arenal, MD; José Miguel Ormaetxe, MD; Agustín Pastor, MD; Julián P. Villacastín, MD; Olga Medina, MD; Rafael Peinado, MD; Juan L. Delcán, MD

Background The differential diagnosis of supraventricular tachycardia with concentric atrial activation usually requires the inducibility of sustained tachycardia and needs a complex and time-consuming electrophysiological evaluation. To develop a simple test to establish if ventriculatrial conduction uses a posteroseptal accessory pathway or the normal conduction system, we compared the ventriculatrial intervals during right ventricular pacing from apical and posterobasal sites.

Methods and Results Continuous pacing was performed from an apical and a posterobasal right ventricular site in 34 patients with retrograde conduction over the normal conduction system (group A) and in 22 patients with conduction over a posteroseptal accessory pathway (group B). During apical pacing, ventriculatrial intervals in group A (176±40 milliseconds) were not significantly different than those in group B (197±47 milliseconds, P=NS). During posterobasal pacing, group B patients had significantly shorter ventriculatrial intervals than group A patients (158±46 versus 197±39 milliseconds, P<.01). The difference between the ventriculatrial interval obtained during apical pacing and that obtained during posterobasal pacing (ventriculatrial index) discriminated between the two groups without overlapping: It was positive in all group B patients (39±19; range, +10 to +70 milliseconds) and negative in all except two group A patients (−21±13; range, −50 to +5 milliseconds; P<.001).

Conclusions This ventriculatrial index can identify accurately and in the absence of tachycardia whether concentric retrograde conduction is proceeding over a posteroseptal accessory pathway or over the normal conduction system. (Circulation. 1994;89:1060-1067.)

Key Words • tachycardia • conduction • intervals

The diagnosis of a concealed atrioventricular accessory pathway frequently requires some electrophysiological workup, since the electrocardiographic signs of preexcitation during sinus rhythm are lacking and the ability of the standard surface ECG to recognize its presence during tachycardia is limited.1 If retrograde atrial activation during supraventricular tachycardia (SVT) is concentric, the diagnostic possibilities include atrioventricular nodal reentry (AVNR) and atrial tachycardia as well as atrioventricular orthodromic reciprocating tachycardias (AVRT) mediated by the septal accessory pathway. The latter is established if ventricular extrastimuli delivered during His bundle refractoriness advance the subsequent atrial depolarization.2 Recently, several other phenomena have been described (usually as indexes) that can aid in distinguishing AVRT from AVNR.3,4 However, the recognition of these phenomena requires the inducibility of tachycardia, needs a complex and time-consuming electrophysiological evaluation, and may be difficult in the presence of multiple tachycardia mechanisms.

The purpose of the present study was to develop a simple test to establish if ventriculatrial conduction uses a posteroseptal accessory pathway. We tested the following hypothesis: If ventriculatrial conduction proceeds over a posteroseptal accessory pathway, the ventriculatrial interval (VAI) during stimulation from right ventricular posterobasal sites would be shorter than during stimulation from right ventricular apical sites because of a closer proximity of the former sites to the ventricular insertion of the accessory pathway; conversely, if ventriculatrial conduction proceeds over the normal conduction system, the VAI during stimulation from right ventricular apical sites may be shorter than from right ventricular posterobasal sites because of an earlier invasion of the His-Purkinje system at the former sites.

Methods

Study Patients

We performed prospectively 56 studies in 47 patients in whom a complete electrophysiological workup disclosed the mechanism of 1:1 ventriculatrial conduction during ventricular stimulation, that being the normal conduction system (group A), or a posteroseptal accessory pathway (group B). Group A (control group) included 34 patients, 20 men and 14 women aged 15 to 65 years (mean, 37±15), and group B included 22 patients, 9 men and 13 women aged 15 to 64 years (mean, 34±16). Twenty patients had a right posteroseptal pathway and two patients had a left posteroseptal pathway, both defined by catheter positioning at the successful ablation site. Nine patients served as their own controls because they

Received July 22, 1993; revision accepted November 16, 1993.

From the Clinical Electrophysiology Laboratory, Servicio de Cardiología, Hospital General Gregorio Marañón, Madrid, Spain.

