Reseting of Ventricular Tachycardia by Single Extrastimuli
Relation to Slow Conduction Within the Reentrant Circuit

G. Neal Kay, MD, Andrew E. Epstein, MD, and Vance J. Plumb, MD

Although both transient entrainment and resetting with single extrastimuli have been demonstrated during sustained ventricular tachycardia related to previous myocardial infarction, the relation between these phenomena has not been defined. Because transient entrainment is only demonstrated when the mechanism of a tachycardia is reentry with an excitable gap, the resetting response to timed premature extrastimuli was studied in patients with ventricular tachycardia and correlated with the ability to demonstrate transient entrainment. The importance of the location of pacing and recording electrodes relative to regions of slow conduction within the reentrant circuit for demonstrating specific characteristics of the resetting response after single extrastimuli was examined in 16 patients with 21 distinct morphologies of ventricular tachycardia related to coronary artery disease. At electrophysiological study, intracardiac electrograms were recorded simultaneously from four sites in the right ventricle and four sites in the left ventricle during ventricular tachycardia. Both resetting and transient entrainment could be demonstrated for 18 of the 21 (86%) ventricular tachycardias. The resetting response at each intracardiac recording site was defined as orthodromic or antidromic, based on the conduction time from the pacing stimulus to the recording site and the morphology of the captured (advanced) electrogram. An orthodromic resetting response was associated with demonstration of transient entrainment at 76 of 82 (93%) recording sites, implying that the pacing site was proximal and the recording site was distal to a region of slow conduction. In contrast, an antidromic resetting response was associated with transient entrainment at only six of 154 (4%) recording sites, suggesting that the pacing site was not separated from the recording site by a region of slow conduction (p=0.001). The return cycle at the site of pacing exceeded the tachycardia cycle length in all episodes of ventricular tachycardia. At orthodromically activated recording sites, however, resetting was associated with a return cycle less than the tachycardia cycle length. Thus, orthodromic resetting demonstrates that a pause is not an integral part of the resetting response but that premature extrastimuli preexcite the reentrant circuit by entering the excitable gap, conducting through a region of slow conduction, and emerging distally without a change in activation sequence. In all episodes of ventricular tachycardia, the slope of the return cycle at the pacing site was determined by the conduction properties to the orthodromically activated sites, with increasing patterns (n=6) produced by progressive conduction delay in the reentrant circuit at shorter coupling intervals and flat patterns (n=3) produced by a constant orthodromic conduction interval. A mixed return cycle length pattern (n=9) was observed when the orthodromic conduction interval was constant at long extrastimulus coupling intervals and increasing at shorter intervals. Thus, orthodromic resetting of ventricular tachycardia by single extrastimuli correlates with the ability to demonstrate transient entrainment, suggesting that this response results from entry of a wave of depolarization into an excitable gap with penetration of a region of slow conduction in the reentrant circuit. The demonstration of orthodromic resetting indicates the site of pacing is proximal and the recording site is distal to a region of slow conduction in the reentrant circuit. Both orthodromic and antidromic resetting responses can be observed during a single episode of ventricular tachycardia, depending on the location of pacing and recording electrodes relative to regions of slow conduction within the reentrant circuit. The characteristics of the return cycle during resetting (flat, increasing, or mixed) are well explained by the conduction properties in regions of slow conduction. (Circulation 1990, 81:1507–1519)
Resetting, the advancement of a tachycardia by timed premature electrical stimuli with a pause that is less than fully compensatory, can be demonstrated for most episodes of sustained ventricular tachycardia. However, because resetting has been demonstrated for tachycardias based on several different mechanisms, including reentry, normal or abnormal automaticity, and triggered activity, the value of the resetting response for defining the mechanism of a clinical arrhythmia is uncertain. Although certain features of the resetting response, such as the pattern of the return cycle at progressively shorter coupling intervals, have been proposed as more specific for reentry, similar patterns can be observed with tachycardias based on other mechanisms.

