Further observations on transient entrainment: importance of pacing site and properties of the components of the reentry circuit

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ABSTRACT Transient entrainment of circus-movement tachycardia utilizing an atroventricular (AV) bypass pathway was studied in 13 patients (nine with the orthodromic form, two with the antidromic form, and two with both the orthodromic and antidromic forms). All patients had a left-sided AV bypass pathway. Pacing at selected rates faster than the spontaneous rate was performed during the tachycardia at a site proximal or distal to the AV node, an area of slow conduction within the reentry loop. Rapid pacing from a site proximal to the AV node (from the right atrium during the orthodromic form of the arrhythmia or the right ventricle during the antidromic form of the arrhythmia) always demonstrated at least one of the three entrainment criteria: constant fusion beats except for the last captured beat, which was entrained but not fused (first criterion); progressive fusion (second criterion); localized conduction block to a site(s) for 1 paced beat associated with interruption of the tachycardia followed by activation of that site(s) by the next paced beat from a different direction and with a shorter conduction time (third criterion). In contrast, rapid pacing from a site distal to the AV node (from the right ventricle during the orthodromic form of the arrhythmia, or the right atrium during the antidromic form of the arrhythmia) transiently entrained the tachycardia, but never demonstrated any entrainment criteria because the antidromic wave front from the pacing impulse always blocked in the AV node (concealed entrainment). We conclude that the location of the pacing site relative to the components of a reentry loop is critical to the demonstration of the criteria of transient entrainment; i.e., if it is proximal to an area of slow conduction and/or unidirectional block within a reentry loop, transient entrainment should be demonstrable, but if it is distal, it will not be demonstrable.


TRANSIENT ENTRAINMENT of a tachycardia during rapid pacing is an increase in rate of the tachycardia to the faster pacing rate, with resumption of the intrinsic rate of the tachycardia on either termination of pacing or slowing of the pacing rate below the intrinsic rate of the tachycardia.1–10 We, and subsequently other investigators, have presented evidence that this is best explained by continuous early entrance of the wave front from the pacing impulse into the excitable gap of a tachycardia’s reentry loop.3–8, 10 Furthermore, we3–5 have suggested three criteria, any one of which, if present, establishes transient entrainment of a tachycardia. These criteria are as follows: (1) The demonstration of constant fusion beats in the electrocardiogram during pacing at a constant rate faster than the rate of the tachycardia, except for the last captured beat, which is entrained but not fused. (2) The demonstration of progressive fusion, i.e., constant fusion beats in the electrocardiogram during rapid pacing at any constant rate faster than the rate of the tachycardia, but different degrees of constant fusion beats at different rapid pacing rates. (3) The interruption of a tachycardia during pacing at a rate faster than the rate of the tachycardia associated with localized conduction block to a site or sites for 1 beat followed by activation of that site or sites from a different direction and with a shorter conduction time by the next pacing impulse. We have, however, emphasized that transient entrainment may be present despite the inability to demonstrate any of these criteria,3, 4 and recently two studies10, 11 have suggested the importance of the pacing site for the demonstration of the entrainment criteria.

In this article, using the model of circus-movement tachycardia utilizing an atroventricular (AV) bypass pathway, a rhythm well accepted to be due to reentry,
we further explore transient entrainment and interruption phenomena and demonstrate the importance of the pacing site relative to the components of the reentry loop for the demonstration of transient entrainment.

Methods

Thirteen patients with circus-movement tachycardia utilizing an AV bypass pathway were studied with the use of standard cardiac electrophysiologic techniques. The diagnosis of this tachycardia was made by use of standard criteria.12-15 All patients had a left-sided AV bypass pathway established by standard electrophysiologic criteria.12-15 At the time of the study, 12 patients were not receiving any cardioactive drugs, and one was receiving oral amiodarone. All patients gave informed consent before the study.

Studies were divided into two groups based on the type of tachycardia present. Group 1 (table 1) consisted of 11 patients with circus-movement tachycardia of the orthodromic type (five with manifest Wolff-Parkinson-White syndrome and six with a so-called concealed AV bypass pathway). Group 2 (table 1) consisted of four patients with circus-movement tachycardia of the antidromic type (all manifest Wolff-Parkinson-White syndrome) using a left-sided AV bypass pathway as the antegrade limb of the reentry loop during the tachycardia and the specialized AV conduction system as the retrograde limb. Two patients were included in each group, since they manifested each form of the arrhythmia.

TABLE 1
Patient population, characteristics of the tachycardia, and results of rapid pacing

<table>
<thead>
<tr>
<th>Patient No</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Site of AV-BP</th>
<th>Rate of tachycardia (beats/min)</th>
<th>Rate for interruption (beats/min)</th>
<th>Difference in rates (beats/min)</th>
<th>Site of pacing</th>
<th>Transient entrainment</th>
<th>Criterion for entrainment</th>
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<tr>
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<td>167</td>
<td>24</td>
<td>RV</td>
<td>+</td>
<td>-</td>
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<tr>
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<td>HRA</td>
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</table>

AV-BP = AV bypass pathway; CMT = circus-movement tachycardia utilizing an AV-BP; HRA = high right atrium; LRA = low right atrium; RV = right ventricle; + = present; - = not present.

