Characterization of Double Potentials During Ventricular Tachycardia
Studies During Transient Entrainment

Brian Olshansky, MD; Dalmo Moreira, MD; and Albert L. Waldo, MD

Background. Double potentials have been recorded during reentrant tachycardias in animal models. Although they have also been recorded during ventricular tachycardia in humans, their meaning is uncertain.

Methods and Results. We used transient entrainment as a method to help further understand the meaning of double potentials recorded during ventricular tachycardia in humans. Three patients with ventricular tachycardia (cycle lengths, 500, 450, and 290 msec) were studied. During transient entrainment of ventricular tachycardia (pacing cycle length, 470–260 msec), both double potential deflections were captured at the pacing cycle length. One deflection was captured with a short activation time, and the other deflection was captured with a long activation time. During ventricular pacing, the deflections were associated by the long rather than the short interdeflection interval. At termination of pacing, each double potential deflection was associated with separate but sequential QRS complexes, and each deflection maintained the same electrogram morphology at relatively "slow" overdrive pacing rates. The short interdeflection interval shortened further with faster pacing rates (to less than the ventricular refractory period), making it unlikely that both deflections of the double potential represent active depolarization of the same tissue. In two patients, at a critically rapid pacing rate, one of the double potential deflections changed morphology abruptly, associated with a shortened stimulus-to–double potential time interval (from 520 to 110 msec and from 530 to 240 msec, respectively), indicating a change in the direction of activation that caused that deflection. Interruption of ventricular tachycardia was associated with disappearance of the double potentials at the same recording site. The double potentials did not immediately bracket an area of slow conduction.

Conclusions. These data suggest that double potentials recorded only during ventricular tachycardia represent activation wave fronts on either side of an area of block within a reentrant circuit. Thus, double potentials recorded during ventricular tachycardia in these patients do not appear to represent slow conduction per se but rather appear to represent an area of block at the center of a reentrant circuit around which the reentrant wave front circulates. (Circulation 1993;87:373–381)

KEY WORDS • tachycardia, ventricular • potential, double • entrainment • reentry

Double potentials have been recorded during various tachycardias in humans as well as in animals.1–10 Animal models of reentrant tachycardia, particularly atrial flutter, have demonstrated that double potentials occur in the functional or anatomic center of the reentrant circuit.1,2,6,7,9,10 Using the concepts of transient entrainment to help clarify the relation of the double potential deflections during human atrial flutter, we proposed that double potentials, which are recorded only during human atrial flutter, may result from a similar mechanism, i.e., that they represent activation at the functional center of a reentrant circuit.5 Recently, Kay et al8 recorded double potentials during human ventricular tachycardia, but they interpreted their data as indicating that the double potentials immediately bracketed an area of slow conduction in the reentrant circuit.

In this study, we used the concepts of transient entrainment to help understand the nature of double potentials recorded during human ventricular tachycardia. We tested the hypotheses that 1) the concepts of transient entrainment will help determine the mechanism responsible for double potentials present only during ventricular tachycardia; 2) some double potential electrograms are associated by a long interdeflection interval during transient entrainment of ventricular tachycardia such that each deflection is associated with separate but consecutive QRS complexes; and 3) double potential electrograms recorded during human ventricular tachycardia do not directly reflect conduction across an area of slow conduction critical to the tachycardia reentrant circuit but rather reflect an area of functional block around which the reentrant wave front circulates.

