Transient Entrainment and Interruption of the Atrioventricular Bypass Pathway Type of Paroxysmal Atrial Tachycardia

A Model for Understanding and Identifying Reentrant Arrhythmias

ALBERT L. WALDO, M.D., VANCE J. PLUMB, M.D., JOAQUIN G. ARCINIEGAS, M.D., WILLIAM A. H. MACLEAN, M.D., TERRY B. COOPER, M.D., MARSHALL F. PRIEST, M.D., AND THOMAS N. JAMES, M.D.

SUMMARY We studied transient entrainment and interruption of atrioventricular (AV) bypass pathway–type paroxysmal atrial tachycardia in 15 patients during overdrive pacing from selected atrial sites. Overdrive atrial pacing at less than a critically rapid rate for interruption transiently entrained the tachycardia. Transient entrainment was due to repeated early entrance of the wave front from the pacing impulse into the reentry loop in both antidromic and orthodromic directions. The antidromic wave front of each pacing impulse was repeatedly blocked as it collided with the orthodromic wave front of the previous beat, in effect extinguishing the tachycardia. However, the early entrance of the orthodromic wave front of each pacing impulse repeatedly reset the tachycardia. The result was that during transient entrainment, the tachycardia rate increased to the pacing rate. Interruption of the tachycardia occurred when overdrive pacing produced block within the reentry loop of both the antidromic and orthodromic wave fronts of the same pacing impulse, the block occurring either at separate sites within the reentry loop or at the same site. Atrial fusion beats were demonstrated during transient entrainment in nine patients and resulted from intraatrial collision of the antidromic wave front from the pacing impulse with the orthodromic wave front of the previous beat. The presence of fusion beats depended critically on the relationship of the pacing site to the reentry loop and the duration of conduction around the reentry loop, particularly through the area of slow conduction.

The data from this study suggest that (1) if one can demonstrate constant fusion beats during transient entrainment of a tachyarrhythmia except for the last transiently entrained beat; or (2) if during transient entrainment of a tachyarrhythmia at two or more different pacing rates, one can demonstrate constant fusion at each of the different pacing rates, but different degrees of fusion at the different rates; or (3) if interruption of a tachyarrhythmia by overdrive pacing is associated with localized conduction block to a site followed by activation of that site by the next pacing impulse from a different direction and with a shorter conduction time, then the underlying mechanism of the arrhythmia can be best explained by reentry.

Paroxysmal atrial tachycardia that involves antegrade conduction from the atria through the atrioventricular (AV) node–His-Purkinje system to the ventricles with retrograde conduction from the ventricles via an AV bypass pathway back to the atria is the best understood example of putative reentrant rhythms.¹⁻⁴ This arrhythmia is often found in patients with the Wolff-Parkinson-White syndrome and in patients with a so-called concealed AV bypass pathway, i.e., one that conducts only in the retrograde direction. The many types of treatment for this arrhythmia include its interruption by overdrive cardiac pacing.⁵ We have found that overdrive pacing used to interrupt paroxysmal atrial tachycardia of this variety has much in common with our earlier descriptions of overdrive pacing to interrupt classic (type I) atrial flutter,⁶⁻⁹ ventricular tachycardia,⁷,⁹,¹⁰,¹¹ and ectopic atrial tachycardia,⁸,⁹,¹² i.e., at overdrive pacing rates slower than a critically rapid rate, it demonstrates transient entrainment.

We have defined transient entrainment of a tachycardia with overdrive pacing as an increase in the rate of all tissue of the cardiac chamber being paced to the pacing rate (which is faster than the intrinsic rate of the tachycardia), with resumption of the intrinsic rate of the tachycardia upon either abrupt cessation of pacing or slowing of the pacing rate below the intrinsic rate of the tachycardia.¹⁰ In this report, we both demonstrate and identify the mechanism of transient entrainment and interruption of the AV bypass pathway type of paroxysmal atrial tachycardia. We believe that these observations can help explain transient entrainment and interruption of other tachyarrhythmias with overdrive pacing, and provide evidence that reentry is the underlying mechanism of the tachyarrhythmia.