^Not present because the second pacing rate used interrupted the tachycardia.
leads were recorded simultaneously on photographic paper with either an Electronics-for-Medicine DR-12 or VR-16 switched-beam oscilloscopic recorder. The data were also recorded simultaneously on a Honeywell 5600C FM tape recorder for subsequent playback and analysis. All pacing was performed at twice diastolic threshold with a Medtronic 1349A programmable, battery-powered pacemaker. The pacing procedures in both group 1 and group 2 varied somewhat from patient to patient, depending on the clinical status and requirements of the patient during each study.

Study protocols. In group 1 patients, after the orthodromic form of circus-movement tachycardia utilizing an AV bypass pathway was initiated with standard cardiac pacing techniques, rapid ventricular pacing was performed from the right ventricular apex during the tachycardia. Ventricular pacing was initiated at a rate of 5 to 10 beats/min faster than the spontaneous rate, continued for up to 10 sec, and terminated abruptly. If the tachycardia was not interrupted, rapid ventricular pacing was again initiated, but with an increment in rate of 5 to 10 beats/min. This procedure was repeated until the tachycardia was interrupted. In four of these patients, the same rapid pacing protocol was also performed from the high right atrium during circus-movement tachycardia.

In group 2 patients, the antidromic form of circus-movement tachycardia utilizing an AV bypass pathway was induced with standard techniques. In one patient, both rapid atrial pacing from the low right atrium close to the AV nodal region and rapid ventricular pacing from the right ventricular apex were performed during the tachycardia. In another patient rapid atrial pacing from the high right atrium was performed, and in two others, rapid ventricular pacing from the right ventricular apex was performed during the tachycardia. Consequently, rapid atrial pacing was performed in two patients and rapid ventricular pacing was performed in three patients during antidromic circus-movement tachycardia. For both rapid atrial pacing and rapid ventricular pacing, the pacing protocol was the same as that used in group 1 patients.

Results

Transient entrainment and interruption of the orthodromic form of circus-movement tachycardia utilizing an AV bypass pathway — studies in group 1 patients

Ventricular pacing. Ventricular pacing at a critically rapid rate interrupted the tachycardia in 10 of the group 1 patients, the pacing rate required ranging from 20 to 53 beats/min (mean 30) faster than the spontaneous rate of the tachycardia (table 1). The mean pacing rate required to interrupt the tachycardia was 119% of the spontaneous rate, the range being 113% to 136%. In all instances, rapid ventricular pacing at rates slower than the critically rapid rate only transiently entrained the tachycardia. During ventricular pacing at all rates, the morphology of the QRS complex in all recorded electrocardiographic leads was always the same and was indistinguishable from that during ventricular pacing from the same site during sinus rhythm. Furthermore, the sequence of retrograde atrial activation and morphology of recorded atrial electrograms during the spontaneous tachycardia and during rapid ventricular pacing initiated during the tachycardia were identical. The following is a representative example.

Transient entrainment during ventricular pacing. Figure 1, A shows a recording of the tachycardia in a patient (No. 8) with manifest Wolff-Parkinson-White syndrome. This patient was receiving oral amiodarone at the time of the study. The spontaneous rate of the tachycardia was 128 beats/min (cycle length 470 msec) and the earliest recorded activation in the atria was at the distal coronary sinus site. Initially, rapid ventricular pacing at a rate of 138 beats/min (cycle length 434 msec) was performed for 5 sec, completely capturing the ventricles with 1:1 retrograde activation of the atria (figure 1, B). Both the morphology of all recorded atrial electrograms (the high right atrial site, coronary sinus site, and low right atrium recorded from the His bundle recording site) and the atrial activation sequence during the spontaneous tachycardia (figure 1, A) and during rapid ventricular pacing (figure 1, B) were the same. When rapid ventricular pacing was terminated (figure 2, A), the spontaneous tachycardia resumed promptly after the last captured atrial beat (indicated by asterisks). The first spontaneous cycle length of the tachycardia was longer than that before pacing, primarily because of a very long AV nodal conduction time.

When the ventricular pacing rate was increased to 143 beats/min (cycle length 419 msec), the ventricles were again captured, the atrial rate again increased to the pacing rate, and the morphology of the atrial electrograms and the atrial activation sequence again remained identical to those during the tachycardia at its spontaneous rate (figure 2, B). Once again, with termination of ventricular pacing, the spontaneous tachycardia resumed after the last captured atrial beat (indicated by asterisks). Note that during ventricular pacing at each of these rates (143 and 138 beats/min), although complete capture of the atria and ventricles was evident, and after termination of pacing at each rate, the tachycardia resumed promptly, none of the criteria for the demonstration of transient entrainment was present.