Reprint requests to Jesús Almendral, MD, Cardiología (planta 5), Hospital General Gregorio Marañón, Doctor Esquerdo, 46, 28007-Madrid, Spain.
were studied before and after ablation of an accessory pathway; therefore, they were included in both groups. The reasons for the electrophysiological study in group A patients included sustained monomorphic ventricular tachycardia (7 cases), atrioventricular nodal reentrant tachycardia (5 cases), and radiofrequency ablation of an accessory pathway (22 cases).

**Electrophysiological Study**

All patients gave written informed consent for the electrophysiological study, which was performed in the nonsedated or slightly sedated (morphine or midazolam) postsensory state. Antiarrhythmic drugs were discontinued at least for five half-lives in all patients. No patient had received amiodarone in the previous 3 months. For the purpose of the diagnostic study, and depending on the characteristics of the documented arrhythmias, we positioned under fluoroscopic guidance two to six quadripolar, hexapolar, or decapolar electrode catheters with an interelectrode distance of 0.2 to 1 cm. Stimulation was performed using a programmable stimulator (Biotronik UHS 20). Bipolar cathodal stimulation was performed at the distal pair of each multipolar catheter. Stimuli were rectangular pulses 1 millisecond in duration at twice diastolic threshold. Data were recorded with a photographic recorder (Honeywell VR 12) at a 100- to 150-mm/s paper speed and stored on magnetic tape using a cassette tape recorder (Honeywell 4728) for later analysis. Three to six surface ECG leads (at least one, aVF and V1) were recorded along with at least two intracardiac bipolar electrograms (atrial and ventricular electrograms). All intracardiac electrograms were filtered at 30 to 500 Hz.

**Electrophysiological Diagnosis**

In group A patients, the presence of an atrioventricular accessory pathway was excluded according to the following criteria: (1) in the 7 patients with sustained ventricular tachycardia: (a) no antegrade preexcitation during sinus rhythm or atrial pacing, (b) no previous history or inducible narrow QRS complex tachycardia, and (c) ventriculoatrial conduction with decremental properties as shown by incremental ventricular pacing and extrastimuli; (2) in the 5 patients with AVNR: (a) VAI during sustained SVT shorter than 60 milliseconds when measured in the His bundle electrogram (mean, 25 milliseconds); (b) atrial activation sequence beginning at the interatrial septum both during SVT and ventricular stimulation, (c) inability of ventricular extrastimuli delivered during SVT to advance the subsequent atrial depolarization when the His bundle was refractory, and (d) absence of transient entrainment of SVT with ventricular fusion during ventricular stimulation (all cases); and (3) in 22 cases after accessory pathway radiofrequency ablation: (a) atrial activation sequence beginning at the interatrial septum during ventricular stimulation, (b) ventriculoatrial conduction with decremental properties as shown by incremental ventricular pacing and extrastimuli, and (c) transient block in ventriculoatrial conduction after ATP infusion (8 cases).

In group B patients, the diagnosis of a posteroseptal accessory pathway with retrograde conduction was established according to standard criteria. In particular, in the cases of these series the following observations were made: (1) an atrial activation sequence occurring during SVT identical to that during ventricular pacing, with earliest atrial activation at the coronary sinus os or low septal right atrium (all cases), (2) ability to advance the subsequent atrial activation with a premature ventricular extrastimulus delivered during SVT at a time when the His bundle was refractory (18 cases), (3) a left bundle branch block–induced increase in the VAI during SVT of no more than 15 milliseconds (5 cases), and (4) presence of transient entrainment of SVT with ventricular fusion during ventricular stimulation (10 of 11 cases).

**Identification of the Retrogradely Conducting Pathway in Group B Cases**

 Patients with a posteroseptal accessory pathway have two potential pathways for retrograde conduction with an earliest septal atrial activation. Therefore, special care was taken to ensure that retrograde atrial depolarization occurred exclusively as a result of ventriculoatrial conduction over the accessory pathway and that potential conduction over the normal conduction system did not contribute to retrograde atrial depolarization.

In 5 group B patients, the study pacing protocol (see below) was performed during orthodromic reciprocating tachycardia producing transient entrainment of tachycardia with ventricular fusion. By so doing, it is certain that retrograde conduction proceeds exclusively over the accessory pathway since the normal conduction system is being used for antegrade conduction.

