Ventricular Fusion During Resetting and Entrainment of Orthodromic Supraventricular Tachycardia Involving Septal Accessory Pathways

Implications for the Differential Diagnosis With Atrioventricular Nodal Reentry

José M. Ormaetxe, MD; Jesús Almendral, MD; Angel Arenal, MD; Jesús D. Martínez-Alday, MD; Agustín Pastor, MD; Julián P. Villacastín, MD; Juan L. Delcán, MD

Background. Ventricular fusion during transient entrainment of orthodromic atrioventricular reciprocating tachycardias (OAVRT) was originally found to be absent and recently observed only with left ventricular stimulation. However, previous studies were restricted to cases with a left free wall accessory pathway. The hypothesis of the present study was that fusion is likely during resetting and entrainment of OAVRT with right ventricular stimulation if the accessory pathway is septally located, since its insertion is relatively close to the stimulation site. This phenomenon can help in the differential diagnosis with atrioventricular nodal reentry (AVNR).

Methods and Results. We performed programmed right ventricular stimulation during regular inducible supraventricular tachycardia with concentric atrial activation in 44 patients—20 with OAVRT and 24 with AVNR. Fusion in the ECG morphology of extrastimuli producing resetting was observed in 19 of 19 OAVRT but in 0 of 11 AVNR reset (P<.001). Transient entrainment was demonstrated in all 31 cases undergoing rapid ventricular pacing (14 OAVRT and 17 AVNR). Entrainment with fusion occurred in 13 of 14 OAVRT and in 0 of 17 AVNR (P<.001). Fusion was critically dependent on the coupling intervals or pacing rates, sometimes having a narrow window for its observation.

Conclusions. The relative proximity (conduction time) among pacing site, site of entrance to a reentrant circuit, and site of exit from the circuit to the paced chamber are critical for the occurrence of fusion during resetting and/or entrainment. The presence or absence of fusion during these phenomena can help in the differential diagnosis of certain supraventricular tachycardias. (Circulation. 1993;88:2623-2631.)

KEY WORDS • atrioventricular node • tachycardia

Transient entrainment and resetting have been described in a variety of supraventricular tachycardias. When transient entrainment of orthodromic atrioventricular reciprocating tachycardias (OAVRT) was first observed in response to ventricular stimulation, fusion was not found in the surface QRS complex. However, in this study stimulation was performed at the right ventricle, and all cases had a single left free wall accessory pathway. More recently, entrainment with fusion has been found in OAVRT incorporating a left free wall accessory pathway when stimulation was performed at the left ventricle, demonstrating that the presence of fusion was dependent on the location of the pacing site relative to the accessory pathway. In a study of resetting and entrainment of ventricular tachycardia, it was suggested that the presence of fusion was dependent on the proximity of the stimulating site to the entrance of the reentrant pathway. Accordingly, we postulated that in OAVRT with septally located pathways, resetting and entrainment of tachycardia in response to right ventricular apical stimulation would be likely to display fusion in the QRS complex, since the ventricular insertion of the accessory pathway in these cases is relatively close to the stimulation site. In fact, such a finding was shown in a simple case of OAVRT using an accessory pathway with long retrograde conduction times. If that were generally the case in OAVRT, this phenomenon could be of help in the differential diagnosis of supraventricular tachycardia.

The aims of this study were to analyze if ventricular fusion could be detected on the surface ECG during resetting and/or transient entrainment with right ventricular stimulation of OAVRT incorporating septally located accessory pathways and the extent to which the presence and characteristics of resetting and entrainment could contribute to the differential diagnosis of supraventricular tachycardia with concentric atrial activation.
## Electrophysiological Characteristics of the Tachycardias

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SVE indicates single right ventricular extrastimuli; RVP, rapid right ventricular pacing; SVT, supraventricular tachycardia; CI, cycle length; CFI, coupling interval; and PCL, pacing cycle length.

