Reseting of ventricular tachycardia with electrocardiographic fusion: incidence and significance

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ABSTRACT The incidence and significance of fusion of the QRS complex during resetting of sustained ventricular tachycardias (VTs) was determined in 53 VTs induced by programmed stimulation in 46 patients with prior myocardial infarction. All 53 VTs were reset with one or two extrastimuli delivered at the right ventricular apex (RVA); 29 (54.7%) demonstrated fusion of the VT QRS complex coincident with the extrastimulus resetting the VT. Activation time at the RVA during VT (measured from the onset of the VT QRS complex to the first rapid deflection of the RVA electrogram) was longer in VT reset with fusion compared with those without fusion (91 ± 30 vs 33 ± 32 msec; p < .001). A right bundle branch block VT QRS morphology and a rightward and inferior axis were more common in VT reset with electrocardiographic (ECG) fusion. Additionally, the shortest return cycle following the extrastimulus resetting the VT was shorter in VT reset with ECG fusion compared with those without (327 ± 66 vs 423 ± 84 msec; p < .001). Fusion of the endocardial electrogram recorded at the site of VT origin was noted in 11 of 15 VTs that were reset while a recording catheter was positioned at this site, including all eight VTs with evidence of surface ECG fusion and three of seven VTs without fusion. Seventeen VTs were reset from the right ventricular outflow tract as well as the RVA; eight demonstrated QRS fusion at both sites, five from the right ventricular outflow tract only, and four from neither site. In conclusion (1) the phenomenon of resetting of sustained VT with concomitant fusion on the surface ECG and on endocardial electrograms recorded from the site of VT origin is common and is most consistent with a reentrant mechanism with separate entrance and exit sites in the reentrant VT circuit; (2) the proximity of the pacing site to the exit site of the VT circuit is a major determinant of ECG fusion during resetting; and (3) because of the shorter return cycles during VT resetting with ECG fusion, it is postulated that such VTs are likely to have more widely separated entrance and exit sites in the reentrant circuit than VTs reset without fusion.


SUSTAINED uniform ventricular tachycardia (VT) occurring in the setting of prior myocardial infarction has been thought to be due to a reentrant mechanism.1–3 The ability to initiate and terminate this arrhythmia during programmed electrical stimulation has been used as evidence to support this.1–3 However, triggered rhythms may behave similarly, making this a nonspecific finding with respect to the arrhythmic mechanism.4,5

Resetting of sustained rhythms in response to programmed extrastimuli has been observed.5–15 Although fixed fusion of the surface electrocardiographic (ECG) complex during pacing has been proposed as a criterion to establish reentry as the mechanism for the rhythm,6, 8, 10, 16–19 the significance of QRS fusion during resetting of sustained VT with one or two extrastimuli has not been analyzed. We therefore undertook this study to assess the incidence and significance of ECG fusion during resetting of a large number of tachycardias induced by programmed stimulation. The determinants of this phenomenon and its implications for reentry as the likely mechanism of VT in this patient population are discussed.
TABLE 1
Characteristics of VTs studied

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Value</th>
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<tbody>
<tr>
<td>Number</td>
<td>53</td>
</tr>
<tr>
<td>Mean cycle length (msec)</td>
<td>373 ± 60</td>
</tr>
<tr>
<td>QRS morphology</td>
<td></td>
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<tr>
<td>RBBB/LBBB</td>
<td>27/26</td>
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<tr>
<td>QRS axis</td>
<td></td>
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<tr>
<td>Inferior/superior</td>
<td>14/39</td>
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<tr>
<td>Right/left</td>
<td>22/31</td>
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</tbody>
</table>

LBBB = left bundle branch block; RBBB = right bundle branch block.

Methods

Study population. Fifty-three morphologically distinct sustained VTs occurring in 46 patients (mean age 63 ± 9 years) were included for analysis. Characteristics of these VTs are shown in table 1. A right bundle branch block configuration of VT was defined by monophasic, biphasic, or triphasic R waves in lead V1 or a QR wave in lead V4, and a left bundle branch block VT morphology was defined by QS, Rs, or qrS waves in lead V6. All 46 patients had remote myocardial infarctions (anterior 18, inferior 25, anterior and inferior 3), occurring at least 1 month before electrophysiologic study.