From the University Hospitals of Cleveland/Case Western Reserve University, Cleveland, Ohio.
Supported in part by grant RO1-HL-38404 from the National Institutes of Health, National Heart, Lung, and Blood Institute, Bethesda, Md.; a Research Initiative Award from the American Heart Association, Northeast Ohio Affiliate, Cleveland, Ohio; and a grant from the Wuliger Foundation, Cleveland, Ohio.
Address for correspondence: Brian Olshansky, MD, Associate Professor of Medicine, Division of Cardiology, Loyola University Medical Center, 2160 South First Avenue, Maywood, IL 60153.
Received May 4, 1992; revision accepted September 30, 1992.
Table 1. Individual Patient Data: Relations of the Double Potentials During Transient Entrainment of Ventricular Tachycardia

<table>
<thead>
<tr>
<th>Patient</th>
<th>VT CL (msec)</th>
<th>PCL (msec)</th>
<th>St-x (msec)</th>
<th>St-y (msec)</th>
<th>St-y* (msec)</th>
<th>SI (msec)</th>
<th>LI (msec)</th>
<th>PCL (no.)</th>
<th>XY (msec)</th>
<th>YX (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>500</td>
<td>470–340</td>
<td>170</td>
<td>495</td>
<td>110‡</td>
<td>180</td>
<td>320</td>
<td>14</td>
<td>290</td>
<td>180</td>
</tr>
<tr>
<td>1†</td>
<td>465–450</td>
<td>430–390</td>
<td>130</td>
<td>480</td>
<td>120</td>
<td>120</td>
<td>330</td>
<td>5</td>
<td>340</td>
<td>90</td>
</tr>
<tr>
<td>2</td>
<td>290</td>
<td>280–260</td>
<td>90</td>
<td>360</td>
<td>60</td>
<td>230</td>
<td>3</td>
<td>260</td>
<td>20</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>450</td>
<td>370–280</td>
<td>230</td>
<td>440</td>
<td>240§</td>
<td>180</td>
<td>270</td>
<td>10</td>
<td>190</td>
<td>180</td>
</tr>
</tbody>
</table>

VT CL, ventricular tachycardia cycle length; PCL, range of pacing cycle lengths demonstrating transient entrainment; St-x, time from the last pacing stimulus to the first double potential electrogram during transient entrainment before the fourth criterion was demonstrated. The value reported was determined at the longest pacing cycle length at which transient entrainment was demonstrated; St-y, time from the last pacing stimulus to the second double potential electrogram during transient entrainment before the fourth criterion was demonstrated. The value reported was determined at the longest pacing cycle length at which transient entrainment was demonstrated; St-y*, timing to the y potential with the fourth criterion for transient entrainment demonstrated in the double potential; SI, shorter interval by which the double potential deflections appeared to be associated; LI, longer interval by which the double potentials appeared to be associated; PCL, number of ventricular pacing cycle lengths used; XY, xy interval during ventricular pacing at the longest paced cycle length causing transient entrainment; YX, xy interval during ventricular pacing at the longest paced cycle length causing transient entrainment.

†The same patient as patient 1 on a different day studied off of procainamide.

‡At PCL=440 msec. This abrupt change is quite impressive, because the previous St-y recorded with a long value at a slightly longer pacing cycle length (but much shorter than at the longest pacing cycle length) was 520 msec.

§At PCL=280 msec. This abrupt change is quite impressive, because the previous St-y recorded with a long value at a slightly longer pacing cycle length (but much shorter than at the longest pacing cycle length) was 530 msec.

Methods

Three patients with both spontaneous and inducible sustained monomorphic, hemodynamically stable ventricular tachycardia were studied. Two patients had coronary artery disease, had had a prior myocardial infarction, and had evidence of left ventricular dysfunction (left ventricular ejection fraction <0.40). One of these two patients had been treated chronically with amiodarone before study. The other patient was studied in the absence of antiarrhythmic drug therapy. The third patient had had surgical repair of tetralogy of Fallot as a child and had both right ventricular dilatation and depressed right ventricular function. He was studied in the absence of antiarrhythmic medication on first evaluation but was then restudied while taking procainamide. Data from both studies performed on this latter patient are included. Thus, a total of four studies were performed on three patients.