Figure 3 diagrammatically illustrates the cardiac activation sequences consistent with the data obtained during the spontaneous tachycardia, during rapid ventricular pacing, and just after the cessation of the pacing, which resulted in a resumption of the spontaneous tachycardia. During the tachycardia (left panel), the excitation wave front enters the ventricles through the normal AV conduction pathway and returns retrogradely to the atria through the left-sided AV bypass pathway. During rapid ventricular pacing (center panel), each pacing impulse enters into the excitable gap of the reentry loop and preexcites it antidromically and
orthodromically. Each antidromic wave front from the pacing impulse is blocked retrogradely, either because it collides with the orthodromic wave front from each preceding beat (presumably in the specialized AV conduction system) or because it is simply blocked in the AV node due to AV nodal refractoriness. The early entrance of each orthodromic wave front from the pacing impulse results in repeated resetting of the tachycardia, thereby increasing the tachycardia rate to the pacing rate. The atria are activated only orthodromically. Thus, the atrial activation sequence during rapid ventricular pacing is exactly the same as that during the spontaneous tachycardia. Therefore, the tachycardia is transiently entrained by rapid ventricular pacing without the demonstration of any constant fusion beats. With cessation of rapid ventricular pacing (right panel), the orthodromic wave front from the last pacing impulse travels in the atria and reenters the ventricles through the AV node because there is no opposing wave front. This reentering orthodromic wave front then continues the tachycardia. The same was seen in all group 1 patients. Thus, during rapid ventricular pacing, none of the criteria for transient entrainment could be demonstrated, although the tachycardia was, in fact, entrained (concealed entrainment).

**FIGURE 1.** Electrocardiographic leads I and V₁ recorded simultaneously with bipolar electrograms from the proximal and distal pairs of electrodes placed in the high right atrium (HRAp and HRAd, respectively), in the His bundle position (HBP and HBd, respectively), from the proximal, middle, and distal pairs of electrodes placed in the coronary sinus (CSp, CSm, and CSD, respectively), and from the electrodes placed in the right ventricular apex (RV) in a patient with the orthodromic form of circus-movement tachycardia utilizing an AV bypass pathway. A. The spontaneous tachycardia at a rate of 128 beats/min (cycle length 470 msec). B. Rapid ventricular pacing at a rate of 138 beats/min (cycle length 434 msec) initiated during the tachycardia resets the tachycardia rate to the pacing rate. See text for discussion. In this and subsequent figures A = atrial potential; H = His bundle potential; V = ventricular potential; AH = AH interval; S = stimulus artifact; time lines are at 1 sec intervals; and all other numbers are in msec.

**Interruption during ventricular pacing.** In all patients in group 1, interruption of the tachycardia during ventricular pacing resulted when the antidromic and orthodromic wave fronts of the same pacing impulse blocked during the same beat. Two modes of interruption were observed. One mode is illustrated in figure 4 in the same patient whose recordings are shown in figures 1 and 2. When the ventricular pacing rate was increased to 148 beats/min (cycle length 406 msec), the tachycardia was interrupted due to block of both the antidromic and the orthodromic wave fronts of the same pacing impulse in the specialized AV conduction system, presumably in the AV node.

The second mode of interruption is illustrated in figure 5, A, recorded from another patient (No. 1), with a tachycardia at a rate of 143 beats/min. Rapid ventricular pacing at rates slower than 167 beats/min had failed to interrupt the tachycardia, but pacing at a rate of 167 beats/min did interrupt it. During rapid ventricular pacing at this rate, the ventricles were completely captured but, after prolongation of conduction time from the ventricular pacing site to the atria, 2:1 retrograde conduction block to the atria developed. Termination of pacing immediately after a blocked retrograde beat resulted in interruption of the tachycardia.
When subsequent atrial activation of retrograde ventriculoatrial conduction block (figure 5, B), resumption of the spontaneous tachycardia occurred (termination with reinitiation),\(^6\)\(^,\)\(^6\)\(^,\)\(^17\) and the sequence of subsequent atrial activation after retrograde conduction block was the same as that before retrograde conduction block developed.

A diagrammatic illustration consistent with the data during each of the two modes of interruption of the orthodromic form of circus-movement tachycardia caused by rapid ventricular pacing is shown in figure 6. During the period of transient entrainment, the antiodromic wave front of each pacing impulse was always blocked in the specialized AV conduction system. With interruption of the tachycardia, the orthodromic wave front from the last pacing impulse now also was blocked in the AV node in seven patients (left panel) or in the AV bypass pathway in three patients (center panel). All patients in the latter group had a so-called concealed AV bypass pathway and, moreover, showed a long ventriculoatrial conduction time during the tachycardia (241, 205, and 214 msec) compared with those in the former group (range 75 to 154 msec, mean 113). The mean pacing rate required for interruption of the tachycardia was 197 beats/min in the former group and 181 beats/min in the latter. In the remaining group 1 patient, rapid ventricular pacing at rates up to 273 beats/min did not interrupt the tachycardia. It was then elected to perform rapid atrial pacing, which interrupted the tachycardia at a rate of 286 beats/min.

The retrograde conduction time through the AV bypass pathway during the critical pacing rate for interruption of the tachycardia was constant in all patients in whom the orthodromic wave front from the pacing impulse blocked in the AV node, while it showed clear
Wenckebach periodicity and/or 2:1 conduction to the atria in all patients in whom this same wave front blocked in the AV bypass pathway. In the former group, during the critically rapid pacing rate required for interruption of the tachycardia, localized conduction block was not demonstrated. In fact, in this group, only by terminating pacing could one know that interruption of tachycardia had occurred. Therefore, in these patients, the third criterion for transient entrainment also could not be demonstrated. In the latter group, although interruption of the tachycardia was associated with localized conduction block for 1 paced beat in the AV bypass pathway and activation of the site distal to this block by the next paced beat was of shorter conduction time (because of Wenckebach periodicity of retrograde conduction in the AV bypass pathway), it was from the same direction as previously (figure 6, right). The third criterion for the demonstration of transient entrainment was therefore not fulfilled. Nevertheless, because of this localized block for 1 beat, one could anticipate interruption of the tachycardia if pacing was terminated with this blocked beat.\(^3\)

