Demonstration of the presence of slow conduction during sustained ventricular tachycardia in man: use of transient entrainment of the tachycardia

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ABSTRACT To test the hypothesis that an area of slow conduction is present during reentrant ventricular tachycardia in man, and that the earliest activation site during ventricular tachycardia is within or orthodromically just distal to the area of slow conduction in the reentry loop, we studied 12 episodes of ventricular tachycardia (mean rate 185 ± 32 beats/min) that were induced in nine patients with ischemic heart disease. Rapid ventricular pacing was performed at selected sites during ventricular tachycardia while recording electrograms from an early activation site relative to the onset of the QRS complex (site A) and from a site close to the pacing site (site B). Rapid pacing from the right ventricular apex during ventricular tachycardia with a right bundle branch block pattern and from selected left ventricular sites during ventricular tachycardia with a left bundle branch block pattern (mean pacing rate 202 ± 38 beats/min) resulted in constant ventricular fusion beats on the electrocardiogram except for the last captured beat (i.e., the ventricular tachycardia was entrained) in 11 of 12 episodes. During entrainment: (1) sites A and B were activated at the pacing rate, (2) conduction time from the last pacing impulse to the last captured ventricular electrogram at site A (St-A interval) was 359 ± 69 msec and spanned the diastolic interval, while that at site B (St-B interval) was only 28 ± 13 msec, (3) site A had the same ventricular electrogram morphology as that during ventricular tachycardia, while site B had a different electrogram morphology, indicating that site A was activated in the same direction during entrainment as during ventricular tachycardia. Eight episodes of ventricular tachycardia were entrained at two or more different pacing rates. The St-A interval increased during pacing at the faster rate(s) in four of eight episodes, while the St-B interval remained unchanged. Rapid ventricular pacing performed from the same site during sinus rhythm (mean pacing rate 201 ± 37 beats/min) resulted in an St-A interval of 103 ± 37 msec (p < .001 vs the value during entrainment) and an St-B interval of 31 ± 15 msec (p = NS vs the value during entrainment). It is concluded that an area of slow conduction not demonstrable during sinus rhythm exists during ventricular tachycardia, and that the earliest activation site during ventricular tachycardia is at or orthodromically distal to this area of slow conduction.


REENTRY has been widely accepted as the major mechanism of ventricular tachycardia, especially that associated with ischemic heart disease. It is generally accepted that reentry usually requires a circuit with unidirectional block for its initiation and an area of slow conduction for both its initiation and its maintenance. Recent experimental studies of canine ventricular tachycardia after myocardial infarction not only demonstrated reentry as its mechanism, but also demonstrated slow conduction and unidirectional conduction block (functional block) in the reentry circuit. In human ventricular tachycardia, the presence of an area of slow conduction has generally been assumed, but it has not been directly demonstrated.

When a reentrant tachyarrhythmia is transiently entrained during rapid pacing, all the tissue related to the tachycardia, including the area of conduction in the reentry circuit, will be activated by the pacing impulse...
at the pacing rate. Thus, during transient entrainment it is possible to determine conduction time between the pacing site and any selected site or in relation to the reentry loop simply by measuring the time interval between the pacing stimulus and the electrogram recorded at the selected site(s) that results from that pacing impulse.

In the present study, rapid ventricular pacing was performed from selected sites during sustained ventricular tachycardia induced in patients with ischemic heart disease. By analyzing data obtained during transient entrainment of the tachycardia, we tested the hypothesis that an area of slow conduction is present during the tachycardia, and furthermore, that a site activated early relative to the onset of each QRS complex during the tachycardia is within or orthodromically just distal to the area of slow conduction in the reentry loop.

Methods

Nine patients with clinically sustained ventricular tachycardia were studied in the cardiac catheterization laboratory after informed consent was obtained. All patients had a left ventricular aneurysm or akinetic region due to a previous myocardial infarction (table 1). All antiarrhythmic drugs were discontinued at least 24 hr before the study. Two patients had previously undergone coronary artery bypass graft surgery and left ventricular aneurysmectomy. In one of these two patients, an electrophysiologic study was performed both before and after the surgery.