Electrophysiological Study

After giving informed consent, patients were studied in the postabsorptive, nonsedated state in the cardiac electrophysiology laboratory by standard electrophysiological techniques. Four ECG leads (I, II, III, V1) filtered between 0.05 and 50 Hz were recorded simultaneously with intracardiac electrograms filtered between 30 and 500 Hz. Pacing was performed at twice diastolic threshold with a Medtronic 1349A programmable stimulator. All data were recorded on an Electronics-for-Medicine VR 16 oscilloscopic recorder and also simultaneously on either a Honeywell model 5600C or 100 FM tape recorder for subsequent playback and analysis. All measurements were made from data recorded on paper at a speed of 100 mm/sec.

Ventricular tachycardia was initiated with two premature ventricular stimuli delivered from the right ventricle after a constant drive train of eight beats. Because the patients could tolerate ventricular tachycardia for the duration of the study, mapping was performed to determine the "site of origin" (i.e., earliest ventricular activation with respect to the onset of the QRS complex) of the ventricular tachycardia as well as to characterize double potential deflections before and during ventricular pacing. An exploring USC1 quadripolar catheter electrode with either a 2-mm or 5-mm interelectrode spacing between each electrode of the pair and with a 5-mm spacing between electrode pairs was used for this purpose. Mapping was performed in the left ventricle in the two patients with coronary artery disease and in the right ventricle in the patient with tetralogy of Fallot. Biplane fluoroscopy was used to guide placement of the mapping electrode catheter. The mapping catheter was positioned at a site at which the double potentials could be recorded and at which the recording remained stable throughout the study.

After all the catheter electrodes were at their desired locations, rapid ventricular pacing at twice stimulus threshold from selected ventricular sites at a rate faster than the ventricular tachycardia rate was initiated during the ventricular tachycardia to demonstrate the ECG criteria for transient entrainment. After ventricular capture was established (this required acceleration of all recorded ventricular electrograms to the pacing cycle length), rapid ventricular pacing was performed as tolerated for at least 10 seconds from the right ventricular apex at several pacing cycle lengths (see Table 1). On cessation of pacing, if ventricular tachycardia was not interrupted, the pacing cycle length was shortened by 10 msec, and ventricular pacing was performed again in the same manner. In one patient, pacing was also performed near the site where double potentials were recorded. After this pacing protocol, ventricular tachycardia was terminated by rapid ventricular pacing in all patients, and ventricular electrograms were recorded during sinus rhythm at the same site where double potentials were recorded during ventricular tachycardia. Rapid ventricular pacing was performed again from the same site as during transient entrainment of ventricular tachycardia to observe the influence of pacing on the electrogram morphology at the site where double potentials were present previously.

Definitions

Double potential: A double potential is shown by an electrogram with two distinct deflections per beat at one recording site, with each potential of the double poten-
ventricular tachycardia rate, constant fusion beats were demonstrated in the ECG except for the last captured beat, which was not fused—the first criterion; 2) progressive fusion was demonstrated in the ECG (i.e., constant ventricular fusion beats occurred in the ECG during rapid ventricular pacing at two or more constant rates faster than the ventricular tachycardia rate but with different degrees of constant fusion at each pacing rate)—the second criterion. In addition, the fourth criterion was also used to analyze the data. It states that during tachycardia, when comparing pacing from the same site at two constant rates that are faster than the rate of the tachycardia but fail to interrupt the tachycardia, there is a change in conduction time to and electrogram morphology at a constant electrogram recording site.\textsuperscript{14}

Results

All patients had reproducibly inducible sustained monomorphic ventricular tachycardia of the same 12-lead ECG morphology as during the spontaneous ventricular tachycardia. Both the first and second criteria for transient entrainment were demonstrated for each patient during rapid pacing of ventricular tachycardia. Rapid ventricular pacing at sufficiently rapid rates terminated ventricular tachycardia in all patients. As summarized recently,\textsuperscript{15} these results provide strong evidence that these episodes of ventricular tachycardia were caused by reentry.