In the remaining patients, one of the following criteria was met: (1) absence of ventriculoatrial conduction after radiofrequency ablation of the posteroseptal accessory pathway, making it unlikely that retrograde conduction was proceeding over the normal conduction system in the preablation state (6 cases) or (2) identical atrial activation sequence during SVT and ventricular stimulation, with the earliest atrial electrogram recorded at the coronary sinus os, and being the atrial electrogram at the His bundle site relatively late (all cases); had a retrogradely conducting normal conduction system contributed to retrograde atrial depolarization during ventricular stimulation, a change in the atrial activation sequence would have been expected during SVT when retrograde conduction occurs exclusively over the accessory pathway; in 3 of these patients, additional evidence for the lack of contribution of the normal conduction system to retrograde atrial depolarization was obtained by the absence of change in atrial activation sequence or VAI after ATP infusion (20 mg IV bolus).

**Study Protocol: Catheter Positioning, Pacing, Measurements, and Ventriculoatrial Index**

Ventricular pacing was performed on each patient from two right ventricular positions, an apical site, and a posteroobasal site. Fig 1 illustrates the position of the catheters for the study. The apical catheter position was always confirmed on the right
TABLE 1. Clinical and Electrophysiological Characteristics of Group A

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age, y</th>
<th>Sex</th>
<th>ED</th>
<th>PCL, ms</th>
<th>VAI Apex, ms</th>
<th>VAI Base, ms</th>
<th>A-B</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>65</td>
<td>M</td>
<td>VT</td>
<td>600</td>
<td>145</td>
<td>180</td>
<td>−15</td>
</tr>
<tr>
<td>2</td>
<td>33</td>
<td>F</td>
<td>AVNRT</td>
<td>450</td>
<td>165</td>
<td>160</td>
<td>+5</td>
</tr>
<tr>
<td>3</td>
<td>39</td>
<td>F</td>
<td>AVNRT</td>
<td>600</td>
<td>175</td>
<td>220</td>
<td>−45</td>
</tr>
<tr>
<td>4</td>
<td>43</td>
<td>M</td>
<td>VT</td>
<td>600</td>
<td>240</td>
<td>260</td>
<td>−20</td>
</tr>
<tr>
<td>5</td>
<td>43</td>
<td>F</td>
<td>PA</td>
<td>600</td>
<td>235</td>
<td>255</td>
<td>−20</td>
</tr>
<tr>
<td>6</td>
<td>17</td>
<td>M</td>
<td>PA</td>
<td>400</td>
<td>100</td>
<td>145</td>
<td>−45</td>
</tr>
<tr>
<td>7</td>
<td>45</td>
<td>M</td>
<td>PA</td>
<td>600</td>
<td>180</td>
<td>190</td>
<td>−10</td>
</tr>
<tr>
<td>8</td>
<td>36</td>
<td>F</td>
<td>PA</td>
<td>500</td>
<td>170</td>
<td>180</td>
<td>−10</td>
</tr>
<tr>
<td>9</td>
<td>50</td>
<td>M</td>
<td>AVNRT</td>
<td>500</td>
<td>225</td>
<td>250</td>
<td>−25</td>
</tr>
<tr>
<td>10</td>
<td>65</td>
<td>M</td>
<td>VT</td>
<td>550</td>
<td>220</td>
<td>230</td>
<td>−10</td>
</tr>
<tr>
<td>11</td>
<td>51</td>
<td>F</td>
<td>VT</td>
<td>600</td>
<td>120</td>
<td>130</td>
<td>−10</td>
</tr>
<tr>
<td>12</td>
<td>34</td>
<td>M</td>
<td>PA</td>
<td>600</td>
<td>130</td>
<td>150</td>
<td>−20</td>
</tr>
<tr>
<td>13</td>
<td>46</td>
<td>M</td>
<td>VT</td>
<td>500</td>