The inclusion criteria for VT in this study were: (1) uniform morphology, (2) induction of the tachycardia by programmed electrical stimulation, (3) a stable cycle length, varying by no more than 20 msec over 20 consecutive beats, (4) hemodynamic tolerance of the arrhythmia by the patient, and (5) demonstrated resetting of VT with either single and/or double programmed ventricular extrastimuli delivered at the right ventricular apex (RVA).

Electrophysiologic studies. Electrophysiologic studies were performed in the postabsorptive state after informed consent was obtained. Thirty-six of the 53 VTs were induced while the patient was on antiarrhythmic therapy (type IA 23, type IB 2, types IA and IB 1, amiodarone 10); this was necessary because VT induced in the baseline state was too rapid to be tolerated hemodynamically.

After the insertion of one to five quadripolar electrode catheters, programmed stimulation was performed with a custom-designed programmable stimulator (Bloom Associates Ltd). Three surface ECG leads (I, aVF, and V1) and intracardiac recordings (filtered at 30 to 500 Hz) were displayed on a multichannel oscilloscope (Electronics for Medicine VR-16) and recorded with an ink-jet recorder (Siemens Elema, Magnigraph) at 100 to 250 mm/sec. A standard protocol of programmed electrical stimulation with up to three ventricular extrastimuli and bursts of rapid ventricular pacing were used to induce VT. In selected tachycardias, left ventricular endocardial catheter mapping was performed according to previously described techniques to determine the site of VT origin. This site was defined as the endocardial location recording the earliest electrical activity in the second half of diastole. Fifteen tachycardias had a recording catheter positioned at the site of VT origin during the resetting protocol.

Pacing protocol during VT. All 53 VTs were reset with ventricular extrastimuli delivered at the RVA. In addition, 17 of these VTs also were reset with extrastimuli delivered at the right ventricular outflow tract (RVOT). Programmed extrastimuli were delivered in the following manner:

(1) Single ventricular extrastimuli were delivered with increasing prematurity beginning 20 msec less than the VT cycle length. The coupling interval of the extrastimulus was progressively decreased by 10 msec decrements until ventricular refractoriness was reached, termination occurred, or a sustained change in the VT morphology or cycle length occurred.

(2) Double ventricular extrastimuli were delivered with the first extrastimulus fixed so that it did not reset the VT. To accomplish this, the coupling interval of the first extrastimulus was set either 20 msec longer than the longest coupling interval with which single extrastimuli reset VT or 20 msec longer than the effective refractory period of the single extrastimulus if no VT resetting had been observed with single ventricular extrastimuli. The second extrastimulus was made more premature by 10 msec decrements, beginning with a coupling interval from the first extrastimulus equal to the VT cycle length. The end points of stimulation were the same as with the delivery of single ventricular extrastimuli.

Single ventricular extrastimuli were delivered at the right ventricular apex during all 53 VTs while double extrastimuli from the same site were delivered in 30 VTs. Additionally, 17 tachycardias also had single and/or double extrastimuli delivered at the RVOT, which also reset the VT. This allowed for analysis of the role of stimulation site on producing resetting of VT with concomitant fusion.

Failure to complete the pacing protocol, including single and double extrastimuli in all VTs, was due to (1) acceleration or degeneration of VT to a less hemodynamically tolerated form or (2) a decision on the part of the electrophysiologist that the induced VT should be immediately terminated for the patient’s well-being.

Definitions

(1) Resetting of VT in response to single or double ventricular extrastimuli was said to occur if (a) the interval from the last VT beat before programmed stimulation to the first nonpaced beat was less than fully compensatory by at least 20 msec and (b) VT had the same morphology and cycle length (±20 msec) resumed immediately after the paced beat.