**Atrial pacing.** In four group 1 patients, transient entrainment was also studied with rapid atrial pacing during the orthodromic form of circus-movement tachycardia. Rapid atrial pacing at rates 21 to 76 beats/min (mean 37) faster than the spontaneous rate of the tachycardia was required to interrupt the tachycardia. The mean pacing rate required to interrupt the tachycardia was 120% of the spontaneous rate (range 115% to 136%). Atrial pacing at rates faster than the spontaneous rate of the tachycardia, but slower than the rate required for interruption, always transiently entrained the tachycardia. As described previously,\(^3\) during transient entrainment, the wave front from each pacing impulse entered the reentry circuit via its excitable gap and then traveled both orthodromically and antidromically. Each orthodromic wave front from the atrial pacing impulse repeatedly reset the tachycardia to the pacing rate, while each antidromic wave front from the atrial pacing impulse collided with the orthodromic wave front of the preceding beat. Consequently, during transient entrainment, constant atrial fusion beats, except for the last captured beat (first criterion), were demonstrated in all four patients (table 1). Furthermore, in one of them, progressive atrial fusion (second criterion) was shown in the surface electrocardiogram at different rapid atrial pacing rates.

**FIGURE 4.** Electrocardiographic leads I and V\(_1\) recorded simultaneously with electrograms from the same recording sites in the same patient as in figures 1 and 2. Rapid ventricular pacing at a rate of 148 beats/min (cycle length 406 msec) is initiated during the tachycardia and results in its interruption due to antegrade conduction block in the AV node. Abbreviations as in figure 1.
movement tachycardia, the first criterion of transient entrainment was present in all four patients, the second criterion in one patient, and the third criterion in two of four patients.

Transient entrainment and interruption of the antidromic form of circus-movement tachycardia utilizing an AV bypass pathway — studies in group 2 patients

Ventricular pacing. Rapid ventricular pacing was performed during the antidromic form of circus-movement tachycardia utilizing an AV bypass pathway in

FIGURE 5. Electrocardiographic leads II and V1 recorded simultaneously with bipolar electrograms from electrodes placed in the high right atrium (HRA), His bundle position (HB), right ventricular apex (RV), and from the proximal and distal pairs of electrodes placed in the coronary sinus (CSp and CSd, respectively) in a group 1 patient. The spontaneous tachycardia rate just before the initiation of rapid ventricular pacing was 138 beats/min (cycle length 436 msec). A, Rapid ventricular pacing at a rate of 167 beats/min (cycle length 360 msec) initiated during the tachycardia results in 2:1 ventriculoatrial conduction. The tachycardia is interrupted with the cessation of rapid pacing immediately after retrograde conduction block to the atria. B, Rapid ventricular pacing at the same rate initiated during the same tachycardia as in A, demonstrating termination with reinitiation of the tachycardia when the pacing is abruptly stopped after retrograde activation of the atria via the AV bypass pathway. See text for discussion. Other abbreviations as in figure 1.
three patients (table 1). At a critically rapid rate, ventricular pacing interrupted the tachycardia in all three patients. Rapid ventricular pacing at rates faster than the spontaneous rate but slower than this critical rate always transiently entrained the tachycardia, fulfilling the proposed criteria. The following are representative examples.

**Transient entrainment during ventricular pacing.** Figure 7 was recorded during an antidromic form of circus-movement tachycardia (rate of 190 beats/min; cycle length 316 msec) in patient 12. In this patient who had a left-sided free wall AV bypass pathway, ventricular pacing performed during sinus rhythm always showed an atrial activation sequence starting from the low right atrium, and premature ventricular stimulation produced both a His bundle potential before an atrial potential and an increase in the time interval between the His potential and atrial potential in the His bundle electrogram recording. Therefore, in this patient, the left-sided AV bypass pathway demonstrated unidirectional block, allowing only antegrade conduction.

Initially, rapid ventricular pacing at a rate of 200 beats/min (cycle length 300 msec) was performed for 10 sec. Figure 8, A shows the recording at the termination of pacing at this ventricular pacing rate. During rapid ventricular pacing, ventriculoatrial conduction time from the right ventricular recording site to the coronary sinus recording site was 210 msec. In contrast to ventricular pacing during the orthodromic form of circus-movement tachycardia, the QRS complexes during rapid ventricular pacing now clearly demonstrated constant fusion beats. Furthermore, after cessation of ventricular pacing, the spontaneous tachycardia resumed promptly after the last captured ventricular beat, which was entrained but not fused (i.e., it occurred at the pacing cycle length of 300 msec but had the QRS morphology of the spontaneous antidromic tachycardia). A different degree of fusion (progressive fusion) of the QRS complexes occurred when the ventricular pacing rate was increased to 210 beats/min (cycle length 286 msec) (figure 8, B). When ventricular pacing at this rate was terminated, the spontaneous antidromic tachycardia resumed in exactly the same manner as observed after termination of ventricular pacing at 200 beats/min; i.e., the last captured beat was entrained but not fused.