<td>130</td>
<td>140</td>
<td>−10</td>
</tr>
<tr>
<td>14</td>
<td>47</td>
<td>M</td>
<td>PA</td>
<td>500</td>
<td>200</td>
<td>210</td>
<td>−10</td>
</tr>
<tr>
<td>15</td>
<td>33</td>
<td>M</td>
<td>PA</td>
<td>600</td>
<td>160</td>
<td>200</td>
<td>−40</td>
</tr>
<tr>
<td>16</td>
<td>40</td>
<td>F</td>
<td>VT</td>
<td>700</td>
<td>190</td>
<td>215</td>
<td>−25</td>
</tr>
<tr>
<td>17</td>
<td>29</td>
<td>F</td>
<td>AVNRT</td>
<td>550</td>
<td>170</td>
<td>195</td>
<td>−25</td>
</tr>
<tr>
<td>18</td>
<td>17</td>
<td>M</td>
<td>PA</td>
<td>600</td>
<td>165</td>
<td>180</td>
<td>−15</td>
</tr>
<tr>
<td>19</td>
<td>46</td>
<td>F</td>
<td>PA</td>
<td>500</td>
<td>170</td>
<td>185</td>
<td>−15</td>
</tr>
<tr>
<td>20</td>
<td>57</td>
<td>M</td>
<td>VT</td>
<td>400</td>
<td>180</td>
<td>200</td>
<td>−20</td>
</tr>
<tr>
<td>21</td>
<td>26</td>
<td>M</td>
<td>PA</td>
<td>500</td>
<td>155</td>
<td>180</td>
<td>−25</td>
</tr>
<tr>
<td>22</td>
<td>61</td>
<td>M</td>
<td>PA</td>
<td>500</td>
<td>235</td>
<td>235</td>
<td>0</td>
</tr>
<tr>
<td>23</td>
<td>57</td>
<td>F</td>
<td>PA</td>
<td>500</td>
<td>135</td>
<td>155</td>
<td>−20</td>
</tr>
<tr>
<td>24</td>
<td>32</td>
<td>M</td>
<td>PA</td>
<td>500</td>
<td>160</td>
<td>190</td>
<td>−30</td>
</tr>
<tr>
<td>25</td>
<td>40</td>
<td>F</td>
<td>PA</td>
<td>500</td>
<td>150</td>
<td>200</td>
<td>−50</td>
</tr>
<tr>
<td>26</td>
<td>17</td>
<td>M</td>
<td>PA</td>
<td>400</td>
<td>180</td>
<td>200</td>
<td>−20</td>
</tr>
<tr>
<td>27</td>
<td>27</td>
<td>F</td>
<td>PA</td>
<td>500</td>
<td>185</td>
<td>205</td>
<td>−20</td>
</tr>
<tr>
<td>28</td>
<td>17</td>
<td>F</td>
<td>PA</td>
<td>500</td>
<td>250</td>
<td>270</td>
<td>−20</td>
</tr>
<tr>
<td>29</td>
<td>25</td>
<td>F</td>
<td>PA</td>
<td>550</td>
<td>140</td>
<td>155</td>
<td>−15</td>
</tr>
<tr>
<td>30</td>
<td>27</td>
<td>M</td>
<td>PA</td>
<td>600</td>
<td>230</td>
<td>245</td>
<td>−15</td>
</tr>
<tr>
<td>31</td>
<td>15</td>
<td>M</td>
<td>PA</td>
<td>600</td>
<td>130</td>
<td>140</td>
<td>−10</td>
</tr>
<tr>
<td>32</td>
<td>25</td>
<td>M</td>
<td>PA</td>
<td>400</td>
<td>125</td>
<td>180</td>
<td>−55</td>
</tr>
<tr>
<td>33</td>
<td>16</td>
<td>F</td>
<td>PA</td>
<td>500</td>
<td>240</td>
<td>250</td>
<td>−10</td>
</tr>
<tr>
<td>34</td>
<td>45</td>
<td>M</td>
<td>AVNRT</td>
<td>500</td>
<td>200</td>
<td>240</td>
<td>−40</td>
</tr>
</tbody>
</table>

Mean±SD 37±15 20M/14F 524±80 176±40 197±39 −21±13

ED indicates electrophysiological diagnosis; PCL, pacing cycle length; VAI, ventriculoatrial interval; A-B, difference between VAI from apex and base; VT, ventricular tachycardia; AVNRT, atrioventricular nodal reentrant tachycardia; and PA, postablation of an accessory pathway.

Anterior oblique view. When approaching posterobasal sites, care was taken to position the catheter as basal (close to the tricuspid ring) as was possible. The pacing catheter was usually a Polaris 7F with 2–5-mm interelectrode distance (Mansfield/Webster, Watertown, Mass), since its deflectable tip allowed for a more precise positioning, particularly at the posterobasal sites.