In contrast to resetting, transient entrainment of a tachycardia by a train of pacing impulses has been demonstrated only for tachycardias based on reentry with an excitable gap. Using transient entrainment as a probe of tachycardia mechanism, most episodes of recurrent sustained ventricular tachycardia related to coronary artery disease are based on reentry with a gap of excitability between the advancing wave of depolarization and the preceding trail of refractoriness. Because both transient entrainment and resetting of a reentrant tachycardia are produced by excitation of the circuit by a paced wave front, it is likely that specific features of the resetting response can be identified that are more strongly associated with reentry with a gap of excitability and can be used to explore the mechanism of tachycardias. Additionally, because the demonstration of transient entrainment is critically dependent on the location of pacing and recording electrodes relative to regions of slow conduction in the reentry circuit, it is likely that certain resetting response patterns can demonstrate similar requirements.

To rigorously examine the resetting response in a population of patients with ventricular tachycardia based on reentry with an excitable gap as demonstrated by transient entrainment, we prospectively analyzed these responses in a consecutive series of patients with sustained ventricular tachycardia related to coronary artery disease. We specifically examined the features of resetting that are associated with the ability to demonstrate transient entrainment, the importance of pacing and recording sites relative to regions of slow conduction in the reentry circuit for demonstrating these features, and the factors that determine the pattern of the return cycle.

These studies explain the mechanism of different resetting response patterns, allowing the use of this technique to more definitively explore the role of reentry in clinical arrhythmias and to probe the functional components of the reentrant circuit.

**Methods**

**Entry Criteria**

Patients were included in the study if all the following criteria were met: 1) greater than 70% luminal diameter stenosis of at least one coronary artery documented by coronary arteriography with segmental wall motion abnormalities by left ventriculography; 2) the documented spontaneous occurrence of sustained unimorphic ventricular tachycardia; 3) sustained unimorphic ventricular tachycardia inducible with programmed electrical stimulation; 4) consistency with previous studies of the resetting response, in which the induced ventricular tachycardia had a maximum cycle length variability of less than 20 msec over 20 consecutive beats; 5) ventricular tachycardia hemodynamically well tolerated, allowing completion of the study protocol; and 6) written informed consent.

**Electrophysiological Study Protocol**

Electrophysiological studies were performed in the fasting state at least four half-lives after discontinuation of antiarrhythmic drugs. In all patients, two quadrupolar electrode 6F catheters with an interelectrode distance of 0.5 cm (USCI, Josephson) were introduced from the femoral vein and positioned at the right ventricular apex (RVA) and the right ventricular outflow tract (RVOT). A specially designed octapolar catheter (USCI) was advanced from the femoral artery to the left ventricle (LV) and positioned with the two proximal pairs of electrodes recording activation of the inferior LV and the two distal pairs recording from the anterior LV. Surface electrocardiographic leads I, II, III, and V1, and bipolar intracardiac electrogams filtered at a bandpass of 30–500 Hz were recorded on photographic paper using a switched-beam oscilloscope recorder (model VR-16, Electronics for Medicine) and onto FM tape by a Honeywell 101 recorder (Honeywell, Inc.). A standard 12-lead electrocardiogram was also recorded during the tachycardia.

Programmed electrical stimulation was performed with a pulse width of 2.0 msec and a stimulus amplitude of twofold diastolic current threshold. Sustained unimorphic ventricular tachycardia was induced using one, two, or three extrastimuli coupled to an eight-beat S1 drive.

**Single Extrastimuli During Ventricular Tachycardia**

After induction of sustained unimorphic ventricular tachycardia, single extrastimuli were introduced into the tachycardia from the RVA beginning with a coupling interval equal to the tachycardia cycle length. The coupling interval of the premature
extrastimulus was decreased in 10-msec decrements until termination of the tachycardia or failure of ventricular capture occurred. Single extrastimuli were then introduced during ventricular tachycardia, in the same manner, from the RVOT and the LV.

Rapid Pacing During Ventricular Tachycardia

Rapid pacing was performed during ventricular tachycardia from the RVA at a cycle length 10–20 msec less than that of the tachycardia, with the first stimulus delivered synchronously at the tachycardia cycle length. The pacing train was continued for 15 beats, and then abruptly terminated. If ventricular tachycardia continued, pacing was performed at the same cycle length from the RVOT and then from the LV. The pacing cycle length was decreased in 10-msec decrements, and the same sequence was repeated until the tachycardia had been interrupted or hemodynamic collapse requiring cardioversion occurred. The same sites that had been used to deliver single extrastimuli were used for rapid pacing during evaluation of transient entrainment.