(2) Fusion of the QRS complex coincident with the premature extrastimulus resetting VT was present if (a) this QRS complex was intermediate in morphology between a fully paced complex and a spontaneous VT beat or (b) the extrastimulus causing resetting occurred after the onset of the QRS complex (figures 1 and 2). Three independent observers used surface leads I, aVF, and V1 to determine whether fusion was present.

(3) Local fusion was defined as being present if the premature extrastimulus causing resetting occurred after the onset of the local electrogram at the site of origin without any change in electrogram morphology in the portion of the electrogram preceding the extrastimulus (figures 2 and 3); that is, the initial component of this electrogram remained unchanged when compared with baseline.

(4) The return cycle after premature extrastimulation was measured from the rapid deflection of the local electrogram produced by the premature extrastimulus resetting VT to the onset of the first nonpaced VT QRS complex.

(5) Local activation time at the pacing site was defined as the interval from the onset of the VT QRS complex to the rapid deflection of the local electrogram at the pacing site.

(6) Reset zone was defined as the range of coupling intervals of extrastimuli resulting in VT resetting.

Statistical analysis. Statistical analysis was performed with the unpaired t test (two-tailed) for comparison of continuous variables and the chi-square test or Fisher’s exact test for comparison of categorical variables.

Results

Incidence and determinants of resetting with fusion. All 53 VTs included in this study were reset with programmed extrastimuli delivered at the RVA. Twenty-
nine VTs (54.7%) demonstrated fusion of the QRS complex coincident with the RVA extrastimulus that reset the tachycardia. Eleven of these 29 VTs (37.9%) were initially reset with the stimulus artifact occurring after the onset of the VT QRS complex.

Tachycardias reset with concomitant QRS fusion by extrastimuli delivered at the RVA were compared with VTs reset without QRS fusion (table 2). A right bundle branch block QRS morphology of VT as well as inferior and rightward QRS axes were significantly more common in those VTs reset with fusion compared with those without fusion. Activation time during VT at the RVA was significantly longer (91 ± 30 vs 33 ± 32 msec; p < .001) in those VTs reset with QRS fusion. Although the width of the reset zone determined with single extrastimuli was not significantly different between VT reset with and without fusion, the reset zone determined with double ventricular extrastimuli was 84 ± 41 msec in VTs reset with fusion vs 56 ± 36 msec in VTs reset without fusion, (p = .067). The longest mean coupling interval of single extrastimuli causing resetting with fusion did not differ significantly from the longest mean coupling interval of single ventricular extrastimuli causing resetting without fusion (305 vs 306 msec; p = NS). A similar finding was observed when the longest mean coupling interval of double ventricular extrastimuli resulting in resetting with ECG fusion was compared with that resetting VT without fusion (623 vs 601 msec; p = NS).

The shortest return cycle after single or double RVA extrastimuli was significantly shorter (328 ± 66 vs 423 ± 84 msec; p < .001) in VTs reset with fusion. When the shortest return cycle was corrected for VT cycle length and expressed as a fraction of the tachycardia cycle length, it was still significantly shorter in VTs reset with fusion than in those without fusion (0.89 ± 0.15 vs 1.12 ± 0.09; p < .001). Overall, 23 of the 29 VTs reset with fusion (79%) had a return cycle that was less than the VT cycle length compared with only one of 24 (4.2%) reset without fusion (p < .001).

**Local fusion at the site of origin.** Fifteen VTs were reset with extrastimuli from the RVA during simultaneous recording of the local electrogram at the site of VT origin. Overall, 11 of the 15 VTs demonstrated local fusion as observed by means of the intracardiac electrogram recorded at the site of VT origin during resetting (figures 1 and 2). All eight VTs reset with surface ECG fusion showed local fusion, whereas three of the seven VTs without surface ECG fusion demonstrated this finding. In the remaining four tachycardias, resetting with fusion was not demonstrated on the surface ECG or electrogram recorded at the site of VT origin.