Stimulation and recording characteristics were similar to those during the diagnostic study, as described above. Continuous ventricular pacing was performed at each site, with an identical cycle length for each patient. The pacing cycle length necessarily varied from case to case: shorter than the tachycardia cycle length in cases where stimulation was performed during tachycardia and longer than the pacing cycle length at which the ventriculoatrial Wenckebach phenomenon occurred in group A cases. The mean pacing cycle length was 524±80 milliseconds in group A and 447±106 milliseconds in group B (P<.01).

The VAI was measured from the stimulus artifact to a stable atrial reference located at the high right atrium. Care was taken to exclude simultaneous stimulation of atrial and ventricular chambers during stimulation from posterobasal sites, since this would have resulted in a spuriously short VAI.
The ventriculoatrial index was defined as the difference in the VAI during pacing at the apical site and during pacing at the posterobasal site at an identical rate.

**Data Analysis**

Continuous data are presented as mean±SD. Differences between groups in proportional data were tested using the χ² test. A two-tailed value (for paired and unpaired data) of \( P<.05 \) was considered statistically significant. Sensitivity, specificity, and positive predictive accuracy were calculated in the usual manner.

**Results**

Clinical and electrophysiological data of individual patients are presented in Tables 1 and 2. There were no statistically significant differences between groups regarding age and sex.

During apical pacing, VAIs in group A patients (176±40 milliseconds) were not significantly different from those in group B patients (197±47 milliseconds, \( P=NS \)). In contrast, during pacing from posterobasal sites, group B patients had significantly shorter VAIs than group A patients (158±46 versus 197±39 milliseconds, \( P<.01 \)). This difference appeared despite longer paced cycle lengths in group A that would have favored shorter intervals in this group. However, as shown in Fig 2, significant overlapping in individual cases precluded the use of this value to discriminate between groups.

When values for VAIs obtained from the two pacing sites were compared on each patient (ventriculoatrial index), a different behavior in the two groups was obvious (Tables 1, 2, and 3 and Fig 3). In group B, the VAI obtained during apical pacing was longer than during posterobasal pacing in all patients (positive ventriculoatrial index). An example of this behavior is shown in Fig 4. Conversely, in group A, as illustrated in Fig 5, the VAI during apical pacing was shorter than during posterobasal pacing in all except 2 patients (negative ventriculoatrial index); in 1 of these 2 cases (patient 22, Table 1), the ventriculoatrial index was 0, and in the remaining case (patient 2, Table 1), it was +5 milliseconds. Since the ventriculoatrial index in group B always exceeded 10 milliseconds, a value for this index ≥10 milliseconds establishes the presence of a posteroseptal accessory pathway in the present series (Fig 3) with 100% sensitivity, specificity, and positive predictive value.

**Discussion**

The major finding of this study is that the ventriculoatrial index can identify accurately and in the absence
of tachycardia whether concentric retrograde conduction is proceeding over a posteroseptal accessory pathway or over the normal conduction system.

**Evaluation of the Working Hypothesis**

**Retrograde Conduction Over a Posteroseptal Accessory Pathway**

As anticipated, the VAIs in these cases (group B) were dependent on the proximity of the stimulating site to the ventricular insertion of the accessory pathway and consequently were shorter when stimulating from a posteroseptal site as opposed to an apical site. However, it is of note that this observation holds true for all cases despite catheter positioning according to pure radiological landmarks without a specific mapping effort to position the catheter exactly at the ventricular insertion of the accessory pathway. Moreover, with such simple positioning maneuvers, the ventriculoatrial index was ≥10 milliseconds in all group B cases. On the other hand, the wide range of values of the ventriculoatrial index (from 10 to 70 milliseconds; mean, 39±19) was surprising; minor differences in catheter positioning and accessory path-

**TABLE 3. Major Statistical Findings**

<table>
<thead>
<tr>
<th></th>
<th>VAI Apex</th>
<th>VAI Base</th>
<th>A-B</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group A</td>
<td>176±40 ms</td>
<td>197±39 ms</td>
<td>−21±13</td>
</tr>
<tr>
<td>Group B</td>
<td>197±47 ms</td>
<td>158±46 ms</td>
<td>+39±19</td>
</tr>
<tr>
<td>Probability</td>
<td>NS</td>
<td><em>P&lt;.01</em></td>
<td><strong>P&lt;.001</strong></td>
</tr>
</tbody>
</table>

VAI indicates ventriculoatrial interval; A-B, difference in VAI from apex and base.