*Patients with long retrograde conduction time of the accessory pathway during the orthodromic reciprocating tachycardia; †patient with right anterior septal accessory pathway; ‡patient with the fast-slow form of atrioventricular nodal reentry.
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Methods

Study Patients
We studied 44 consecutive patients who underwent an electrophysiological examination and had an inducible sustained supraventricular tachycardia that met the following inclusion criteria: a septally initiated atrial depolarization during tachycardia (earliest atrial electrogram less than 1 cm away from the os of the coronary sinus as visually determined by fluoroscopy or at the His bundle electrogram position), a stable cycle length (±10-millisecond difference in the cycle length of 10 consecutive beats), and right apical programmed ventricular stimulation performed during tachycardia. Patients were excluded if more than one mechanism of tachycardia or accessory pathway were detected. Antiarrhythmic drugs were stopped at least five half-lives before the study, except in one patient who was taking 200 mg/day flecainide. Patients were divided into two groups according to their tachycardia mechanism, as determined by standard criteria.17-20

Group 1. Twenty patients (13 women; mean age, 36 years; age range, 7 to 68 years) with an OAVRT incorporating a septal or paraseptal accessory pathway. The specific location of the accessory pathways included 15 right posteroseptal, 2 right posterior paraseptal, 2 left posteroseptal, and 1 right anteroseptal accessory pathway. Nine cases had the Wolff-Parkinson-White syndrome and the remaining 11 had concealed atrioventricular bypass tract. Four of them presented long retrograde conduction times associated with the incessant form of supraventricular tachycardia.

Group 2. Twenty-four patients (19 women; mean age, 44 years; range, 30 to 60 years) with atrioventricular nodal reentrant (AVNR) tachycardia, one of them with the fast-slow form and an incessant clinical presentation.

No patient had detectable evidence of organic heart disease, except one group 1 patient who presented with an Ebstein anomaly. The QRS during SVT was narrow in all group 2 patients and in all except 3 group 1 patients, who had right bundle branch block–organic in 1 patient and functional in the remaining 2.

Electrical Stimulation and Recordings
With standard techniques, multipolar catheter electrodes (with 1- to 5-mm interelectrode distance) were placed in the high right atrium, His bundle position, right ventricular apex, and in all except 2 patients in whom it could not be catheterized (and an electrode was placed via femoral artery in the left ventricle), in the coronary sinus. Electrodes were used selectively to record electrograms and to pace the heart. Stimulation was performed using a programmable stimulator (Jansen Inc). Bipolar cathodal stimulation was performed at the distal pair of each multipolar catheter. Stimuli were rectangular pulses 1 millisecond in duration at twice-diastolic threshold. Data were recorded with an ink-jet recorder (Siemens Elema, Mingograph 4) or with a photographic recorder (Honeywell VR 12) at a paper speed of 100 mm/s. Three to seven surface ECG leads were recorded along with three to five bipolar intracardiac electrograms. All intracardiac electrograms were filtered at 30 to 500 Hz.

During spontaneous tachycardia or after its induction with standard cardiac pacing techniques, right apical ventricular extrastimuli were introduced in all patients, scanning diastole in 10-millisecond decrements beginning with a coupling interval 10 milliseconds less than the tachycardia cycle length until the tachycardia was interrupted or the ventricular effective refractory period was reached. Multiple synchronized trains of rapid ventricular pacing at a constant rate were delivered from the same site to overdrive the tachycardia in 31 patients (14 group 1 and 17 group 2 patients). The duration of each train was at least 20 seconds. The pacing cycle length was initially set at 5 to 10 milliseconds less than the tachycardia cycle length and subsequently decreased in 5- to 10-millisecond decrements until the tachycardia was interrupted. Once the patient was in sinus rhythm, to compare with the QRS morphology during resetting and entrainment, single ventricular extrastimuli and trains of rapid ventricular pacing were introduced at similar coupling intervals or cycle lengths.