Definitions

Resetting. Resetting of ventricular tachycardia by single extrastimuli was defined as advancement of the tachycardia cycle length by at least 20 msec, measured from the last surface QRS before the extrastimulus (V1) to the first nonpaced surface QRS after the extrastimulus (V3). The paced QRS complex and intracardiac electrograms immediately after the extrastimulus were designated V2. An additional requirement for defining resetting was constant QRS and intracardiac electrogram morphologies for the V1 and V3 complexes.

Flat, increasing, decreasing, and mixed resetting response patterns. The resetting response pattern at the pacing site was characterized by plotting the return cycle length after the extrastimulus (V2-V3) against the coupling interval of the extrastimulus (V1-V2). The response pattern was defined as flat if the return cycle length varied less than 10 msec during a range of extrastimulus coupling intervals of at least 30 msec. A resetting response pattern was
defined as increasing if prolongation of the return cycle length occurred with progressive prematurity of the extrastimulus coupling intervals and was defined as decreasing if the return cycle shortened. Resetting patterns with zones of both increasing and flat return cycle lengths were considered mixed. The V1-V2 and V2-V3 intervals at each of the right and left ventricular recording sites were plotted against the coupling interval of the extrastimulus in the same manner.

Orthodromic and antidromic resetting responses. Two distinct patterns of resetting at each intracardiac recording site were recognized based on the conduction interval from the pacing stimulus to the captured (advanced) electrogram and the morphology of the electrogams at each recording site. An orthodromic resetting response (Figure 1) occurred at an intracardiac recording site if resetting was associated with advancement of an intracardiac electrogram with a conduction interval from the pacing stimulus to the captured electrogram that was greater than the tachycardia cycle length and identical morphology of the spontaneous and captured electrograms (Figure 1, upper panel). An antidromic resetting response (Figure 2) occurred at an intracardiac recording site if resetting was associated with advancement of an intracardiac electrogram with a conduction interval from the pacing stimulus to the captured electrogram that was less than the tachycardia cycle length and differing morphology of the spontaneous and captured electrograms (Figure 2, upper panel).

Criteria for Transient Entrainment

The criteria for the demonstration of transient entrainment of a tachycardia have been previously reported.14,24 At each intracardiac recording site, localized transient entrainment by rapid pacing was considered to be present when the site was activated at the pacing cycle length with a constant morphology of the intracardiac electrogram during both pacing and spontaneous tachycardia and when, after termination of pacing, ventricular tachycardia continued with the last captured intracardiac electrogram.

![Figure 2. Tracings showing antidromic resetting (upper panel) and failure to demonstrate transient entrainment (lower panel). Surface electrocardiographic leads (I, II, III, and V1) and intracardiac recording sites during same episode of ventricular tachycardia as in Figure 1. In upper panel, single premature extrastimulus delivered from RVOTd electrode pair antidromically resets tachycardia (see text). In lower panel, rapid pacing from same site (RVOTd) during same episode of ventricular tachycardia fails to demonstrate transient entrainment. (See text for discussion.) RVAp, right ventricular apex proximal; RVAp, right ventricular apex distal; RVOTp, right ventricular outflow tract proximal; RVOTp, right ventricular outflow tract distal; LV3-8 and LV3-6, inferior left ventricular recording sites and electrode pairs; LV5-6 and LV1-2, anterior left ventricular recording sites and electrode pairs.](http://circ.ahajournals.org/content/circulation/81/5/1510)
TABLE 1. Patient Population