Characterization of Double Potentials

Double potentials were recorded from at least one ventricular site in each patient during induced ventricular tachycardia (Figure 1). The relation of the deflection of the double potentials was best determined by assessment of the relation of each deflection at the cessation of rapid ventricular pacing, which demonstrated transient entrainment (three to 14 separate pacing cycle lengths per patient; see Table 1).

Characterization of Double Potentials During Transient Entrainment

During transient entrainment, the following observations were consistently made (Figures 1–4): 1) Both deflections of the double potential were captured during transient entrainment and showed a morphology of each deflection similar to that during the spontaneous ventricular tachycardia, suggesting orthodromic capture of the double potential deflections\textsuperscript{5,12–15}; 2) one deflection of the double potential was always captured with a short stimulus-to-deflection interval (St-x interval; see Table 1) and the other with a much longer stimulus-to-deflection interval (St-y interval; see Table 1); 3) the y deflection was associated with a different (separate) QRS complex than the x deflection, suggesting that at least one deflection of the double potential (the y deflection) was captured after conduction of the circulating reentrant wave front through an area of slow conduction\textsuperscript{5,15,16}; 4) the double potential deflections were seen to be associated by the long rather than the short interdeflection interval, with the long interval always containing an isoelectric baseline that spanned diastole.
Analysis of Temporal Superimposition of Double Potentials During Rapid Pacing Provides Some Special Insights

As indicated above, the two deflections (y and x) of the double potential were seen to be associated by the long interdeflection interval when pacing was terminated, which demonstrated one of the criteria for transient entrainment. With transient entrainment at increasingly faster pacing rates, the short interdeflection interval shortened further such that the two deflections of the double potential ultimately superimposed temporally (Figures 1–4). Such a finding would be unexpected if the potentials were associated by the short interdeflection interval. Shortening of the interdeflection interval was not caused by lengthening of the long interdeflection interval but rather principally by entrainment of the potentials to the increasingly shorter pacing cycle length. Furthermore, this superimposition indicates that the double potentials cannot represent active depolarization of the same tissue, important for the hypothesis that double potentials reflect activation on either side of an area of block around which the reentrant wave front circulates.

Relation of Double Potentials to an Area of Slow Conduction in a Reentrant Circuit

The recording of double potentials may indicate the presence of an area of slow conduction. For example, the atrio-His (A-H) interval is a double potential that reflects slow conduction through the atrioventricular (AV) node, and the H potential reflects the "exit point," that is, His bundle activation after the impulse exits from the AV node. But neither the A nor the H potential is recorded from the area of slow conduction (i.e., from the AV node). In ventricular tachycardia, if recorded double potentials similarly bracketed an area of slow conduction, then the first deflection would represent activation immediately orthodromically proximal to the area of slow conduction, and the second deflection would represent the exit point from that region. However, three lines of evidence from this study indicate that double potentials do not immediately bracket an area of slow conduction.

1) Exit point: In our studies, the second (y) deflection of the double potential was not the earliest site of activation (the exit point) from the area of slow conduction (Figures 1 and 2) during ventricular tachycardia. A distinctly different recording site that did not demonstrate double potentials was closer to the point of exit from an area of slow conduction (Figures 1 and 2). Activation of this latter different site was determined to be through an area of slow conduction for the following reasons: During transient entrainment of ventricular tachycardia, the stimulus-to-electrogram interval recorded at this site was long (Figures 1 and 2); this electrogram preceded the onset of the QRS complex during ventricular tachycardia and preceded the second...
(y) deflection of the double potential, as documented after cessation of pacing (transient entrainment) during the ventricular tachycardia. The latter indicates that during ventricular tachycardia, a site distinctly different from the double potential recording site is closer to the area of slow conduction.