Electrophysiological Diagnosis
The diagnosis of AVNR and OAVRT was based on established criteria.17-20 Of note were the following: (1) In all except one group 1 tachycardias, a premature ventricular extrastimulus delivered during His bundle refractoriness advanced atrial depolarization with the same activation sequence. (2) In 3 group 1 patients, a premature ventricular extrastimulus delivered during His bundle refractoriness and not conducted to the atria resulted in tachycardia termination. (3) In 23 group 2 tachycardias, atrial activation began before the end of the QRS complex and had an HA interval of less than 60 milliseconds; the remaining group 2 case had the fast-slow form of AVNR. (4) In this patient, the diagnosis was established by the absence of atrial advancement by extrastimuli delivered when the His was refractory; additional evidence was obtained since ablation attempts with DC current resulted in complete atrioventricular nodal block with maintenance of the tachycardia at a similar rate on the atrial level. (5) In 19 group 2 patients, a discontinuous atrioventricular nodal conduction curve was demonstrated. (6) In all cases, ventricular pacing eventually reproduced the atrial activation sequence observed during tachycardia, making the diagnosis of atrial tachycardia unlikely.

Definitions
Resetting of tachycardia by a single right apical extrastimulus is an advancement of the subsequent atrial depolarization in at least 20 milliseconds (10 milliseconds if totally regular rhythm), followed by continuation of the tachycardia. This definition was based on the concept of resetting in reentrant tachycardias being fulfilled as long as one necessary component of the circuit is advanced (the atria itself in OAVRT and the retrograde atrioventricular nodal pathway in AVNR expressed by the subsequent atrial depolarization) and was used because in some instances compensatory delays in the antegrade conduction over the atrioventricular node obscured the full expression of the resetting phenomenon.

Entrainment of tachycardia during right apical ventricular continuous pacing is an increase in atrial and ventricular rates to the pacing rate with resumption of the tachycardia at its intrinsic rate on cessation of pacing.
Resetting and entrainment zones were the ranges of coupling intervals of extrastimuli or cycle lengths of rapid pacing over which resetting or entrainment was observed.

Ventricular fusion was considered to be present when the QRS morphology in the surface ECG during resetting or entrainment of supraventricular tachycardia with right apical ventricular stimulation was different from the QRS morphology obtained during stimulation from the same site without supraventricular tachycardia (during sinus rhythm). The presence of different degrees of fusion at different paced cycle lengths or at different coupling intervals of the extrastimuli was defined as “progressive fusion.” One of the investigators reviewed the surface QRS recordings blindly and decided whether fusion was present or absent.

Statistical Analysis

The data are presented as mean±SD. Categorical variables were compared using the χ² test or Fisher’s exact test. Continuous variables were compared using an unpaired t test. A value of P<.05 was considered statistically significant. Sensitivity (Se) and specificity (Sp) were calculated following the usual equations.

Results

The tachycardia was reproducibly induced and terminated by programmed electrical stimulation in all patients. The relevant electrophysiological variables are depicted in the Table. Tachycardia cycle lengths ranged from 250 to 430 milliseconds (mean, 338±61 milliseconds) in group 1 and from 250 to 460 milliseconds (mean, 346±52 milliseconds) in group 2 (P=NS).

Resetting of tachycardia in response to single right apical ventricular extrastimuli was observed in all except one (95%) group 1 and in 11 of 24 (46%) group 2 tachycardias (P<.001, Table). The mean longest coupling interval of ventricular extrastimuli producing resetting of tachycardia was 284±59 milliseconds (84% of the tachycardia cycle length) in group 1 and 226±39 milliseconds (65% of the tachycardia cycle length) in the 11 group 2 tachycardias that were reset (P<.001, Table). The mean resetting zone was 49±26 milliseconds in group 1 and 34±15 milliseconds in group 2 (P=NS, Table).