<table>
<thead>
<tr>
<th>Patient (n)</th>
<th>VT morph</th>
<th>VT axis</th>
<th>VT CL</th>
<th>Reset</th>
<th>Resetting pattern</th>
<th>Ortho set</th>
<th>Transient entrainment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>LBBB</td>
<td>LIA</td>
<td>340</td>
<td>+</td>
<td>Flat</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>2</td>
<td>LBBB</td>
<td>LSA</td>
<td>320</td>
<td>+</td>
<td>Mixed</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>3</td>
<td>RBBB</td>
<td>RSA</td>
<td>310</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>4</td>
<td>LBBB</td>
<td>LSA</td>
<td>360</td>
<td>+</td>
<td>Flat</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>5</td>
<td>RBBB</td>
<td>LSA</td>
<td>340</td>
<td>+</td>
<td>Mixed</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>6</td>
<td>LBBB</td>
<td>LSA</td>
<td>410</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>7</td>
<td>RBBB</td>
<td>LSA</td>
<td>405</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>8</td>
<td>LBBB</td>
<td>LSA</td>
<td>435</td>
<td>+</td>
<td>Mixed</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>9</td>
<td>RBBB</td>
<td>RSA</td>
<td>415</td>
<td>+</td>
<td>Mixed</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>10</td>
<td>LBBB</td>
<td>LIA</td>
<td>380</td>
<td>+</td>
<td>Mixed</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>11</td>
<td>RBBB</td>
<td>RSA</td>
<td>505</td>
<td>+</td>
<td>Incr</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>12</td>
<td>RBBB</td>
<td>RIA</td>
<td>330</td>
<td>+</td>
<td>Incr</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>13</td>
<td>LBBB</td>
<td>LIA</td>
<td>350</td>
<td>+</td>
<td>Mixed</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>14</td>
<td>LBBB</td>
<td>LSA</td>
<td>300</td>
<td>+</td>
<td>Incr</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>15</td>
<td>RBBB</td>
<td>LSA</td>
<td>360</td>
<td>+</td>
<td>Flat</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>16</td>
<td>RBBB</td>
<td>LSA</td>
<td>420</td>
<td>+</td>
<td>Incr</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>LBBB</td>
<td>LIA</td>
<td>440</td>
<td>+</td>
<td>Incr</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
</tbody>
</table>

VT, ventricular tachycardia; morph, QRS morphology in ventricular tachycardia; CL, cycle length; reset, tachycardia resetting response demonstrated; Ortho, orthodromic resetting response; LBBB, left bundle branch block QRS morphology; LIA, left inferior axis; (+), present; flat, flat resetting response; LSA, left superior axis; mixed, mixed resetting response; RBBB, right bundle branch block QRS morphology; RSA, right superior axis; (−), absent; incr, increasing resetting response; RIA, right inferior axis.

occurred at the pacing cycle length and was associated with a nonfused surface QRS. This is the intracardiac equivalent of the first criterion of transient entrainment.19

Morphology of Ventricular Tachycardia

Ventricular tachycardias were classified by the morphology of the QRS complex in lead V₁, as either left bundle branch block if the predominant deflection was negative or right bundle branch block if the predominant deflection was positive.

Data Analysis

The resetting and entrainment responses at each intracardiac recording site were recorded during stimulation at the RVA, the RVOT, and one or two left ventricular pacing sites. Statistical analysis was performed using the χ² test or Fisher’s exact test.

Results

Frequency of Resetting and Transient Entrainment of Ventricular Tachycardia

Sixteen patients met the entry criteria and were included in the study. A total of 21 distinct ventricular tachycardias were studied, including 11 with left bundle branch block QRS morphology and 10 with right bundle branch block QRS morphology (Table 1). The mean tachycardia cycle length was 378.8±56.3 msec. Resetting by single extrastimuli was observed for 18 of 21 (86%) episodes of ventricular tachycardia (nine of 10 right bundle branch block and nine of 11 left bundle branch block morphologies, p=NS). The resetting response pattern was flat for three of the 18 ventricular tachycardias, mixed for nine, and increasing for six. The criteria for transient entrainment were satisfied for 18 of 21 ventricular tachycardias, all of which also demonstrated resetting by single extrastimuli regardless of whether the resetting response pattern was flat, mixed, or increasing. Transient entrainment was never observed when resetting could not be demonstrated with single extrastimuli delivered from at least one of the right or left ventricular pacing sites (three patients).

Orthodromic Resetting and Transient Entrainment

The relation of an orthodromic resetting response to transient entrainment is illustrated in Figure 1. In the upper panel, ventricular tachycardia with a left bundle branch block QRS morphology and a cycle length of 435 msec is shown. A single premature extrastimulus (S) delivered from the inferior LV (LV₅₋₆) resets the tachycardia. Note that the V₂ electrograms (marked by asterisks) at the RVAₚ (proximal), RVA₃ (distal), RVOTₚ, RVOT₃, LV₃₋₄, and LV₁₋₂ recording sites are not advanced after the premature stimulus. The V₃ electrogram at these sites (marked by closed circles), however, is advanced with a constant electrogram morphology and a
stimulus-to-captured electrogram interval that exceeds the tachycardia cycle length. For example, the interval from the stimulus to the advanced electrogram measures 502 msec at the RVOTd recording site. This response of the tachycardia to premature extrastimuli is defined as an orthodromic resetting response at each of the intracardiac recording sites because the conduction interval to the captured electrograms exceeds the tachycardia cycle length and is associated with a constant electrogram morphology. The long conduction interval indicates that the paced wave front must have traversed a region of slow conduction before activating the captured recording sites.21 A constant electrogram morphology at the recording sites suggests that the recording site is activated by the wave front of activation spreading from the extrastimulus in the same direction as during spontaneous tachycardia.