**Fig 2.** Density dot chart display of the ventriculoatrial interval during basal stimulation. There was a significant difference in ventriculoatrial interval between groups (shorter in group B, patients with a posteroseptal Kent pathway). However, note that the degree of overlapping between groups is high.

**Fig 3.** Density dot chart display of the ventriculoatrial indexes in both groups. This index was significantly greater in group B (patients with a posteroseptal Kent pathway) than in group A (control patients) (*P<.001*). Note that this index separates individual cases of both groups without overlapping: No group A cases exceeded +5 milliseconds, whereas all group B cases had an index ≥+10 milliseconds.

way location could explain in part this finding; the possible role of individual differences in intraventricular conduction times remains speculative.

**Retrograde Conduction Over the Normal Conduction System**

We hypothesized that the VAI during retrograde conduction over the normal conduction system would be shorter with stimulation from an apical site than from a posterobasal site. This hypothesis was based on the classic study by Durrer and coworkers on the ventricular endocardial activation sequence. These investigators demonstrated that the endocardial activation of the right ventricular posterobasal area was late (60 to 70 milliseconds later than the midseptum), whereas the activation of the apical area occurred relatively early (within the first 20 milliseconds of ventricular activation). Data from animal studies and intraoperative epicardial mapping of the human heart in a small number of patients are also consistent with these observations. It is unclear whether differences in distribution of Purkinje fibers or in fiber orientation in the septum account for this activation pattern. We were not aware of similar information about retrograde conduction. However, the hypothesis was formulated with the expectation that the pattern of retrograde conduction would follow, at least in a relative fashion, that of antegrade conduction. In fact, in patients with ventriculoatrial conduction proceeding normally (group A), the mean VAI with apical stimulation was significantly shorter than with posterobasal stimulation (Table 3). However, in most cases we observed only minor differences, between −30 and −10 milliseconds in 24 of 32 patients (75%), in VAIs, and in 2 patients (patients 2 and 22) the VAIs were identical or even 5 milliseconds shorter with posterobasal stimulation. These results suggest that in the majority of patients the retrograde
conduction times (comparing apical with posterobasal sites) follow qualitatively the antegrade conduction times. However, there are exceptions in whom the differences are minor or even reversed. Individual differences in the distribution of the Purkinje network could account for these individual differences in the conduction patterns.

Capacity of VAIs and Ventriculoatrial Index to Identify the Route of Retrograde Conduction

The VAIs measured during stimulation from the right ventricular apex tended to be shorter in group A (conduction over the normal conduction system) than in group B (conduction over a posteroseptal accessory pathway, Table 3). Conversely, when VAIs were measured during posterobasal stimulation, the mean value was significantly shorter in group B than in group A (Table 3). However, as shown in Fig 2, there was considerable overlapping between groups in individual values of the VAIs. In contrast, as depicted in Fig 3, the individual values of the ventriculooatrial index showed no overlap between groups. A ventriculoatrial index ≥10 milliseconds detected correctly in all cases the presence of a posteroseptal accessory pathway.

Comparison With Other Means of Electrophysiological Differential Diagnoses

VAIs during tachycardia can help to distinguish AVNR from AVRT, but some values are nondiagnostic. The ability of ventricular extrastimuli, delivered during tachycardia at a time when the His bundle is refractory, to advance the subsequent atrial depolarization usually identifies the presence of a posteroseptal accessory pathway. The absence of this phenomenon is generally taken as evidence for AVNR tachycardia. However, this technique cannot be used if the tachycardia is unsustained, markedly irregular, or hemodynamically unstable or may have limited value if a short tachycardia cycle length or a long ventricular refractory period precludes the introduction of extrastimuli with short coupling intervals.

In an attempt to avoid these limitations, several other methods have been proposed. Miles et al compared the longest coupling interval of ventricular extrastimuli that advanced atrial activation during SVT with the SVT cycle length. The difference between both values (called preexcitation index) was found to be small in AVRT with septal accessory pathway; in contrast, in AVNR, the values for the preexcitation index were greater or “unmeasurable” (the atria could not be preexcited) and therefore able to specifically distinguish these tachycardia mechanisms. More recently, a similar index was derived for entrainment (“entrainment index”) but was only studied for AVRT. However, these indexes also require a sustained and regular tachycardia.