Electrophysiologic study was performed by standard techniques, USCI Josephson quadripolar electrode catheters were placed in the right ventricular apex and in the right ventricular outflow tract of each patient. Also, a Josephson or USCI octapolar electrode catheter was introduced in the left ventricular cavity to allow left ventricular endocardial mapping during ventricular tachycardia. The interelectrode distances for the Josephson-electrode catheters were all 5 mm. The octapolar catheter was of special design, consisting of four electrode pairs (pair Nos. 1, 2, 3, and 4, distal to proximal, respectively) in which the electrodes of each pair were separated by 2 mm. Pairs 1 and 2 were separated by 1 cm, pairs 3 and 4 by 1 cm, and pairs 2 and 3 by 5 cm, permitting electrograms to be recorded from four sites while the catheter sat in the left ventricle in a U position. The bend in the U was between pair 2 and pair 3. Bipolar electrograms filtered between a bandpass of 10 to 500 Hz were recorded from all the electrode catheters on photographic paper simultaneously with surface electrocardiographic leads I, II, III, and V1 with use of an Electronics for Medicine VR-16 oscilloscopic recorder. All data were also recorded on a Honeywell 5600 FM tape recorder for subsequent playback and analysis. A Medtronic 1349A battery-powered programmable pacemaker was used for the ventricular pacing. All the pacing was done at a stimulus strength of twice the diastolic threshold.

Study protocol. After ventricular tachycardia was induced by standard pacing techniques, left ventricular endocardial catheter mapping was performed during the tachycardia to identify the earliest activation site relative to the onset of the QRS complex. The position of the catheter in the left ventricle was determined by the use of biplane fluoroscopy. An average of 26 sites was mapped for each tachycardia (range eight to 40 sites). While bipolar electrograms were recorded from the electrodes placed at this earliest activation site or at an early activation site close to the earliest site, transient entrainment of ventricular tachycardia by rapid ventricular pacing was attempted as follows: Rapid pacing was initiated during ventricular tachycardia from the right ventricular apex and/or from a selected site in the left ventricle at a rate 5 to 20 beats/min faster than the spontaneous rate of the tachycardia. The ventricular pacing site was usually in the ipsilateral chamber of the bundle branch block pattern of the tachycardia, since rapid pacing from such a site was expected to demonstrate transient entrainment. When the earliest activation site was located in the ventricular septum of the left ventricle and the ventricular tachycardia had a left bundle branch block pattern, rapid pacing was performed from the posterobasal portion of the left ventricle with the use of the most distal pair of electrodes (pair No. 1) of the octapolar electrode catheter, while bipolar electrograms were recorded from the earliest or a relatively early activation site with one of the proximal two pairs (Nos. 3 or 4) of electrodes. Rapid pacing was continued for up to 10 sec and then terminated abruptly. If the tachycardia still continued, rapid ventricular pacing was again performed from the same site, but with an increment in the pacing rate of about 10 beats/min (range 9 to 17 beats/min). This pacing procedure was repeated until either the ventricular tachycardia was interrupted or pacing had to be discontinued because of associated hemodynamic instability or concern that ventricular pacing at too rapid a rate would precipitate ventricular fibrillation.

During subsequent sinus rhythm, rapid ventricular pacing from the same pacing site at about the same rate as used during the entrainment study was repeated while electrograms were recorded from the same sites as before. During such pacing, the relative activation sequence of the ventricular electrograms and the conduction times from the pacing stimulus to each recording site were compared with those obtained during the prior entrainment study.

All measurement data are the mean ± SD. Statistical analysis was performed with the Student t test.