In the lower panel, rapid pacing in the same episode of ventricular tachycardia from the same inferior left ventricular pacing site as in the upper panel (LV2,6) activates all recording sites at the pacing cycle length of 390 msec. After termination of pacing, the RVAp, RVOTp, RVOTd, and LV1,2 recording sites are activated at the paced-cycle length (marked by closed circles) and are associated with a nonfused QRS morphology (marked by a star). Note that there is a constant electrogram morphology during pacing and spontaneous ventricular tachycardia at these sites. After pacing, the cycle length of ventricular tachycardia continues at the previous spontaneous interval (435 msec). Thus, pacing from the inferior LV orthodromically resets and transiently entrains this episode of ventricular tachycardia. It should be emphasized that the RVA and LV3,4 electrode pairs are activated orthodromically during resetting with long extrastimulus-coupling intervals but antidromically during rapid pacing. This change from orthodromic to antidromic activation at these sites has been described as a fourth criterion of transient entrainment.19

Antidromic Resetting and Failure to Demonstrate Transient Entrainment

Figure 2 illustrates an antidromic resetting response and failure to demonstrate transient entrainment by pacing from the RVOT during the same episode of ventricular tachycardia as in Figure 1. In the upper panel, a single premature extrastimulus delivered from the RVOTd electrode pair resets the tachycardia. Note that the paced wave front captures all sites with a short conduction interval and a change in electrogram morphology (marked by the letter “a”), indicating that the spread of activation from the pacing site did not traverse a region of slow conduction and that the recording site was activated in a different direction than during spontaneous tachycardia. The return cycle length between the captured electrogram and the next complex of ventricular tachycardia exceeds the tachycardia cycle length at each site. This response of the tachycardia to prema-

ture extrastimuli is defined as an antidromic resetting response.

In the lower panel, rapid pacing from the same site activates all recording sites at the paced-cycle length (390 msec). After termination of pacing, all recording sites associated with the next QRS complex are activated with a cycle length exceeding that of the pacing interval (marked by asterisks). Thus, the criteria for transient entrainment are not satisfied with pacing from this site.

Importance of Orthodromic and Antidromic Resetting Responses for the Demonstration of Transient Entrainment

All episodes of ventricular tachycardia that could be reset by single extrastimuli demonstrated both orthodromic and antidromic resetting responses at different intracardiac recording sites, depending on the location of pacing and recording electrodes. The resetting response was orthodromic at 82 recording sites and antidromic at 154 recording sites. Whether transient entrainment of a recording site was observed during rapid pacing was strongly influenced by whether the resetting response to single extrastimuli at that site was orthodromic or antidromic (Figures 1–4). When an orthodromic resetting response to single extrastimuli was observed, 76 of 82 recording sites (93%) also demonstrated transient entrainment during rapid pacing from the same site (Figures 1, 3, and 4). Only six of 154 antidromically reset sites (4%), however, were transiently entrained by pacing at the same site (p = 0.0001) (Figure 2). The site of pacing also influenced the likelihood of demonstrating an orthodromic resetting response (Table 2). In general, tachycardias with a right bundle branch block QRS morphology were more likely to be orthodromically reset with extrastimuli delivered from the right ventricle. Tachycardias with a left bundle branch block QRS morphology were more likely to be orthodromically reset by extrastimuli from the LV, analogous to previous observations during transient entrainment.12 These findings suggest that orthodromic resetting is more likely to be demonstrated when the pacing site is far from the site of exit from the region of slow conduction in the tachycardia circuit.