2) Demonstration of the fourth criterion of transient entrainment: The fourth criterion was demonstrated during recording of the double potential electrogram in two patients (Figures 1, 2, and 5). In these two patients, at a critically short pacing cycle length, the second (y) deflection of the double potential manifested an abrupt decrease in activation time (410 and 290 msec, respectively; Table 1) and an abrupt change in morphology. Both indicate a change in direction of the activation wavefront responsible for generating this deflection (Figure 5). However, the other (x) potential of the double potential changed neither its morphology nor its activation time (Figures 1, 2, and 5). This provides further evidence that the double potentials during ventricular tachycardia do not necessarily immediately bracket activation of an area of slow conduction. Moreover, this demonstration of the fourth criterion of transient entrainment strongly suggests that the double potentials reflect activation wavefronts on either side of the center of block around which the reentrant wavefront circulates. This is illustrated diagrammatically in Figure 6. Thus, initially, one deflection (x) represents the wavefront orthodromically proximal to an area of slow conduction, and the other (y) represents the wavefront orthodromically distal to the same area of slow conduction. The long interval between each of the deflections of the double potentials reflects conduction of the circulating reentrant activation wavefront as it travels through portions of the reentrant circuit, including a region of slow conduction (Figure 6). With the pacing demonstration of the fourth criterion (Figure 6), one deflection (x) still represents the wavefront activated orthodromically proximal to an area of slow conduction. But now the other (y) deflection represents activation by an antidromic wavefront on the other side of the central area of block.

From this analysis of the x and y potentials, it follows that if the site from which the double potentials were recorded did immediately bracket an area of slow conduction (which is not our contention), a change in the direction of activation of the y potential should interrupt the tachycardia (it did not). Furthermore, because the interval from the stimulus to the first (x) deflection was short, it does not reflect activation through an area of slow conduction. And as the interval from the stimulus to the second (y) deflection changed timing (greatly decreased), this too, indicates that this interval does not reflect activation through an area of slow conduction.

Further analysis of Figures 1, 2, and 5 lends still more support to the conclusion that each potential of the double potential does not immediately bracket an area of slow conduction. In Figures 1 and 2, the last captured (paced) and entrained potential of the double potential (y*) follows the last captured (paced) potential at the posterior right ventricular outflow tract. Both potentials are activated through a region of slow conduction, but the y* deflection follows the site at which no double
potential was recorded. Thus, y* is unlikely to represent an exit point from that area, because it follows activation of the right ventricular outflow tract (which clearly is not the exit point from the area of slow conduction). Also, in Figure 5, with the last paced (captured) beat, the y* potential appears during the QRS complex of the last captured beat, and the x* potential appears early in the diastolic interval. As evident from the considerable period in diastole from x* to the next y*, the reentrant impulse is still traveling through the area of slow conduction. In Figure 5, activation of y* and the right ventricular outflow tract is not through a region of slow conduction but instead occurs by way of anterograde capture. Activation of y* is earlier in Figure 5, suggesting that the anterograde activation pathway is different from activation through a region of slow conduction and that it is farther from the region of slow conduction.

3) Pacing near the double potential site: For one patient, left ventricular pacing during ventricular tachycardia from a site close to the site from which double potentials were recorded demonstrated a long activation time both to the paced QRS complex and to the recorded right ventricular electrograms (Figure 7). The fact that the paced QRS complex morphology was grossly different from what it was during the ventricular tachycardia suggests that this pacing site was not in the critical area of slow conduction in the ventricular tachycardia reentrant circuit. If it were, and pacing captured the reentrant circuit orthodromically, the QRS complex morphology would be similar during pacing of ventricular tachycardia and during ventricular tachycardia, i.e., there would be concealed entrainment.\(^{15,16,19-22}\) Double Potentials Are Reproducible and Present After Procainamide Infusion

Reinduction of the same ECG QRS complex morphology ventricular tachycardia was associated with the recording of double potentials at the same approximate location during the same tachycardia in another study performed on another day in the patient with tetralogy of Fallot (Figure 8). These double potentials responded to pacing in a manner similar to the first study. Procainamide neither influenced the presence of double potentials nor their response to pacing (transient entrainment).