### TABLE 1
Clinical characteristics of the patients

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Period after initial MI</th>
<th>Site of LV aneurysm or akinetic region</th>
<th>Other relevant data</th>
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<tr>
<td>1</td>
<td>55</td>
<td>F</td>
<td>6 weeks</td>
<td>AL, Ap</td>
<td>—</td>
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<td>2</td>
<td>69</td>
<td>M</td>
<td>4 years</td>
<td>AL, Ap</td>
<td>—</td>
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<tr>
<td>3</td>
<td>53</td>
<td>M</td>
<td>2 months</td>
<td>D, PB</td>
<td>—</td>
</tr>
<tr>
<td>4</td>
<td>46</td>
<td>M</td>
<td>4 years</td>
<td>AL</td>
<td>Post CABG and aneurysmectomy</td>
</tr>
<tr>
<td>5</td>
<td>51</td>
<td>M</td>
<td>2 years</td>
<td>D</td>
<td>—</td>
</tr>
<tr>
<td>6</td>
<td>60</td>
<td>M</td>
<td>11 years</td>
<td>AL, Ap</td>
<td>Study was done both before and after CABG and aneurysmectomy</td>
</tr>
<tr>
<td>7</td>
<td>66</td>
<td>M</td>
<td>6 weeks</td>
<td>D, PB</td>
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</tr>
<tr>
<td>8</td>
<td>66</td>
<td>M</td>
<td>4 months</td>
<td>AL, Ap</td>
<td>—</td>
</tr>
<tr>
<td>9</td>
<td>70</td>
<td>M</td>
<td>&lt;6 months</td>
<td>D, PB</td>
<td>—</td>
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MI = myocardial infarction; LV = left ventricular; AL = anterolateral portion; Ap = apical portion; D = diaphragmatic portion; PB = posterobasal portion; CABG = coronary artery bypass graft surgery.
Results

Twelve morphologically distinct episodes of sustained ventricular tachycardia were induced in nine patients. Eight episodes had a right bundle branch block pattern (RB-VT) on the electrocardiogram and four had a left bundle branch block pattern (LB-VT). The characteristics of each episode of ventricular tachycardia, including the rate of the tachycardia and the earliest activation site during the tachycardia, are shown in table 2.

**Transient entrainment of ventricular tachycardia.** Rapid ventricular pacing from the right ventricular apex during RB-VT and that from a selected site in the left ventricle (indicated in table 2) during LB-VT resulted in constant ventricular fusion beats on the electrocardiogram, except for the last captured beat (the first criterion of entrainment).\(^{11,12,16}\) in seven of eight episodes and four of four episodes, respectively. The mean pacing rate was 205 ± 41 beats/min (14 ± 9 beats/min higher than the rate of tachycardia) for RB-VT and 188 ± 37 beats/min (20 ± 14 beats/min higher than the rate of tachycardia) for LB-VT. In the one remaining episode of RB-VT (tachycardia rate 205 beats/min), rapid pacing at 230 beats/min from multiple ventricular sites, including the right ventricular apex, right ventricular outflow tract, and left ventricular apex, resulted in QRS complexes identical to those seen during rapid pacing from each of the respective sites during sinus rhythm. Therefore, seven of eight episodes of RB-VT and four of four episodes of LB-VT were found to be transiently entrained during rapid ventricular pacing, permitting the conclusion that they were due to reentry with an excitable gap.\(^{11,12,16}\) Since one remaining episode of RB-VT was not shown to be transiently entrained during rapid pacing, its underlying mechanism could not be determined with certainty. Therefore, the latter episode was excluded from further analysis. The other 11 episodes of ventricular tachycardia were analyzed further. This analysis is presented below.

**Conduction times during transient entrainment.** Our previous observations of transient entrainment of tachyarrhythmias indicate that when a tachycardia is entrained by rapid pacing, all the tissue responsible for sustaining the tachycardia is activated by the pacing impulse at the pacing rate, either orthodromically (i.e., just as during the spontaneous tachycardia) or antidromically (i.e., from a different direction than during the spontaneous tachycardia).\(^{10-12,16}\) As illustrated in figure 1, when rapid pacing is performed from a site relatively proximal to the area of slow conduction in the reentry loop, site A is activated by the orthodromic wave front of the pacing impulse from the same direction as during spontaneous tachycardia. As a result, the...