A diagrammatic illustration consistent with the data shown in figure 8, A and B is shown in figure 9. The left panel in figure 9, A illustrates the spontaneous tachycardia. During the period of transient entrainment during rapid ventricular pacing (center panel), the wave front from each pacing impulse enters the reentry loop via the excitable gap. Each antidromic pacing wave front collides in the ventricles with the orthodromic wave front of the preceding beat, resulting in ventricular fusion beats. However, each orthodromic pacing wave front resets the tachycardia to the pacing rate. The orthodromic wave front from the last pacing impulse travels around the reentry circuit unopposed by a subsequent pacing impulse (as pacing has ceased), so that the last ventricular beat is entrained at the pacing rate, but is not fused (right panel).

The left panel in figure 9, B illustrates the sponta-
FIGURE 8. Electrocardiographic leads I and V\textsubscript{1} recorded simultaneously with electrogram from the same recording sites in the same patient as in figure 7. The arrows in the V\textsubscript{1} recording indicate the onset of the QRS complexes. A, Termination of rapid ventricular pacing initiated during the antidromic form of circus-movement tachycardia utilizing an AV bypass pathway at a rate of 200 beats/min (cycle length 300 msec). During rapid pacing, the ventricular potential recorded in the coronary sinus electrogram results from antegrade activation through the AV bypass pathway. Note the presence of constant ventricular fusion beats, shown in the surface electrocardiogram (indicated by open circles), except for the last captured beat (asterisk), which is entrained (it occurs at the pacing cycle length) but is not fused. The spontaneous tachycardia resumes promptly after the last entrained beat.

B, Termination of rapid ventricular pacing initiated during the tachycardia at a rate of 210 beats/min (cycle length 286 msec). Note the constant ventricular fusion beats (indicated by open circles), with a different degree of fusion from that shown in A. The last captured beat again is entrained at the pacing cycle length, but is not fused (asterisk). The spontaneous tachycardia again resumes after termination of the pacing.

C, Rapid ventricular pacing initiated during the tachycardia at a rate of 220 beats/min (cycle length 273 msec). After the stimulus captures the ventricles (indicated by the star), ventricular fusion beats occur and ventriculoatrial conduction time (measured from the right ventricular electrogram to the atrial potential in the coronary sinus electrogram) gradually prolongs until finally, retrograde conduction block in the AV node develops (blocked curved arrow). This results in interruption of the tachycardia. The two subsequent ventricular paced beats (indicated by black dots) no longer are ventricular fusion beats (note the sudden change in the morphology of the last two QRS complexes). During these 2 beats, note that the ventricular potential in the coronary sinus electrogram (indicated by the cross), which previously had resulted from antegrade activation through the AV bypass pathway, is now activated from a different direction and with a shorter conduction time. Abbreviations as in figures 1 and 7.
Interruption during ventricular pacing. Increasing the pacing rate for this same patient to 220 beats/min (cycle length 273 msec) interrupted the tachycardia (figure 8, C). Three beats after initiation of ventricular pacing, ventricular capture occurred. This was associated with both ventricular fusion beats and a gradual prolongation of ventriculoatrial conduction time. The latter finally resulted in retrograde block in the specialized AV conduction system, presumably in the AV node, so that for one ventricular paced beat, neither the antidromic nor the orthodromic wave front of that ventricular pacing impulse was conducted to the atria. This was associated with interruption of the tachycardia. The two subsequent ventricular paced beats (black dots) that followed the localized conduction block to the atria captured the ventricles, but did not produce any fusion of the associated QRS complex (note the sudden change in the morphology of the last two QRS complexes). Also, these last two ventricular paced beats were conducted retrograde to the atria over the normal specialized AV conduction system, because retrograde AV conduction over the AV bypass pathway was never demonstrated in this patient.

A diagrammatic illustration consistent with the data recorded in figure 8, C, is shown in figure 10. The orthodromic wave front from the ventricular pacing impulse is always blocked due to collision with the orthodromic wave front from the preceding beat (left panel), while the orthodromic wave front from the pacing imp-
pulse shows decremental conduction in the AV node, finally resulting in conduction block (center panel). The latter resulted in interruption of the tachycardia.

The next ventricular paced beat totally captures the ventricles, since there is no orthodromic wave front from a preceding beat with which to collide (right panel). Also, this paced beat enters the atria via the normal specialized AV conduction system (because no retrograde conduction via the AV bypass pathway is present), so that the atria are activated in the same manner as before interruption of the tachycardia. However, note that the part of the ventricles previously activated by the orthodromic wave front from the pacing impulse is now activated from a different direction and with a shorter conduction time (thus fulfilling the third criterion for the demonstration of transient entrainment).

In the other two patients (Nos. 6 and 11) in whom rapid ventricular pacing was performed during antidromic circus-movement tachycardia, retrograde conduction via the AV bypass pathway was present. In these patients, localized conduction block in the specialized AV conduction system (presumably in the AV node) occurred during rapid pacing as described above and was associated with interruption of the tachycardia (figure 11). However, during the next pacing impulse, ventricular and atrial activation in the region from which the coronary sinus electrogram was recorded occurred from a different direction (note the change in morphology of the respective complexes in the coronary sinus electrogram recording in figure 11) and with a shorter conduction time, as this pacing impulse, now unopposed by a preceding orthodromic wave front, spread "antidromically" to these sites. Therefore, in these patients, the third criterion was demonstrated more clearly.