Double Potential Electrograms Disappear With Tachycardia Termination

 Interruption of ventricular tachycardia was associated with disappearance of the double potentials. A fractionated electrogram was then present at the same recording site during sinus rhythm in all patients (Figure 9).
Subsequent ventricular pacing at the tachycardia cycle length from a site other than the double potential recording site during sinus rhythm did not elicit double potentials at the same recording site (Figure 9).

**Discussion**

In this study of patients, the techniques and principles of transient entrainment helped us to understand the nature of double potential electrograms recorded during ventricular tachycardia. Specifically, the data from these studies demonstrate that 1) double potentials recorded only during ventricular tachycardia are related to the reentrant circuit; 2) double potential deflections recorded during ventricular tachycardia neither immediately bracket an area of slow conduction in the reentrant circuit nor identify the immediate point of exit of the circulating reentrant wave front from an area of slow conduction; and 3) during transient entrainment a) double potentials were associated with the longer interdeflection interval, each deflection being associated with separate but consecutive QRS complexes, and b) the deflections of the double potential can be dissociated by the short interdeflection interval during transient entrainment. The demonstrated relation between each deflection of the double potential suggests that the double potential represents an area of functional block about which the reentrant wave front circulates, with each deflection of the double potential reflecting conduction on either side of this area of functional block.

These data and their interpretation are consistent with studies in animal models of atrial flutter, in which double potentials have been found to represent the functional center of the reentrant circuit,1,2,4,7,9 and with studies in human atrial flutter.5

**Cause of the Double Potentials**

Whether the second (y) deflection of the double potential electrogram described in this paper is caused by electrotonus or far-field effect cannot be determined from the available data. However, the data clearly indicate that double potentials do not represent active depolarization of the same tissue, because the temporal superimposition of the x and y deflections during transient entrainment of ventricular tachycardia makes that impossible (because of ventricular refractoriness). And collision of wave fronts would not be associated with double potentials, as shown previously by Spach et al.23

That the double potential reflects an area of functional block is also supported by the absence of double potential recordings during sinus rhythm or overdrive pacing of sinus rhythm at rates similar to those of the spontaneous ventricular tachycardia. Furthermore, analysis of antidromic capture of one of the double potential deflections involved in the reentrant circuit during transient entrainment indicates that each deflection of the double potential originates from a different portion of the reentrant circuit. This not only supports the interpretation that double potentials reflect an area of block around which the reentrant circuit travels but also further supports the above interpretation of the superimposition of the double potentials during transient entrainment.

That double potentials reflect an area of functional block in this study and in studies of atrial flutter in animal models does not mean that double potentials
cannot reflect anatomic block. An example of the latter is readily seen in the double potentials recorded routinely from the coronary sinus or low right atrium during normal sinus rhythm. One potential represents local depolarization of the atrium, and the other represents local depolarization of the ventricle. Clearly there exists an area of anatomic block, the atioventricular groove, between the areas of local atrial and ventricular activation. And, for each recording site (coronary sinus and low right atrium), the ventricular deflection of the double potential results from a far-field effect, because the electrode is not in contact with the ventricular tissue being activated. Interestingly, these examples of double potential recordings in part reflect activation through an area of slow conduction, the AV node, in that the AV interval of the double potential depends in part on conduction time through this area of slow conduction. If the PR interval were very long, as in prolonged first-degree AV block, the prolonged conduction through the AV node would be reflected in the AV interval of this double potential.

**Possible Alternative Explanations**

1) Double potentials represent an area of slow conduction. Double potential electrograms recorded during tachycardia may represent or bracket an area of slow conduction similar to the phenomenon of the His bundle electrogram recording of the AH interval, which reflects AV nodal conduction, or similar to the split His potential (HH' interval), which is thought to indicate an abnormal area of slow conduction in the His bundle. Although this equivalent explanation has been suggested for double potentials recorded in human ventricular tachycardia, our data do not support this conclusion. This is based on three findings in our study: 1) The second deflection of the double potential was not the earliest activation through slow-conducting tissue during transient entrainment of ventricular tachycardia; 2) the fourth criterion of transient entrainment was seen in one double potential deflection, indicating that a change in activation of part of the double potential can occur without influencing the tachycardia; and 3) in pacing during ventricular tachycardia from a site near the double potentials, the paced QRS complex morphology was shown to be markedly different from that during ventricular tachycardia. Thus, concealed entrainment, which would be expected in pacing from the area of slow conduction, is not present.