### TABLE 2

**Characteristics of ventricular tachycardia and results of rapid ventricular pacing**

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>VT rate (bpm)</th>
<th>Earliest activation site during VT</th>
<th>Pacing site</th>
<th>Pacing rate (bpm)</th>
<th>Entrainment criterion</th>
<th>St-A (msec)</th>
<th>St-B (msec)</th>
<th>Pacing during sinus rhythm</th>
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<tr>
<td>RB-VT</td>
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<tr>
<td>1</td>
<td>145</td>
<td>LV-AL</td>
<td>RVA</td>
<td>156</td>
<td>+</td>
<td>467</td>
<td>20</td>
<td>RVA 130 75 26</td>
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<tr>
<td>2</td>
<td>182</td>
<td>LV-AL</td>
<td>RVA</td>
<td>194</td>
<td>+</td>
<td>291</td>
<td>14</td>
<td>RVA 210 114 14</td>
</tr>
<tr>
<td>3</td>
<td>210</td>
<td>LV-DL</td>
<td>RVA</td>
<td>226</td>
<td>+</td>
<td>315</td>
<td>20</td>
<td>RVA 208 121 22</td>
</tr>
<tr>
<td>4</td>
<td>205</td>
<td>LV-AS</td>
<td>RVA</td>
<td>230</td>
<td>-</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>148</td>
<td>LV-L</td>
<td>RVA</td>
<td>158</td>
<td>+</td>
<td>432</td>
<td>20</td>
<td>RVA 154 100 24</td>
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<tr>
<td>6</td>
<td>194</td>
<td>LV-BL</td>
<td>RVA</td>
<td>200</td>
<td>-</td>
<td></td>
<td></td>
<td>RVA 250 200 18</td>
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<tr>
<td>7</td>
<td>237</td>
<td>LV-D</td>
<td>RVA</td>
<td>269</td>
<td>+</td>
<td>290</td>
<td>28</td>
<td>RVA 225 109 28</td>
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<tr>
<td>8</td>
<td>224</td>
<td>LV-AL</td>
<td>RVA</td>
<td>234</td>
<td>+</td>
<td>292</td>
<td>38</td>
<td>RVA 234 98 52</td>
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<tr>
<td>LB-VT</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
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<td></td>
</tr>
<tr>
<td>1</td>
<td>153</td>
<td>LV-S</td>
<td>LV-PB</td>
<td>163</td>
<td>+</td>
<td>430</td>
<td>52</td>
<td>LV-PB 167 95 48</td>
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<tr>
<td>5</td>
<td>146</td>
<td>LV-S</td>
<td>LV-PB</td>
<td>162</td>
<td>+</td>
<td>424</td>
<td>43</td>
<td>LV-PB 188 70 53</td>
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<tr>
<td>6</td>
<td>200</td>
<td>RVA</td>
<td>LVA</td>
<td>240</td>
<td>+</td>
<td>289</td>
<td>17</td>
<td>LVA 214 65 17</td>
</tr>
<tr>
<td>9</td>
<td>176</td>
<td>LV-S</td>
<td>LV-PB</td>
<td>190</td>
<td>+</td>
<td>360</td>
<td>38</td>
<td>LV-PB 231 81 43</td>
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<tr>
<td>Mean</td>
<td>185</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>SD</td>
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</tbody>
</table>

\(VT = \) ventricular tachycardia; \(LV = \) left ventricle; \(AL = \) anterolateral portion; \(D = \) diaphragmatic portion; \(L = \) lateral portion; \(AS = \) apical septum; \(BL = \) basal lateral portion; \(S = \) septum; \(RVA = \) right ventricular apex; \(PB = \) posterobasal portion; \(LVA = \) left ventricular apex.
electrogram recorded at that site shows the same morphology during pacing as during the spontaneous tachycardia. However, during the same pacing episode, site B is activated by the antidromic wave front of the pacing impulse, i.e., from a different direction than during the spontaneous tachycardia. As a result, the electrogram recorded from site B shows a different morphology during pacing than during the spontaneous rhythm. Thus, the time interval between the pacing stimulus and the electrogram at each site that results from that pacing impulse represents conduction time in either an orthodromic (stimulus-to-site A interval) or an antidromic (stimulus-to-site B interval) direction at the given pacing rate. In this study, we measured the time interval between the stimulus artifact of the last pacing impulse and the last electrogram at each site that resulted from that pacing impulse (i.e., the electrogram captured by the last pacing impulse) because it is then quite clear which pacing impulse is responsible for which electrogram.