Factors Determining the Characteristics of the Return Cycle During Resetting

The return cycle at the pacing site after a premature extrastimulus exceeded the tachycardia cycle length in all episodes of ventricular tachycardia. Thus, at the site of pacing, a noncompensatory pause was observed. The V2-V3 return cycle at sites of orthodromic activation, however, was less than the tachycardia cycle length in all episodes of ventricular tachycardia demonstrating resetting (Figures 5–7). For example, in Figure 5 (upper panel), the V1-V2 and V2-V3 intervals in both the right and left ventricles were plotted against the coupling interval of the extrastimulus (V1-S2) during an episode of ventricular tachycardia with left bundle branch block.
QRS morphology. At the site of pacing in the LV, the V2-V3 return cycle length exceeded the tachycardia cycle length (340 msec). Note that the V2-V3 interval at the orthodromically activated recording site in the RVOT is less than the tachycardia cycle length and decreases with progressive prematurity of the extrastimulus. Thus, although a flat resetting pattern is observed at the site of pacing, the orthodromically activated RVOT recording site demonstrates a decreasing pattern of the return cycle. In Figure 5 (lower panel), the V2-V3 return cycle at the orthodromically activated RVOT recording site is plotted against the V1-S2 coupling interval. Note that the return cycle at this site decreases in direct proportion to the prematurity of the extrastimulus and demonstrates no evidence of decremental conduction. Thus, a flat resetting pattern at the pacing site is attributable to a constant conduction interval through the area of slow conduction in the tachycardia circuit.

The V1-V2 and V2-V3 cycles during resetting of an episode of ventricular tachycardia with right bundle branch block QRS morphology is shown in Figure 6 (upper panel). At the RVA pacing site, the V2-V3 return cycle exceeds the tachycardia cycle length (505 msec) and demonstrates an increasing pattern. The V2-V3 return cycle at the orthodromically activated left ventricular recording site is less than the tachycardia cycle length and demonstrates a decreasing pattern. The increasing slope of the return cycle at the pacing site is explained by examining the V2-V3 intervals at the orthodromically activated left ventricular recording site (Figure 6, lower panel). Because the slope of the V2-V3 intervals at the left ventricular site demonstrates decremental conduction properties with divergence from the slope of the V1-S2 line, propagation of the wave front back to the right ventricular pacing site requires a longer interval, and the return cycle at this site increases with progressive prematurity of the extrastimulus.

A mixed resetting pattern with both flat and increasing zones of the return cycle is shown in Figure 7. At the site of pacing in the RVOT, there is a flat V2-V3 return cycle curve at long coupling intervals and an increasing curve at short V1-S2
coupling intervals (upper panel). At the orthodromically activated left ventricular recording site, the V2-V3 curve is decreasing at long coupling intervals and relatively flat at shorter intervals. When the orthodromically activated left ventricular recording site V2-V3 return cycle is plotted against the V1-S2 coupling interval of the extrastimulus (lower panel), the V2-V3 interval decreases in direct proportion to the prematurity of the extrastimulus at the orthodromically activated left ventricular recording site at long coupling intervals. At short coupling intervals, there is divergence of the orthodromic V2-V3 curve from the slope of the V1-S2 line. As noted for flat and increasing resetting patterns, the slope of the conduction interval to the orthodromically activated recording sites explains the pattern of the return cycle at the pacing site. A decreasing pattern of the return cycle was never observed.

### Discussion

**Implications of an Orthodromic Resetting Response**

The results of the present study indicate that orthodromic resetting and transient entrainment are manifestations of the same phenomenon, that is, premature penetration of a tachycardia circuit by a paced wave front. The specific characteristics of the resetting response relate to the location of pacing and recording electrodes relative to the components of the tachycardia circuit and to the functional characteristics of regions of slow conduction. As might be expected from the definition

---

**TABLE 2. Orthodromic Resetting: Influence of Pacing Site and QRS Morphology**

<table>
<thead>
<tr>
<th>Pacing site</th>
<th>RBBB VT</th>
<th>LBBB VT</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>RV apex</td>
<td>6/10</td>
<td>3/11</td>
<td>NS</td>
</tr>
<tr>
<td>RVOT</td>
<td>7/9</td>
<td>2/10</td>
<td>0.01</td>
</tr>
<tr>
<td>LV inferior</td>
<td>4/9</td>
<td>9/10</td>
<td>0.03</td>
</tr>
<tr>
<td>LV anterior</td>
<td>3/8</td>
<td>7/9</td>
<td>NS</td>
</tr>
</tbody>
</table>

All values expressed as number orthodromically reset/total.