To avoid this limitation, Crozier et al and Yamashita et al have developed alternative methods. Crozier et al compared the VAIs during SVT and during right ventricular apical stimulation. The ratio between these two parameters was greater for the 5 patients with posteroseptal accessory pathway than for the 16 patients with
AVRN. Yamashita et al proposed an analytical approach to calculate an index that is essentially identical to the preexcitation index by adding and subtracting certain intervals measured during SVT and during ventricular stimulation. These two methods can be used even if the SVT is unsustained but may be difficult to obtain if tachycardias are irregular and/or VAI is variable. More recently, Miller et al compared the His to atrial interval during right ventricular pacing and during SVT. The latter was always greater than the former in AVRT, whereas the opposite was usually the case in AVNR; a cutoff value of −10 milliseconds had 100% sensitivity, specificity, and positive predictive accuracy. The potential limitation of this elegant observation is the ability to obtain retrograde His deflections during ventricular pacing in some cases. In contrast to the other methods, the ventriculoatrial index described here could be used in the complete absence of tachycardia.

**Study Limitations**

The ventriculoatrial index identifies the actual route of ventriculoatrial conduction and therefore the “fastest” path for this conduction. For example, a slowly conducting accessory pathway would be missed in the presence of fast retrograde conduction over the normal conduction system. Conflicting results could be obtained if conduction takes place simultaneously over an accessory pathway and the normal conducting system or alternatively, over these two routes, depending on the pacing site. Certain maneuvers could help in these difficult settings and may eventually help to disclose the presence of more than one “path” for conduction; these maneuvers include calculation of the index at several pacing cycle lengths (the ventriculoatrial index should be independent of pacing rate and, consequently, different values of the index at different rates would suggest more than one conducting path), after verapamil infusion, and during tachycardia (producing transient entrainment, as in some of our cases).

The possibility that right bundle branch block could alter the significance of the ventriculoatrial index cannot be excluded, particularly when ventriculoatrial conduction proceeds over the normal conducting system. We did not suspect this defect in any of our cases because none of them had antegrade right bundle branch block. However, the pattern of right ventricular activation is unchanged during left bundle branch block and delayed (but without significant changes in the activation sequence) during right bundle branch block.

To the extent that this was the case for retrograde conduction defects, they would not alter the significance of the ventriculoatrial index.

In cases of SVT with concentric atrial activation, a concealed anteroseptal accessory pathway is another possibility. This type of accessory pathway is extremely rare (less than 1%), and we did not have the opportunity to study any of those during the time of the study. Presumably, the ventriculoatrial index for this type of accessory pathway should be obtained with stimulation from the septal aspect of the right ventricular infundibulum.

**Clinical Implications**

This study reports a new and simple test to assess whether retrograde conduction takes place over a pos-teroseptal accessory pathway or over the normal conduction system. This test compares favorably with other methods previously described for this differential diagnosis when inducible SVT is unsustained or markedly
irregular, as discussed above. Therefore, its main clinical value will be in those uncommon but difficult eventualities. It can also be useful during catheter ablation of overt posteroseptal accessory pathways. It has been described that ablation can block selectively antegrade conduction of an overt accessory pathway. Thus, if retrograde conduction persists after ablation of antegrade accessory pathway conduction and SVT is not inducible, the ventriculoatrial index can help in assessing the persistence of conduction over the accessory pathway. This index also could be of clinical help in the differential diagnosis of “long RP” tachycardias, particularly if the decremental properties of the retrograde limb make it difficult to assess atrial preexcitation.

Acknowledgments
This study was supported by a grant of the Miguel Irriarte Institute of Epidemiology from the Basque Country University, Bilbao, Spain.

References
Identification of concealed posteroseptal Kent pathways by comparison of ventriculoatrial intervals from apical and posterobasal right ventricular sites.
J D Martínez-Alday, J Almendral, A Arenal, J M Ormaetxe, A Pastor, J P Villacastín, O Medina, R Peinado and J L Delcán

Circulation. 1994;89:1060-1067
doi: 10.1161/01.CIR.89.3.1060

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1994 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/89/3/1060

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org/subscriptions/