Figure 2 shows an example of transient entrainment of RB-VT by rapid pacing from the right ventricular apex (patient No. 3). During the spontaneous tachycardia (the first half of panel A), ventricular fusion developed over several beats until, as shown in panel B, constant ventricular fusion beats developed. With the termination of ventricular pacing (eighth beat in panel B), the last captured beat was entrained but not fused and spontaneous tachycardia resumed. Of note, for each paced beat, the two right ventricular recording sites (apex and outflow tract) were activated by the pacing impulse with a short conduction time, while the left ventricular site (which was the earliest activation site identified during the spontaneous tachycardia) was activated by the previous pacing impulse with a long conduction time. The time interval between the stimulus artifact and the electrogram recorded at the left ventricular site (St-A interval) was 315 msec, while that between the stimulus artifact and the electrogram recorded at a site close to the pacing site with use of the proximal pair of electrodes of a Josephson quadrupolar electrode catheter (St-B interval) was only 20 msec.

Figure 3 shows an example of transient entrainment of LB-VT by rapid pacing from the posterobasal portion in the left ventricle (patient No. 1). During the spontaneous tachycardia (panel A), the earliest activation site identified was in the right ventricular apex, its activation time being –21 msec relative to the onset of the QRS complex on the electrocardiogram during rapid ventricular pacing from the posterobasal portion of the left ventricle (panel B), constant ventricular fusion beats were demonstrated, except for the last captured beat, which was entrained but not fused. The spontaneous tachycardia resumed after termination of pacing. Again, for each paced beat, two left ventricular sites (posterobasal portion and apex) and the right ventricular outflow tract were activated by the pacing impulse with a short conduction time, while the right ventricular apex (the earliest activation site identified during the spontaneous tachycardia) was activated by the previous pacing impulse with a long conduction time. The St-A interval was 430 msec, while the St-B interval was only 52 msec.

Similar observations were made during transient entrainment in all the other episodes of RB-VT and LB-VT (table 2). The St-A interval for all studies was 359 ± 69 msec (350 ± 73 msec for RB-VT studies and 376 ± 66 msec for LB-VT studies), while the St-B interval was only 28 ± 13 msec for all studies (23 ± 8 msec for RB-VT studies and 38 ± 15 msec for LB-VT studies). Thus, during periods of transient entrainment, the site that was activated earliest during the spontaneous tachycardia was activated by the pacing impulse with a very long conduction time.

In contrast, rapid ventricular pacing from the same
site (i.e., that used during the demonstration of transient entrainment) performed during sinus rhythm never resulted in a long St-A interval such as that seen during entrainment. Figure 4 shows examples from the same patients whose records were illustrated in figures 2 and 3. Rapid ventricular pacing from the right ventricular apex at a rate of 208 beats/min resulted in conduction times of 121 msec, corresponding to the previous St-A interval of 315 msec, and 22 msec, corresponding to the previous St-B interval of 20 msec (figure 4, A). Similarly, rapid ventricular pacing from the posterobasal portion of the left ventricle at a rate of 167 beats/min resulted in conduction times of 95 msec, corresponding to the previous St-A interval of 430 msec, and 48 msec, corresponding to the previous St-B interval of 35 msec (figure 4, B). Similar observations were made in all patients (table 2). During the period of demonstrated transient entrainment of ventricular tachycardia, the St-A interval (mean 359 ± 69 msec) was always significantly longer (p < .001) than the St-A interval (mean 103 ± 37 msec) that occurred during overdrive ventricular pacing of sinus rhythm from the same site at the same or a similar pacing rate. However, under the same pacing circumstances, there was no significant difference between the St-B interval during entrainment (mean 28 ± 13 msec) and the St-B interval during overdrive pacing of sinus rhythm (mean 31 ± 15 msec) (table 2).