### Table 2

<table>
<thead>
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<th>With fusion</th>
<th>Without fusion</th>
<th>p value</th>
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<tr>
<td>VTCL (msec)</td>
<td>370 ± 52</td>
<td>377 ± 68</td>
<td>NS</td>
</tr>
<tr>
<td>LAT-PS (msec)</td>
<td>91 ± 30</td>
<td>33 ± 32</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Shortest RC (msec)</td>
<td>328 ± 66</td>
<td>423 ± 84</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Shortest RC/VTCL</td>
<td>0.89 ± 0.15</td>
<td>1.12 ± 0.09</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>RZ-singles (msec)</td>
<td>66 ± 38</td>
<td>55 ± 35</td>
<td>NS</td>
</tr>
<tr>
<td>RZ-doubles (msec)</td>
<td>84 ± 41</td>
<td>56 ± 36</td>
<td>.067</td>
</tr>
<tr>
<td>VT QRS morphology</td>
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<tr>
<td>BBB (R/L)</td>
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<td>5/19</td>
<td>&lt;.001</td>
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<td>Axis (R/L)</td>
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<tr>
<td>(Inf/Sup)</td>
<td>12/17</td>
<td>2/22</td>
<td>&lt;.001</td>
</tr>
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**Resetting from the RVOT.** Seventeen VTs reset from the RVA were also reset from the RVOT. Eight VTs demonstrated resetting with QRS fusion at both sites, five from the RVOT only, and four from neither site. No characteristic of VT QRS morphology distinguished tachycardias reset with and without fusion with extrastimuli delivered at the RVOT. However, 11 of 13 VTs reset with fusion from the RVOT had a left bundle branch block morphology with a left superior axis.

### Discussion

**Resetting with fusion: implications for the mechanism of sustained VT.** The VTs included in this analysis all occurred in the setting of coronary artery disease with prior infarction and were induced by programmed stimulation. This subset of sustained VT is the most commonly encountered in clinical practice and has been thought to be due to a reentrant mechanism.\(^1\)\(^-\)\(^3\) However, rhythms originating from a discrete focus, particularly triggered rhythms, may occur in a similar pathologic milieu and can be induced by programmed stimulation.\(^4\)\(^,\)\(^5\) In addition, a number of studies have shown that triggered rhythms produced experimentally in isolated Purkinje fibers may be reset with appropriately timed premature extrastimuli.\(^5\) However, the presence of concomitant fusion of surface ECG complexes makes any mechanism of arrhythmia involving a single focus unlikely.

A complex combination of slow conduction, unidirectional block, and a discharging focus would have to be postulated to explain resetting with ECG fusion in automatic or triggered arrhythmias. As shown in figure 3, A, a premature impulse would have to approach the automatic focus via a “conduit” of excitable tissue; such an arrangement would require unidi-
rectional conduction block, which would allow for a separate entrance to the region of the discharging focus. Without this, the premature impulse would be blocked in the intervening tissue between the pacing site and the focal origin of the arrhythmia and resetting could not occur. In addition, one would have to postulate a prolonged conduction time within this area of protected tissue to allow for recovery of excitability of the focus of arrhythmia origin (figure 3, B). It would be much simpler and more plausible to explain this requirement for (1) separate entrance and exit sites from the site of tachycardia origin, (2) unidirectional block, and (3) slow conduction by a reentrant mechanism. The proposed circuit could be conceptualized as having an area of slow conduction between the entrance and exit sites; this was recently documented in a human model of sustained VT by Okumura et al.24 The finding of resetting with QRS fusion with premature stimulation from both the RVA and RVOT in eight VTs makes an arrangement incorporating a single automatic focus even less likely in these VTs; one would have to postulate separate protected pathways of conduction between the RVA and the site of origin and the RVOT and site of VT origin. In light of this, further considerations of the findings of this study will be discussed in the context of a reentrant mechanism for the VTs that demonstrated resetting with fusion.