Ventricular fusion was observed in the surface QRS complex of at least one extrastimulus producing resetting in all group 1 tachycardias (Table). Fig 1 is a representative example. In contrast, resetting with ventricular fusion was never observed in group 2 tachycardias (P<.001, Table and Fig 2). Therefore, the presence or absence of resetting with ventricular fusion distinguishes these two groups of tachycardias without overlapping (Fig 3, top). When ventricular fusion appeared, it always did so on the extrastimuli producing resetting with the longest coupling interval (always longer than 75% of the tachycardia cycle length), eventually disappearing as the coupling intervals of extrastimuli decreased (Table).

Transient entrainment of tachycardia in response to trains of continuous right ventricular stimulation was observed in all cases undergoing this mode of stimulation. In all instances, the slowest pacing cycle length attempted (5 to 10 milliseconds less than the tachycardia cycle length) produced entrainment (Table). No statistically significant differences were observed in the slowest pacing cycle length producing entrainment in both groups.
Fig 2. Resetting during atrioventricular nodal reentrant tachycardia. Panels are organized as in Fig 1, showing recordings during atrioventricular nodal reentrant tachycardia. On A through C, a right apical ventricular extra-stimulus (marked with an arrow) is introduced during tachycardia with progressively shorter coupling intervals, resulting in an advancement of the subsequent atrial depolarization in B and C. However, in this case, there are no differences between the QRS morphology of the extrastimuli of B and C, and both are identical to the QRS of the extrastimulus of A (not producing resetting). Moreover, all have the same morphology as the extrastimulus shown in D delivered in the absence of tachycardia. HRA indicates high right atrium; HBEp and HBed, proximal and distal His bundle electrograms; CSp and CSd, proximal and distal coronary sinus; RVA, right ventricular apex; and T, time lines.

Constant ventricular fusion was observed in the surface QRS complex of at least one pacing train producing entrainment in all except one group 1 tachycardia (Table). A representative example is shown in Fig 4. In contrast, in group 2 tachycardias, entrainment with ventricular fusion was never observed (Table, Fig 5). Therefore, the presence of ventricular fusion during transient entrainment has a 100% positive and a 92% negative predictive value for OAVRT (Fig 3, bottom). When ventricular fusion appeared, it always did so on the slowest pacing train, eventually tending to disappear as the pacing cycle length was decreased (Table, Fig 6).

Discussion
Fusion During Resetting and Entrainment of OAVRT With Right Ventricular Stimulation

Previous studies by Okumura et al and Suyama et al failed to observe ventricular fusion during transient entrainment of OAVRT with right ventricular stimulation. Okumura et al proposed that the absence of fusion was related to the fact that stimulation was performed distal to the area of slow conduction of the reentrant circuit (i.e., the atrioventricular node in OAVRT). In fact, the presence or absence of fusion during transient entrainment has been suggested as a means to localize the area of slow conduction of reentrant tachycardias. In the present study, with a "model" sharing with the former studies the area of slow conduction (the atrioventricular node), the stimulating site (right ventricular apex), and the path of conduction between both (orthodromic conduction), fusion in the QRS complex could be observed in all cases during resetting and in all except one case during transient entrainment. The difference between the previous series and the present study relates to the location of the accessory pathways: they were left sided in all the cases of the former series, whereas they were septal/paraseptal in all of our patients. This finding demonstrates that factors different from the proximity of the stimulating site to the area of slow conduction influence the ability to reset or entrain a tachycardia with fusion.