**Atrial pacing.** In contradistinction to rapid ventricular pacing, rapid atrial pacing during the same antidromic form of circus-movement tachycardia utilizing an AV bypass pathway failed to fulfill any transient entrainment criteria. Figure 12 was recorded from the same patient whose recordings are shown in figures 7 and 8. Rapid atrial pacing was performed from the low right atrium close to the region of the AV node. Rapid atrial pacing at a rate of 220 beats/min (cycle length 273 msec) was performed for about 6 sec during the tachycardia. Each atrial pacing impulse captured the atria and consequently the ventricles. When pacing was terminated, the spontaneous tachycardia resumed promptly after the last captured ventricular beat (figure 11).

![FIGURE 11. Another example of interruption of the antidromic form of circus-movement tachycardia utilizing an AV bypass pathway by rapid ventricular pacing. Electrocardiographic leads I and V1 are shown simultaneously with bipolar electrograms from electrodes placed in the high right atrium (HRA), coronary sinus (CS), and right ventricular apex (RV). The long and short curved arrows represent ventriculoatrial and AV conduction, respectively. The rate of the spontaneous tachycardia is 201 beats/min (cycle length 298 msec). Rapid ventricular pacing at a rate of 216 beats/min (cycle length 278 msec) initiated during the tachycardia produces ventricular fusion beats (indicated by open circles). Ventriculoatrial conduction time measured from the right ventricular stimulus artifact to the atrial potential in the coronary sinus electrogram gradually prolongs during rapid pacing. Finally, retrograde conduction block develops in the AV node (blocked long curved arrow), resulting in interruption of the tachycardia. The subsequent ventricular paced beats totally capture the ventricles (note the sudden change in the morphology of the QRS complexes indicated by black dots) and, moreover, activate the atria through the AV bypass pathway (i.e., from a different direction and with a shorter conduction time). The last ventricular complex (indicated by an asterisk) is a premature beat. Other abbreviations as in figure 1.](http://circ.ahajournals.org/content/64/6/1303/F1.large.jpg)
12, A). No constant fusion beats were manifest during the pacing. This is because each antidromic wave front of each atrial pacing impulse was either blocked in the AV node or collided in the normal specialized AV conduction system with the orthodromic wave front of the previous beat, while the orthodromic wave front of each atrial pacing impulse activated the ventricles via the AV bypass pathway just as during the spontaneous tachycardia (figure 13, left).

Rapid atrial pacing at a rate of 230 beats/min (cycle length 261 msec) caused antegrade conduction block at the AV bypass pathway and terminated the tachycardia (figure 12, B). This occurred because both the antidromic and orthodromic wave fronts of the same pacing impulse were blocked during the same beat, the former in the AV conduction system and the latter in the AV bypass pathway (figure 13, center). It was critical that the pacing be terminated with this blocked beat, because if the pacing was terminated after conduction of an orthodromic wave front (note the presence of 2:1 AV conduction via the AV bypass pathway in figure 12, B and C), the tachycardia continued (termination with reinitiation) (figure 12, C).6, 16, 17 Note that although interruption of the tachycardia was associated with localized conduction block in the AV bypass pathway, the next paced beat activated the ventricles via the AV bypass pathway, i.e., from the same direction as before the localized block (figure 13, right). Therefore, none of the transient entrainment criteria were demonstrated despite the fact that rapid atrial pacing did in fact transiently entrain the antidromic form of circus-movement tachycardia (concealed entrainment). The same observations were made in the other patient in whom rapid atrial pacing was performed from the high right atrium during the antidromic form of circus-movement tachycardia.

**FIGURE 12.** Electrocardiographic leads I and V1 recorded simultaneously with electrograms from the low right atrium (LRA) close to the AV nodal region and at the same recording sites in the same patient as in figures 7 and 8. A, Rapid atrial pacing at a rate of 220 beats/min (cycle length 273 msec) is performed from the low right atrial site during the antidromic form of circus-movement tachycardia utilizing an AV bypass pathway. No constant fusion beats are manifest. With termination of rapid atrial pacing, the spontaneous tachycardia resumes after the last entrained beat (indicated by asterisks). B, Rapid atrial pacing at a rate of 230 beats/min (cycle length 261 msec) causes 2:1 antegrade conduction block at the AV bypass pathway (blocked arrows) and interrupts the tachycardia. C, Rapid atrial pacing at the same rate as shown in B demonstrating termination with reinitiation. See text for discussion. Other abbreviations as in figure 1.