Determinants of resetting with fusion. It is not surprising that tachycardias with a QRS morphology different from that produced by pacing at a particular site are more likely to be reset with ECG fusion than without. For example, tachycardias with a right bundle branch block, inferior axis morphology more commonly demonstrated fusion with RVA premature extrastimuli, which characteristically have a left bundle branch block, superior axis morphology. This confirms prior

FIGURE 1. Resetting of sustained VT with concomitant ECG fusion by a single ventricular extrastimulus. In panels A, B, and C, surface ECG leads I, aVF, and V1 are displayed with the intracardiac electrogram from the RVA. In panels B and C, the electrogram from the left ventricular site of VT origin (LV-SOO) is displayed. In panel A, the QRS morphology of fully paced beats from the RVA are shown. In panel B, a single ventricular extrastimulus with a coupling interval of 260 msec results in a return cycle of 430 msec measured at the RVA. A fully compensatory pause would have yielded a return cycle of 450 msec and therefore the VT is reset. Note that the QRS morphology of the VT beat coincident with the extrastimulus is intermediate in morphology between a fully paced beat and a native VT QRS; surface ECG fusion is therefore present. The extrastimulus follows the onset of the electrogram recorded at the site of VT origin and does not alter the morphology of the initial portion of this electrogram compared with baseline. By definition, there is local fusion as well. In panel C, a more premature ventricular extrastimulus (coupling interval 220 msec) results in a less than compensatory pause with concomitant surface ECG and local fusion. Note that the QRS coincident with the premature beat more closely resembles the fully paced QRS complex as opposed to the native VT beat.
FIGURE 2. Resetting of sustained VT with concomitant ECG fusion during the introduction of double ventricular extrastimuli. Panels A, B, and C are labeled as in figure 1. Panel A demonstrates the morphology of fully paced complexes from the RVA. In panel B, the first extrastimulus is set with a coupling interval of 270 msec so that it does not reset the tachycardia. The second extrastimulus is set with a coupling interval of 320 msec to the first and results in a less than fully compensatory pause. Note that the VT QRS complex coincident with the second extrastimulus is intermediate in morphology between a fully paced and native VT beat; surface ECG fusion is therefore present. The electrogram from the site of VT origin (LV-SOO) is unchanged in morphology before the extrastimulus and local fusion is therefore present as well. In panel C, a more premature second extrastimulus (coupling interval 250 msec) resets the tachycardia. However, the VT complex coincident with the second extrastimulus resembles a fully paced beat; surface ECG fusion is not present. Note that the electrogram from the site of VT origin is fully captured by the extrastimulus and that local fusion is absent.

Observations by Plumb et al. during attempted entrainment of sustained VT. Since a critical mass of tissue must be depolarized by the wavefront exiting the reentrant circuit for surface ECG fusion to be seen, premature stimulation near the site of endocardial breakthrough and by inference, the exit of the VT reentrant circuit, is less likely to result in resetting with fusion. Further confirmation of this comes from the finding that the local activation at the pacing site during VTs reset with fusion was significantly longer than in those reset without fusion; that is, the conduction time, if not the anatomic distance, between the exit from the site of VT origin and the site of pacing is longer when resetting with fusion is present.

Insights into the properties of the proposed reentrant circuit are possible from this analysis. The return cycle after premature stimulation, measured from the premature extrastimulus causing VT resetting to the onset of the next QRS complex, is an estimate of conduction time from the pacing site to the entrance of the VT circuit through the circuit and out the site of exit from the reentrant circuit. Those VTs reset with concomitant fusion had a significantly shorter return cycle, both in absolute terms and when corrected for VT cycle length, than did VTs reset without fusion. Seventy-nine percent of VTs reset with fusion had a return cycle less than the VT cycle length compared with only 4% of VTs reset without fusion. This suggests that in VTs reset with fusion, more of the circuit is "short-circuited" by the premature extrastimulus and that there is a wide separation between the entrance and exit sites of the circuits in these VTs (figure 4). Although the proximity of the pacing site to the entrance of the reentrant circuit may influence the return cycle during resetting, the longest coupling interval causing resetting was not significantly different between VTs reset with and without ECG fusion. This suggests that the difference in return cycle was not a function of the site of pacing.
VTs reset without fusion; this difference was of borderline significance. Therefore, because the full extent of the resetting zone was elucidated with double extrastimuli from the RVA, it appeared that the size of the excitable gap became a determinant of fusion.