Rosenthal et al demonstrated in a group of patients with ventricular tachycardia in the setting of prior myocardial infarction and therefore thought to be due to reentry that ventricular fusion during resetting could be observed if pacing was performed at a site presumably located close to the entrance of the paced wavefronts on the reentrant circuit. Suyama et al and our own group observed fusion during entrainment of OAVRT incorporating a left free wall accessory pathway if stimulation was performed at the left ventricle. In these studies, stimulation was performed close to the site of entrance of the paced wave fronts in the reentrant circuit. The ventricular insertion of the accessory pathway. In the present series and in a previously reported case, the accessory pathways were also located closer to the stimulating site than in previous studies performing right ventricular stimulation because they were septal/paraseptal and therefore, as depicted schematically in Fig 7, closer to the right ventricular
ventricular train

Fig 4. Example of entrainment during orthodromic atrioventricular reciprocating tachycardia. The figure is organized in three panels, all showing the last few beats of a train of rapid ventricular pacing introduced during the tachycardia shown in Fig 1. Only three surface ECG leads and two intracardiac recordings are shown for simplicity. A and B, last beats of a train producing transient entrainment with resumption of tachycardia after cessation of pacing. Note a dramatic change in QRS morphology as a result of a slight change in pacing cycle length (from 245 milliseconds in A to 240 milliseconds in B), demonstrating ventricular fusion during entrainment. The change is even more notorious when compared with the pacing train resulting in tachycardia termination (C). Again, as during resetting, an important increase in QRS duration can be observed as expression of ventricular fusion when C is compared with A (150 versus 110 milliseconds). LRA indicates lateral right atrium and RVA, right ventricular apex.

A  

B  

C  

Absence of Fusion During Resetting and Entrainment of AVNR

Previous studies have demonstrated that rapid ventricular pacing can produce transient entrainment of AVNR. However, in these studies, neither atrial nor ventricular fusion beats were observed during transient entrainment. Our group 2 cases confirm these data, since transient entrainment could always be obtained in response to rapid ventricular pacing during AVNR tachycardias, but ventricular fusion was never observed in the QRS of paced beats producing resetting and/or entrainment of AVNR (Table). In light of our previous discussion regarding OAVRT, this could be considered surprising, since in AVNR the site of entrance of paced ventricular wave fronts in the reentrant circuit (His bundle–low atrioventricular nodal tissue) may be as far from the pacing site as in OAVRT (with septally located pathways). However, as described in the ventricular tachycardia model, another requirement for the presence of fusion (independent of the pacing site) is spatial separation between the sites of entrance to and exit from the reentrant circuit. This requirement cannot be met in AVNR. As depicted in Fig 7C, fusion during entrainment of AVNR would require that paced ventricular wave fronts enter the circuit propagating through the His bundle at the time that impulses are exiting through this same structure (the His bundle is also the site of exit of the tachycardia circuit to the ventricular tissue). Under such circumstances, constant fusion during entrainment is virtually impossible (unless a second connection exists between atria and ventricles).

Study Limitations

Analysis of both the resetting response and the presence of constant fusion during transient entrain-
ent requires stability in tachycardia cycle length. This may restrict the validity of our observations in case of oscillations or irregularity in the tachycardia cycle length. Likewise, this finding could hardly be used in nonsustained tachycardias.

Ventricular fusion was not observed in one of our cases during entrainment. The reason for this was that continuous pacing (even at a cycle length just below the tachycardia cycle length) produced slowing in atrioventricular nodal conduction, allowing the antidromic wave front to invade retrogradely the His bundle, so collision did not take place in ventricular tissue and therefore fusion was absent. Had pacing not been started with cycle lengths immediately below the tachycardia cycle length, fusion would have missed in more cases.

![Fig 6](image-url)  
**Fig 6.** Example of entrainment during orthodromic atrioventricular reciprocating tachycardia showing fusion in the QRS complex that tends to disappear at a critical pacing rate. Panels are organized as in Fig 4, all showing the last few beats of a train of rapid ventricular pacing introduced during tachycardia. Only two intracardiac recordings are shown for simplicity. A and B, last beats of a pacing train producing transient entrainment with resumption of tachycardia after cessation of pacing. C, A faster train that results in tachycardia termination. Note the change in QRS morphology comparing A and B (particularly in lead I) as a result of a change in pacing cycle length (from 370 milliseconds in A to 350 milliseconds in B). However, there is probably only a minor degree of fusion (QRS morphology resembling a "fully paced QRS complex") in B, since a further increase in pacing rate produces a quite similar QRS morphology (C) and results in tachycardia termination. LRA indicates lateral right atrium and RVA, right ventricular atrium.