RBBB, right bundle branch block QRS morphology; VT, ventricular tachycardia; LBBB, left bundle branch block QRS morphology; RV apex, right ventricular apex; RVOT, right ventricular outflow tract; LV, left ventricle.

---

**FIGURE 4.** Tracings of orthodromic resetting (upper panel) and transient entrainment (lower panel). Upper panel illustrates orthodromic resetting of same episode of ventricular tachycardia as in Figure 3 with extrastimuli delivered from RVA. Note that LV recording site is captured orthodromically with stimulus-to-captured electrogram interval of 362 msec and constant electrogram morphology (●). Lower panel demonstrates transient entrainment of ventricular tachycardia by rapid pacing from right ventricular apex during same episode of ventricular tachycardia as in upper panel. Note that inferior left ventricular recording sites (LV and LV) are activated orthodromically (●) and occur at pacing-cycle length of 300 msec associated with nonfused QRS morphology (*). Therefore, electrical pacing from right ventricular apex orthodromically resets and transiently entrains ventricular tachycardia. I, II, III, and V, surface electrocardiographic leads; RVA, right ventricular apex proximal; RVA, right ventricular apex distal; RVOT, right ventricular outflow tract proximal; RVOT, right ventricular outflow tract distal; LV, and LV, inferior left ventricular recording sites and electrode pairs; LV and LV, anterior left ventricular recording sites and electrode pairs.
criteria, an orthodromic resetting response with single extrastimulus correlates with the ability to demonstrate transient entrainment with a train of pacing impulses delivered from the same site. Thus, an orthodromic resetting response supports reentry with a gap of excitability in the reentrant circuit as the most likely mechanism. As has been previously observed with transient entrainment, the ability to demonstrate orthodromic resetting is critically dependent on the location of pacing and recording electrodes. The requirements for the location of pacing and recording electrodes relative to the region of slow conduction in the reentrant circuit for the demonstration of an orthodromic resetting are the same as for demonstrating transient entrainment. Thus, as illustrated diagrammatically in Figure 8, an orthodromic resetting response implies that the pacing site is located proximal to a region of slow conduction in the reentry circuit and that the recording site is located distal to this region.

**Implications of an Antidromic Resetting Response**

Demonstration of an antidromic resetting response can indicate a tachycardia mechanism other than reentry with an excitable gap. An antidromic resetting pattern, however, can also be observed during reentry with a gap of excitability, reflecting the location of pacing and recording electrodes relative to regions of slow conduction in the tachycardia circuit. For example, as illustrated diagrammatically in Figure 9, if the pacing site is located distal to a region of slow conduction in the reentry circuit or if the recording sites are located in regions activated proximal to a region of slow conduction, an antidromic resetting response will be observed. It should be reemphasized that failure to demonstrate an orthodromic resetting response does not exclude reentry with an excitable gap as a possible tachycardia mechanism but can reflect the position of stimulating and recording electrodes relative to the region of slow conduction in the circuit. Indeed, all of the ventricular tachycardias that demonstrated an orthodromic resetting response also showed antidromic resetting when the extrastimulus was delivered at a different site. Therefore, in some cases, antidromic resetting can be a phenomenon analogous to "concealed entrainment."  

**Resetting of Tachycardias With Diverse Mechanisms**  

Abnormal automaticity arising from Purkinje fibers or myocardial cells with reduced membrane potential can respond to timed premature extrastimuli by resetting or acceleration of the cycle.
Although triggered earlier on either dromically and antidromically, the stimulus pacing tachycardia has been demonstrated to be rate dependent, with an increased amplitude and shortened coupling interval of delayed afterdepolarizations in response to more rapid pacing rates.9,10,27 Triggered activity has also been demonstrated to respond to premature extrastimuli by resetting.8-11 In contrast, ouabain-induced ventricular tachycardia cannot be entrained by pacing.28 Furthermore, in either abnormal automaticity or triggered activity, resetting of the arrhythmia by timed premature extrastimuli would require depolarization of the site of origin by the paced wave front. In such instances, the site of origin should be activated earlier than expected with an interval from the pacing stimulus to the captured site that is less than the tachycardia cycle length and a change in activation sequence, thereby producing an antidromic resetting response. An orthodromic response, in which an intracardiac recording site is captured with a conduction interval exceeding the tachycardia cycle length and a constant electrogram morphology of both the spontaneous and captured complexes, would be unlikely to occur with tachycardias based on either abnormal automaticity or triggered activity. Although a long conduction interval between orthodromically and antidromically activated sites is evidence of slow conduction, this does not necessarily prove that the region of slow conduction is an integral portion of the tachycardia circuit. If the third or fourth transient entrainment criteria (a change from orthodromic to antidromic activation with a decrease in the conduction interval) can be demonstrated, however, the critical importance of a region of slow conduction in the reentrant circuit can be inferred.14,17,19,20,23