The fact that double potentials do not reflect activation immediately bracketing an area of slow conduction is not to say that there is no area of slow conduction in the reentrant circuit. The long interdeflection interval of the double potential results in part from orthodromic activation of the circulating wave front through an area of slow conduction. However, the double potential does not immediately bracket the area of slow conduction.

Fitzgerald et al describe abnormal electrograms recorded during human ventricular tachycardia that may represent the area of slow conduction. Catheter ablation of such areas led to long-term prevention of ventricular tachycardia recurrence. However, their data do not present the same type of electrogram recordings we report. Importantly, the electrograms they recorded are not double potentials but rather appear to be fractionated or to have multiple peaks, consistent with electrograms recorded from an area of slow conduction. In their report, they do not show criteria for transient entrainment, making it somewhat difficult to compare their data with ours. It is probable that the abnormal electrograms they report represent a different limb of the reentrant circuit than we report here. Multiple types of abnormal electrograms can be recorded during ventricular tachycardia, but they do not all represent the same phenomenon. Fractionated electrograms appear to be distinctly different from double potentials and probably represent a different phenomenon from that described here.

2) Double potentials represent activation of a dead-end pathway. Another potential interpretation of our data based on the work of Fitzgerald et al is that the double potential represents an area of slow conduction, with the first deflection of the double potential representing conduction just before or at the entry to an area of slow conduction, thereby being a component of the reentrant circuit. However, the second deflection of the double potential could represent activation of a dead-end pathway outside the reentrant circuit. Such an explanation is not compatible with our data, because the second deflection (y) does not appear to represent the immediate exit point from an area of slow conduction. Also, most importantly, the demonstration of the fourth criterion of transient entrainment indicates that it could not represent activation of a dead-end pathway because it is part of the reentrant circuit. Thus, if the y potential reflects activation of a dead-end pathway, during transient entrainment, it necessarily would still reflect activation over the same pathway as during the spontaneous tachycardia. Therefore, the fourth criterion could not be manifest, because activation from a different direction and with a shorter conduction time could not happen.

3) Double potentials represent activation of tissue not a part of the reentrant circuit. Another potential explanation of our data is that both deflections are outside the reentrant circuit, that is, that double potentials represent activation of "innocent bystander" tissue not part of the reentrant circuit. This is unlikely, not only because of the above explanation but also because both during ventricular pacing from the same site during sinus rhythm and during spontaneous sinus rhythm itself, electrograms recorded from the same tissue do not demonstrate double potential deflections. If double potentials represented slow conduction because of rate alone, then with rapid ventricular pacing during sinus rhythm from the same site as during transient entrainment, double potentials should still have occurred. They did not.

In sum, we have considered several possible alternative explanations for double potentials. The data from the patients in this study indicate that these alternative explanations are unlikely. However, it is possible that more than one type of double potential exists and that other mechanisms can explain double potentials during ventricular tachycardia in other patients.

**Conclusions**

Double potential deflections recorded during ventricular tachycardia can be evaluated further by transient entrainment. The double potential deflections demonstrate a changing relation during ventricular pacing and disappear with interruption of ventricular tachycardia.
Data from this study indicate that the most likely explanation is that double potentials represent activation wave fronts on either side of an area of block around which the reentrant circuit circulates in these cases of ventricular tachycardia.

References


Characterization of double potentials during ventricular tachycardia. Studies during transient entrainment.
B Olshansky, D Moreira and A L Waldo

Circulation. 1993;87:373-381
doi: 10.1161/01.CIR.87.2.373
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1993 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/87/2/373

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/