Methods

Each patient gave informed consent. Using standard cardiac electrophysiologic techniques during cardiac catheterization, we studied transient entrainment and interruption of AV bypass pathway–type paroxysmal atrial tachycardia in 15 patients. Twelve were males and three females, ages 8–65 years (mean 36 years). By standard criteria,¹⁻⁴ 11 patients had Wolff-Parkinson-White syndrome (seven type A and four type B)
and four patients had a so-called retrograde concealed AV bypass pathway. At the time of study, no patient was receiving cardioactive drugs. All patients had a history of paroxysmal atrial tachycardia. In each instance, the study was part of a larger clinical study to define the nature of each patient's spontaneous arrhythmia and to find an effective treatment.1-4, 13-15

Using standard techniques,1-4, 13-16 catheter electrodes were routinely placed in the high right atrium. His bundle position, right ventricle and, in all but two patients, in the coronary sinus, and were used selectively to record electrograms and to pace the heart. In eight patients, an additional catheter was placed in the low lateral right atrium, in one patient, a catheter electrode was placed in the inferior left atrium through a patent foramen ovale, and in another patient, a catheter electrode was also placed in the right pulmonary artery to record activation from the superior left atrium.17 In all instances, at least one ECG (lead II) was recorded simultaneously along with electrograms from all recording sites. Whenever possible, ECG leads I, III, and V1 were also recorded. All electrograms were recorded between a bandpass of 12-500 Hz except the His bundle electrogram, which was recorded between a bandpass of 40-500 Hz. All ECGs were recorded between a bandpass of 0.1-50 Hz or 0.1-500 Hz.

After the paroxysmal atrial tachycardia was initiated with standard cardiac pacing techniques, rapid atrial pacing was used to overdrive the tachycardia. Pacing was performed from the high right atrium and coronary sinus in eight patients, from the low right atrium and coronary sinus in one patient, only from the high right atrium in three patients, only from the inferior left atrium in one patient, only from the coronary sinus in one patient, and only from the low right atrium in one patient. Atrial pacing with a Medtronic 1349A programmable stimulator was initiated at a rate of about 5-10 beats/min faster than the spontaneous rate, continued for at least 5 seconds, and then terminated abruptly. If the tachycardia was not interrupted, atrial pacing from the same site was again initiated, but with an increment in rate of 5-10 beats/min. This procedure was repeated until the tachycardia was interrupted. This pacing procedure varied somewhat from patient to patient, as determined by the clinical status and requirements during each study. If second-degree AV block developed during pacing, termination of pacing was attempted after a nonconducted atrially paced beat. This was occasionally difficult, especially in the presence of relatively long Wenckebach cycles. In such instances, the pacing rate was increased by an additional 5-10 beats/min to get a higher degree of second-degree AV block, making termination of pacing after an AV nodally blocked beat easier. The lowest atrial pacing rate that produced second-degree AV nodal block was considered to be the rate that interrupted the tachycardia.

All electrograms and ECGs were recorded simultaneously on photographic paper using an Electronics for Medicine DR-12 switched-beam oscilloscopic recorder. The data also were recorded simultaneously on a Honeywell 5600 FM tape recorder for subsequent playback and analysis. Paper recording speeds varied from 25 to 200 mm/sec. All measurements were made from data recorded at a speed of at least 100 mm/sec.

**Results**

A critically rapid atrial pacing rate was required to interrupt the paroxysmal atrial tachycardia. This rate ranged from 15-63 beats/min (mean 30 beats/min) faster than the spontaneous rate of the tachycardia (table 1), and was always associated with the development of bidirectional block within the reentry loop, a requisite for interruption of a reentrant rhythm by cardiac pacing.

Overdrive atrial pacing at rates slower than a critically rapid rate only transiently entrained the tachycardia. During transient entrainment, each pacing impulse was shown to enter and preexcite the reentry loop antidromically (i.e., in the opposite direction of reentrant activation) and orthodromically (i.e., in the normal direction of reentrant activation) (fig. 1). Each antidromic wave front produced by the pacing impulse collided with the orthodromic wave front of each preceding beat coming around in the reentry loop, which, in effect, thereby repeatedly interrupted the tachycardia. Each orthodromic wave front produced by the pacing impulse resulted in early orthodromic excitation of the reentry loop, repeatedly resetting the tachycardia with a consequent increase in the rate of the tachycardia to the pacing rate. Thus, during transient entrainment, each pacing stimulus terminated the tachycardia antidromically, but reset the tachycardia orthodromically. The following representative study illustrates and explains these observations.