**Importance of Pacing and Recording Sites for Understanding the Resetting Response in Ventricular Tachycardia**

The importance of left ventricular stimulation for demonstration of the resetting response in sustained ventricular tachycardia has been previously emphasized.3 The usefulness of pacing and recording from the LV is underscored by our finding that an orthodromic resetting response would have been detected in only one of 18 ventricular tachycardias without pacing, recording, or both from the LV. The observation that the demonstration of orthodromic resetting usually requires the placement of pacing and recording electrodes in both ventricles is probably explained by the need for pacing stimuli to traverse a region of slow conduction in the tachycardia circuit. Thus, the likelihood of pacing proximal and record-
ing distal to a region of slow conduction is enhanced by placing catheters in both ventricles. The importance of intracardiac electrode location for demonstrating orthodromic resetting underscores the limited insights regarding tachycardia mechanism that can be derived from surface electrocardiographic recordings alone.

**Pattern of the Resetting Return Cycle**

Examination of the resetting response at orthodromically activated recording sites allows recognition that premature extrastimuli advance a tachycardia based on reentry with an excitable gap by entering the gap, traversing a region of slow conduction in the reentrant circuit, and emerging distally to complete the circuit. Rather than producing a pause, the reentrant circuit is preexcited by an amount that is related to the prematurity of the extrastimulus. The pattern of the resetting response return cycle is determined by the conduction properties of the reentrant circuit. Because the pattern of the return cycle (whether flat, increasing, or mixed) can be explained by the slope of the V2-V3 curve at the orthodromically activated site, it is likely that the region of slow conduction in the tachycardia circuit determines the pattern observed. If the conduction time through the region of slow conduction lengthens with progressive prematurity of the extrastimulus, an increasing pattern will be observed at the pacing site. If the conduction time through the region of slow conduction remains constant, the V2-V3 return cycle will decrease in direct proportion to the coupling interval of the extrastimulus at a recording site orthodromically distal to this region, and a flat resetting pattern will be observed at the pacing site. Our analysis also demonstrates that the term "return cycle" might not be appropriate when sites of orthodromic activation are considered because only the V3 electrogram is prematurely activated by the paced wave front.

**Limitations**

The major limitation of the present study is that only patients with ventricular tachycardia related to coronary artery disease were included. The mechanism of ventricular tachycardia in this clinical setting has been demonstrated to be most often explained by reentry with a gap of excitability in the reentrant circuit. Whether the close concordance between an orthodromic resetting response and the ability to demonstrate transient entrainment would be found in patients with ventricular tachycardia related to structural cardiac disorders other than coronary artery disease is uncertain. Another important consideration is that all patients had well-tolerated ventricular tachycardia, allowing completion of the study protocol. Whether more rapid or less well-tolerated
episodes of ventricular tachycardia would demonstrate similar resetting properties is unknown.

Conclusions

The results of this study indicate that resetting and transient entrainment are manifestations of the same phenomenon, that is, premature activation of a tachycardia circuit by a paced wave front. These data suggest that orthodromic activation during resetting with premature extrastimuli is associated with the ability to demonstrate transient entrainment and is dependent on the location of pacing and recording electrodes relative to a region of slow conduction in the reentry circuit. Antidromic activation during
resetting can reflect a pacing site that is distal to the region of slow conduction, a recording site that is proximal to this region, or a mechanism other than reentry with an excitable gap. The properties of the return cycle with resetting are likely to be related to the conduction properties in the region of slow conduction in the reentrant circuit.

References


**Key Words**: electrophysiology • ventricular tachycardia • arrhythmias • transient entrainment
Resetting of ventricular tachycardia by single extrastimuli. Relation to slow conduction within the reentrant circuit.
G N Kay, A E Epstein and V J Plumb

Circulation. 1990;81:1507-1519
doi: 10.1161/01.CIR.81.5.1507

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1990 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/81/5/1507

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/