**FIGURE 13.** Diagrammatic illustrations of cardiac activation sequences consistent with the data shown in figure 12. Left, During rapid atrial pacing initiated during the antidromic form of circus-movement tachycardia utilizing an AV bypass pathway, the pacing impulse enters the reentry loop via the excitable gap. The antidromic (ANTI) wave front from the pacing impulse (Xa) collides with the orthodromic wave front of the previous beat (Xb − 1), presumably in the AV node, while the orthodromic (ORTHO) wave front from the pacing impulse (Xa) resets the tachycardia to the pacing rate. Center, At a critically rapid rate, the orthodromic wave front from the pacing impulse (X) is blocked in the AV bypass pathway. The termination of pacing immediately after the conduction block results in interruption of the tachycardia. Right, The following atrial paced beat enters the ventricles through the AV bypass pathway and activates them in the same manner as before interruption of the tachycardia. Other abbreviations as in figure 3.
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Discussion

These data both confirm and expand our previous observations of transient entrainment and interruption of tachyarrhythmias. They confirm the reliability of the three proposed criteria for its identification, demonstrate and explain that both transient entrainment and interruption of a tachyarrhythmia may occur without fulfilling any of the three criteria (concealed entrainment), permit localization of an area of slow conduction within the reentry loop relative to the pacing site, and force a reexamination of the third criterion concerning localized conduction block associated with interruption of the tachycardia.

Reliability of three criteria proposed for identification of transient entrainment. Through our series of studies of transient entrainment and interruption of tachyarrhythmias during rapid pacing, we have explained its mechanism and have proposed three criteria for its identification, any one of which establishes its presence and, we propose, indicates that the underlying mechanism of the arrhythmia can be best explained by reentry. In the present study, we again observed transient entrainment and interruption of the orthodromic form of circus-movement tachycardia utilizing an AV bypass pathway during rapid atrial pacing, and obtained the same results concerning the three criteria as noted in our previous study when pacing was performed proximal to an area of slow conduction. Furthermore, rapid ventricular pacing was performed during the antidromic form of this well-accepted reentrant tachycardia, and the data not only were consistent with our previous explanation of the mechanism of transient entrainment and interruption of tachyarrhythmias, but also demonstrated the three criteria clearly.

During rapid ventricular pacing of the antidromic form of circus-movement tachycardia, transient entrainment was again found to be consistent with the continuous early entrance of the wave front from the pacing impulse into the excitabile gap of the tachycardia's reentry loop. Constant ventricular fusion beats, except for the last captured beat, were clearly demonstrated in the electrocardiogram during rapid ventricular pacing (the first criterion). This can best be explained by collision of the antidromic wave front from each pacing impulse with the orthodromic wave front of each previous beat except for the last captured beat, which was entrained but not fused. The latter occurred because the orthodromic wave front of the last paced beat had no antidromic wave front from a subsequent pacing impulse with which to collide. As the pacing rate was increased, the antidromic wave front from each pacing impulse penetrated the reentry loop to a greater degree, thus producing a different degree of constant ventricular fusion beats in the electrocardiogram (progressive fusion, the second criterion). Finally, when the pacing rate was increased to a rate critically faster than the rate of the spontaneous tachycardia, it was interrupted, being associated with localized conduction block (at the AV node) of the orthodromic wave front from the pacing impulse followed by subsequent activation of sites distal to the localized block from a different direction (manifest by a different electrogram morphology) and with a shorter conduction time (the third criterion). This change in the sequence of activation after localized conduction block was well shown in the electrocardiogram by the change in the morphology of QRS complex. Therefore, the present study supports the previous observations about transient entrainment and interruption of tachyarrhythmias and, moreover, confirms the reliability of the three criteria proposed for its demonstration.

Concealed entrainment: the importance of pacing site in the inability to demonstrate transient entrainment despite its being operative. As shown by this study, none of the proposed three criteria necessary to establish transient entrainment of a tachycardia and, ergo, reentry as its mechanism, may be demonstrable despite the fact that transient entrainment during rapid pacing is certainly occurring (concealed entrainment). That is, transient entrainment occurred without fulfilling any of the criteria when rapid pacing was performed at sites orthodromically distal to the AV node, an area of slow conduction within the reentry loop. This was manifest during rapid ventriculard pacing of the orthodromic form of circus movement tachycardia utilizing an AV bypass pathway and during rapid atrial pacing of the antidromic form. In each case, the antidromic wave front of each pacing impulse blocked in the specialized AV conduction system, presumably in the AV node, so that the demonstration of fusion beats in the electrocardiogram was not possible. Therefore, fulfillment of either the first or second criterion was impossible.

Recently, the importance of the pacing site in demonstrating the criteria for entrainment has been shown in two studies during rapid ventricular pacing from different sites during ventricular tachycardia. In both studies, rapid ventricular pacing from one site fulfilled all three entrainment criteria, while that from another site did not fulfill any of the criteria. Extrapolating from the data in our present study, the results of these two studies are likely explained by the relationship of the pacing site to an area of slow conduction in the reentry loop of the tachycardia.

In the present study, since the pacing site that failed
to demonstrate the entrainment criteria (the right ventricle in the orthodromic form and the right atrium in the antidromic form of circus-movement tachycardia) was not precisely but rather was relatively distal to the AV node (in terms of the reentry loop), some degree of constant fusion might have been expected during transient entrainment. In fact, no constant fusion beats were demonstrated. This may have been because conduction time of the antidromic wave front from the pacing impulse to the area of slow conduction was not long enough to permit the orthodromic wave front of the previous beat to have conducted through and exited the area of slow conduction and thus permit fusion to occur. In addition, the prolongation of the already slow conduction of the orthodromic wave front in the area of slow conduction in the reentry loop (i.e., in the AV node) caused by the rapid rate of pacing made it even more likely that the antidromic wave front from each pacing impulse would reach and block in the AV node.