**VTs reset without ECG fusion.** Nearly one-half of the VTs in this study did not show resetting with ECG fusion with the introduction of RVA extrastimuli. A large proportion of these VTs had QRS morphologies similar to the paced QRS complexes from this site, making fusion difficult to discern on the three electrocardiographic leads monitored. Recordings of intracardiac activity at the site of VT origin revealed that in 43% of instances where surface ECG fusion was not present, one could detect local fusion by analyzing the timing of the presystolic electrogram and its relation to the ventricular premature depolarization. Assuming that the presystolic activity recorded at these sites represents electrical activity within the circuit before its exit to the surrounding myocardium, this finding indicates fusion within the VT circuit. Therefore, looking at the surface ECG alone may be insufficient to determine the true incidence of fusion, and examination of intracardiac recordings may be required in some cases.

**Limitations.** The tachycardias in this study were all introduced by programmed stimulation in patients with prior myocardial infarction and were all uniform in morphology and hemodynamically well tolerated. In addition, all VTs were reset by single and/or double ventricular extrastimuli. The findings cannot be extended beyond this subgroup.

Only three ECG leads were used in the analysis of surface ECG fusion. Although this allows for the identification of the bundle branch block QRS morphology in V<sub>1</sub> and the QRS axis in leads I and aVF, subtle variations in QRS morphology in other leads could be missed. This could lead to an underestimation of the incidence of this phenomenon. Equipment capable of recording 12 simultaneous surface leads would have to be used or trains of multiple extrastimuli would have to be utilized to entrain the VT instead of single or double extrastimuli.

The performance of the majority of the pacing protocol at the RVA limited our ability to determine QRS fusion in those VTs with a similar morphology to the paced beats from this site. The incorporation of pacing from multiple sites, including left ventricular sites, would probably have increased the sensitivity for detecting fusion.

**Conclusion.** In summary, the phenomenon of resetting with concomitant surface ECG fusion is more compatible with a reentrant mechanism for sustained

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**FIGURE 3.** Schematic representation of a possible explanation of resetting with concomitant surface ECG fusion in a tachycardia originating from a single automatic focus. At the top of the figure simultaneous representations of VT recorded on the surface ECG and an intracardiac recording from the pacing site (PS) are displayed. A single extrastimulus (S) resets the tachycardia and occurs after the onset of the VT QRS complex producing concomitant surface ECG fusion. In panel A, a single discharging focus with wavefronts emanating away from it is displayed during the introduction of an extrastimulus from the pacing site. For the wavefront of activation to reach the automatic focus, a protected pathway of excitable tissue would have to remain, which would prevent the premature wavefront from colliding with the wavefront propagating from the VT focus. In panel B, slow conduction in the area of excitable tissue would have to be present to allow for recovery of the VT focus so that it could be excited by the premature wavefront and the VT could be reset.

Being closer to the entrance of the circuit in VTs reset with ECG fusion. Additionally, there was no difference in VT cycle length between the two groups, suggesting that conduction time within the circuit did not account for this finding.

The resetting zone has been used to quantify the size of the excitable gap in reentrant circuits. Given a reentrant mechanism for the tachycardias in this study, there was no difference between the extent of the excitable gap in VTs reset with fusion as compared with those reset without fusion when the zone was determined by single extrastimuli from the RVA. However, Stamato et al. have shown that the resetting zone determined by introducing double ventricular extrastimuli during VT is larger than that determined by single ventricular extrastimuli because of the presumed removal of intervening tissue delay; therefore double ventricular extrastimuli give a more accurate representation of the size of the excitable gap. In this study, VTs reset with fusion had a longer excitable gap as determined by double ventricular extrastimuli than
VT than a mechanism incorporating a single focus. The results of this study suggest that the proposed reentrant circuits of VTs demonstrating fusion are more likely to have more widely separated entrance and exit sites. In addition, proximity of premature stimulation to the exit site from the circuit is also an important determinant of the ability to demonstrate resetting with fusion.

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