Prolongation of conduction time with an increment in the pacing rate. In four episodes of RB-VT and four episodes of LB-VT, transient entrainment was demonstrated during rapid ventricular pacing at more than
one pacing rate. This permitted the demonstration of progressive fusion, the second criterion of entrainment (i.e., constant ventricular fusion beats at one constant pacing rate but a different degree of constant fusion at different pacing rates\(^1\) in these eight episodes.

Furthermore, in two episodes of RB-VT and in two of LB-VT, the St-A interval was further prolonged with each increment in the pacing rate, and the St-B interval was not. As illustrated in figure 5, during a spontaneous RB-VT, the earliest activation site was recorded
from the anterolateral portion of the left ventricle. Rapid ventricular pacing from the right ventricular apex at 200 beats/min (figure 6, A) showed constant fusion beats, except for the last captured beat, which was entrained but not fused. The St-A and St-B intervals were 360 and 16 msec, respectively. When the pacing rate was increased to 214 beats/min (figure 6, B) and later to 231 beats/min (figure 6, C), the tachycardia was entrained each time, but with different degrees of constant ventricular fusion beats. Note that the St-A interval was prolonged from 360 msec to 380 msec and then to 390 msec at each pacing rate, respectively, while the St-B interval remained unchanged. When the pacing rate was further increased to 250 beats/min (figure 7, A), although the St-B interval remained constant during the pacing, the St-A interval now gradually prolonged until localized conduction block between the pacing site and the left ventricular recording site occurred for 1 beat. This localized conduction block was associated with interruption of the tachycardia. The following paced beats then activated the left ventricular recording site from a different direction (note

**FIGURE 5.** Electrocardiographic leads I, II, III, and V1 recorded simultaneously with bipolar electrograms at the right ventricular outflow tract (RVOT) and the anterolateral portion of the left ventricle (LV). During this ventricular tachycardia with a right bundle branch block pattern, the earliest activation site identified was in the anterolateral portion of the left ventricle and had an activation time of -10 msec relative to the onset of the QRS complex (indicated by an arrow). RVA = right ventricular apex. The time lines are at 1 sec intervals. All numbers are in msec.

**FIGURE 6.** Electrocardiographic leads I, II, III, and V1 recorded simultaneously with bipolar electrograms at the same sites in the same patient as in figure 5. The proximal pair of electrodes at the right ventricular apex (RVA p) was used for recording electrograms and the distal pair (RVA d) was used for pacing. Rapid ventricular pacing from the right ventricular apex at a pacing cycle length of 300 msec (A), 280 msec (B), and 260 msec (C) was initiated during the tachycardia. Note that during each period of pacing, constant fusion beats except for the last entrained beat are demonstrated and, moreover, progression fusion is demonstrated with the shortening of the ventricular pacing cycle length. Also, note that the conduction time from the pacing impulse to the earliest activation site (LV) progressively prolonged with the shortening of the pacing cycle length, although conduction time from the pacing impulse to the right ventricular outflow tract (RVOT) and right ventricular apex remained unchanged. The asterisks indicate the last captured beats. The time lines are at 1 sec intervals. All numbers are in msec. S = stimulus artifact.
the change in the ventricular electrogram morphology) and with a shorter conduction time (200 msec), fulfilling the third criterion of entrainment. In fact, this site now was activated in the same way as during rapid ventricular pacing at the same rate from the same site during sinus rhythm (figure 7, B).

The relationship between the pacing rate and St-A and St-B intervals measured during each constant pacing rate is shown in figure 8 for all the episodes of ventricular tachycardia that were entrained by rapid pacing at two or more different rates. Although the St-B interval was always constant despite the increment of the pacing rate, the St-A interval was prolonged by more than 10 msec in four of eight episodes as the pacing rate increased. In two other episodes, prolongation of less than 10 msec occurred, but pacing at only two rates was performed. In the remaining two episodes, no prolongation of conduction time occurred despite pacing at three rates. Thus, while the St-A interval during transient entrainment of the tachycardia was always long, the fact that it prolonged unpredictably suggests that the properties of this region vary from patient to patient.