The inability to fulfill the third criterion in these same examples can be explained in one of two ways. During rapid pacing at a critical rate, either the antidromic and orthodromic wave fronts from the same pacing impulse both blocked at the same time (the AV node), resulting in the interruption of the tachycardia without producing localized conduction block, or the antidromic wave front from the pacing impulse blocked in the AV node and the orthodromic wave front from the same pacing impulse blocked at the AV bypass pathway, resulting in the interruption of the tachycardia. Actually, in the latter form of interruption, localized conduction block to the atria during rapid ventricular pacing or to the ventricles during rapid atrial pacing did occur (in the AV bypass pathway and activation of sites distal to the AV bypass pathway by the next pacing impulse occurred with a shorter conduction time than immediately before the appearance of localized conduction block. However, this activation occurred from the same direction as before the appearance of the localized conduction block. The third criterion requires activation of the blocked sites by the next paced beat from a different direction.

The occurrence of these two types of interruption depends on the electrophysiologic properties of the AV bypass pathway. Localized conduction block to the atria during rapid ventricular pacing of the orthodromic form of circus-movement tachycardia occurred at the AV bypass pathway in patients who manifested long ventriculoatrial conduction time during the tachycardia. Furthermore, this block was preceded by decremental conduction in the AV bypass pathway. The prolongation of retrograde conduction time in the AV bypass pathway of these patients was different from that in other patients in this study in whom retrograde conduction time in the AV bypass pathway was constant at each pacing rate. In the latter patients, it was not until the cessation of rapid ventricular pacing that termination of the tachycardia could be appreciated. In the former patients, i.e., those with prolonged retrograde conduction time, if rapid pacing was terminated immediately after localized conduction block, it resulted in the interruption of the tachycardia, but if it was terminated even 1 beat beyond that, the tachycardia would resume because the orthodromic wave front from that pacing impulse would continue around the reentry loop unopposed (termination with reinitiation). 6, 16, 17

**Localization of an area of slow conduction and/or unidirectional block — relationship to the pacing site.** This study shows that the location of the pacing site is critical for the demonstration of transient entrainment and interruption of a tachyarrhythmia. When it is relatively proximal to an area of slow conduction within a reentry loop, transient entrainment during rapid pacing usually will be demonstrated by the fulfillment of at least one of the three proposed criteria. If the pacing site is relatively distal to an area of slow conduction, transient entrainment will not be demonstrable, since none of the criteria can be fulfilled.

With the use of the observations obtained in this and previous studies of circus-movement tachycardia utilizing an AV bypass pathway, 3 identification of an area of slow conduction relative to the pacing site may be possible. Thus, if one can demonstrate transient entrainment during rapid pacing initiated during the tachycardia, the pacing site must be located relatively proximal to an area of slow conduction. After transient entrainment is established, and if one paced from another site but cannot demonstrate any of the three criteria (concealed entrainment), the latter pacing site is most likely distal to an area of slow conduction. An exception to the latter might occur when an area of slow conduction and an area of unidirectional block are at separate sites and rapid pacing is performed just distal to an area of unidirectional block but proximal to an area of slow conduction. This will occur during pacing from the coronary sinus just distal to a left-sided AV bypass pathway during the orthodromic form of circus-movement tachycardia utilizing such a pathway when the pathway either doesn’t conduct antegradely at all (a so-called concealed AV bypass pathway) or has a very long antegrade effective refractory period. Although such a pacing site is relatively proxi-
mal to an area of slow conduction, its being just distal to an area of unidirectional block with the aforementioned properties will not permit any of the proposed criteria for the demonstration of entrainment to occur. Nevertheless, if any of the criteria are demonstrable, the conclusion that a pacing site is proximal to an area of slow conduction can be drawn. Thus, observing for transient entrainment from multiple pacing sites during a tachycardia may permit the localization of an area of slow conduction critical for establishing and/or maintaining a reentrant tachycardia, as, for example, in ventricular tachycardia.

Reexamination of the third criterion for the demonstration of transient entrainment. We have proposed that if interruption of a tachycardia is associated with localized conduction block to a site(s) for 1 beat followed by subsequent activation of that site(s) from a different direction and with a shorter conduction time, transient entrainment has been demonstrated. In this and a previous study we have observed that localized conduction block for 1 beat (second-degree block) may occur either in the AV node or AV bypass pathway, followed by activation of the previously blocked site(s) by the next pacing impulse with a shorter conduction time. However, different from the originally proposed third criterion, this subsequent activation may be from the same direction as before, and rather than maintain interruption of the tachycardia, it may result in its reinitiation (termination with reinitiation phenomenon). Thus, if pacing is stopped 1 beat after the pacing beat that produced the localized conduction block, the tachycardia may resume.

We have wondered whether these events should be considered as evidence of transient entrainment, i.e., whether the originally proposed third criterion should be expanded or otherwise amended. As shown in this study, this form of interruption of a tachyarrhythmia can occur without demonstration of either of the other two proposed criteria. However, rapid pacing of a tachycardia due to any mechanism can possibly produce a second-degree block of the type described above so that this form of block may be “an innocent bystander” to the underlying mechanism of the tachycardia. Therefore, we suggest that because of our currently limited understanding of mechanisms of arrhythmias and the importance of the demonstration of transient entrainment as a tool to establish a reentry mechanism, the third criterion should remain as is.

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