![Fig 7](image-url)  
**Fig 7.** Schematic representation offering an explanation for the presence or absence of ventricular fusion in supraventricular tachycardias with a variety of mechanisms. In orthodromic atrioventricular reciprocating tachycardias with a septal accessory pathway (AP) (A), paced impulses (marked with *) can collide with the ongoing tachycardia wave front (empty arrows) after its ventricular breakthrough (VB), while propagation through the ventricles can engage the ventricular insertion of the accessory pathway (because of its relative proximity to the stimulation site) and consequently reset/entrain the tachycardia. In this situation, collision of wave fronts takes place within the ventricular tissue, producing fusion in the QRS complex. If the accessory pathway is left lateral (B) and thus located far from the stimulation site (the right ventricle), a paced wave front that engages the ventricular insertion of the accessory pathway will collide with the ongoing tachycardia wave front proximal to its VB. Consequently, ventricular fusion will be absent, as found in a previous study. In atrioventricular nodal reentry (C), the exit of the tachycardia circuit to the ventricles (His bundle and bundle branches) is the same as the entrance of any paced ventricular impulse into the circuit (provided there is no abnormal connection between ventricles and atria or atrioventricular node). Therefore, in atrioventricular nodal reentry, paced ventricular impulses that enter the circuit will collide with the ongoing tachycardia wave front proximal to the His bundle, thus precluding exit to the ventricles and ventricular fusion. RV indicates right ventricle and LV, left ventricle.

**Other Differences in Resetting and Entrainment of OAVRT and AVNR: Practical Implications of Fusion During Resetting/Entrainment**

It has been previously noted that resetting with single extrastimuli is easier in OAVRT (particularly with septally located pathways) than in AVNR. We also found that resetting was more frequent in OAVRT than in AVNR; however, the incidence of resetting in AVNR approached 50%, higher than in previous studies. Miles et al. noted that the degree of prematurity necessary to advance the atria with ventricular extra-
stimuli delivered during supraventricular tachycardia, the so-called preexcitation index, could distinguish OAVRT from AVNR. Our findings are in complete agreement with this study, since the longest coupling intervals of extrastimuli resetting OAVRT were longer than those resetting AVNR. More recently, a similar index was derived for entrainment (entrainment index) but only studied for OAVRT. The advantage of the entrainment index, in essence similar to the preexcitation index, is its more general application, even in OAVRT, in which the preexcitation index could not be calculated. Our finding that entrainment could always be produced in AVNR suggests that the entrainment index could be used for the differential diagnosis of SVT in a more general fashion.

In view of our findings, the presence or absence of ventricular fusion during resetting or entrainment can be considered among the electrophysiological phenomena useful in the differential diagnosis of supraventricular tachycardia with concentric atrial activation. While sharing with the other described indexes the limitations related to the requirement of a sustained and stable tachycardia, it has its own advantages. First, it can be observed in most instances during rapid ventricular pacing, thus avoiding the limitation appearing when single extrastimuli do not advance the atria (we observed that in one OAVRT and Miles et al observed in 2 cases of OAVRT with posteroseptal pathways). Second, it avoids difficulties in reproducibility of measurements; some authors claim different values for the preexcitation index, and this could be related to factors such as the interelectrode distance of the catheters or the way in which intervals are measured (to the stimulus artifact or to the local electrogram). Third, it can be easier to obtain during the study, since it does not require measurements; this can speed up the diagnostic workup, which is particularly critical in the present ablative “era,” when diagnostic and therapeutic procedures tend to be performed in one session.

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