Discussion

The present study provides strong evidence for the presence of an area of slow conduction in the reentry circuit of ventricular tachycardia in man. First and foremost is the demonstration that during entrainment of ventricular tachycardia, the site of earliest ventricular activation is captured orthodromically with a long (mean 359 ms) conduction time (St-A interval). Second, this long conduction time is not present during pacing from that same ventricular site at the same or a similar rate performed during sinus rhythm. Third, this long conduction time spans the diastolic interval at a time when there is no inscription of any portion of the QRS complex on the electrocardiogram. Fourth, after

FIGURE 7. Electrocardiographic leads I, II, III, and V1 recorded simultaneously with electrograms at the same sites in the same patient as in figures 5 and 6. Rapid ventricular pacing from the right ventricular apex at a pacing cycle length of 240 msec was initiated during ventricular tachycardia (A). Note that localized conduction block occurred between the pacing site and the left ventricular recording site (indicated by a star) and was associated with interruption of the tachycardia. After this localized block, all the recording sites were now captured by the pacing impulse with the same activation sequence as when the pacing was performed during sinus rhythm (B). Also, note that after localized conduction block developed, the electrocardiographic morphologies, especially in leads II and III, changed, as indicated by small notches at the end of QRS complex (curved arrow on the electrocardiogram in A). Actually these electrocardiographic morphologies are identical to those seen during rapid pacing performed during sinus rhythm (B). The time lines are at 1 sec intervals. All numbers are in msec. Abbreviations are as in previous figures. See text for discussion.
termination of pacing that only transiently entrained the ventricular tachycardia, the spontaneous tachycardia resumed following activation of the earliest activation site. Fifth, prolongation of conduction time to the earliest activation site (St-A interval) during transient entrainment at two or more different constant pacing rates was observed in half the episodes analyzed. We submit that these observations can be best explained by the presence of an area of slow conduction in a reentry circuit, with the earliest activation site being located just orthodromically distal to or perhaps within the area of slow conduction.

Other possible explanations of the long conduction time (St-A interval) we observed include the possibility of activation of the earliest activation site during entrainment via a very long pathway. However, it seems unlikely that such a pathway without an area of slow conduction exists during ventricular tachycardia because such a pathway would have to be longer than is physically possible to accommodate the long conduction time. Finally, the possibility of a long stimulus latency period should be considered, but the short St-B interval makes this alternative explanation also quite unlikely.

Relationship of the area of slow conduction to the reentry circuit. The demonstration of an area of slow conduction does not necessarily mean it is a critical part of the reentry circuit. For example, ventriculoatrial conduction time may increase with entrainment of a ventricular tachycardia during ventricular pacing. This would be an example of the demonstration of prolongation of conduction time across an area of slow conduction not related to the reentry circuit of the tachycardia, since the ventricular tachycardia reentry circuit is independent of the area of slow conduction (the atrioventricular node) in this example.

That the area of slow conduction demonstrated in the present study is indeed a critical part of the reentry loop is strongly supported by the following: (1) the site of earliest activation during the spontaneous ventricular tachycardia was activated during entrainment with a long conduction time, and after termination of pacing, always preceded the first beat (i.e., the beat that was entrained but not fused) of the spontaneous tachycardia that resumed, and (2) localized conduction block between the pacing site and the earliest activation site was preceded by the demonstration of a long conduction time, and when it occurred, was always associated with interruption of the tachycardia. Thus, these observations indicate that the area of slow conduction was not an innocent bystander, but rather was central to the reentry circuit.

References

7. Wit AL, Allesie MA, Bonke FIM, Lammers W, Smeets J, Fenoglio JJ Jr: Electrophysiologic mapping to determine the mechanism
of experimental ventricular tachycardia initiated by premature impulses. Experimental approach and initial results demonstrating reentrant excitation. Am J Cardiol 49: 166, 1982
Demonstration of the presence of slow conduction during sustained ventricular tachycardia in man: use of transient entrainment of the tachycardia.
K Okumura, B Olshansky, R W Henthorn, A E Epstein, V J Plumb and A L Waldo

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