Figure 2A demonstrates ECG lead II recorded simultaneously with electrograms from selected sites

<table>
<thead>
<tr>
<th>Pt</th>
<th>Spontaneous rate (beats/min)</th>
<th>Pacing rate (beats/min)</th>
<th>Difference (beats/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>200</td>
<td>235</td>
<td>35</td>
</tr>
<tr>
<td>2</td>
<td>167</td>
<td>188</td>
<td>21</td>
</tr>
<tr>
<td>3</td>
<td>207</td>
<td>225</td>
<td>18</td>
</tr>
<tr>
<td>4</td>
<td>235</td>
<td>275</td>
<td>40</td>
</tr>
<tr>
<td>5</td>
<td>158</td>
<td>176</td>
<td>18</td>
</tr>
<tr>
<td>6</td>
<td>152</td>
<td>189</td>
<td>37</td>
</tr>
<tr>
<td>7</td>
<td>185</td>
<td>222</td>
<td>37</td>
</tr>
<tr>
<td>8</td>
<td>177</td>
<td>215</td>
<td>38</td>
</tr>
<tr>
<td>9</td>
<td>207</td>
<td>222</td>
<td>15</td>
</tr>
<tr>
<td>10</td>
<td>170</td>
<td>190</td>
<td>20</td>
</tr>
<tr>
<td>11</td>
<td>167</td>
<td>230</td>
<td>63</td>
</tr>
<tr>
<td>12</td>
<td>192</td>
<td>214</td>
<td>22</td>
</tr>
<tr>
<td>13</td>
<td>200</td>
<td>230</td>
<td>30</td>
</tr>
<tr>
<td>14</td>
<td>176</td>
<td>210</td>
<td>34</td>
</tr>
<tr>
<td>15</td>
<td>214</td>
<td>240</td>
<td>26</td>
</tr>
<tr>
<td>Mean</td>
<td>187</td>
<td>217</td>
<td>30</td>
</tr>
</tbody>
</table>

Table 1. Comparison of the Spontaneous Rate of the Paroxysmal Atrial Tachycardia with the Overdrive Atrial Pacing Rate Required
(left) The reentry loop during spontaneous atrioventricular (AV) bypass pathway–type paroxysmal atrial tachycardia. The X represents the orthodromic wave front of the reentrant rhythm. (middle) The introduction of the first pacing impulse (X + 1) during overdrive pacing from the high right atrium during the paroxysmal atrial tachycardia. The large arrow indicates the pacing impulse entering into the reentry loop, whereupon it is conducted orthodromically (ORTHO) and antidromically (ANTI). The antidromic wave front from the pacing impulse (X + 1) collides with the orthodromic wave front of the previous spontaneous beat (X). (right) The introduction of the second pacing impulse (X + 2) during overdrive pacing from the high right atrium during the atrial tachycardia. The large arrow again indicates the pacing impulse entering into the reentrant loop, whereupon it is conducted orthodromically and antidromically. This next antidromic wave front (X + 2) collides with the orthodromic wave front of the previous paced beat (X + 1). The arrows indicate the direction of spread of the impulse, and the serpentine line indicates slow conduction in the AV node. A = atria; V = ventricles. See text for discussion.

ECG lead II recorded simultaneously with bipolar electrograms from the proximal pair of electrodes from the electrode catheter placed in the high right atrium (HRAp), the coronary sinus (CS), the low lateral right atrium (LRA), the distal pair (HBd) and proximal pair (HBp) of electrodes of the tripolar catheter placed in the His bundle position during a period of atrioventricular bypass pathway–type paroxysmal atrial tachycardia at a 339-msec cycle length (rate of 177 beats/min) (A), and during a period of overdrive pacing from the distal pair of electrodes from the high right atrial electrode catheter at a 308-msec cycle length (rate of 195 beats/min) (B). Time lines are at 1-second intervals. V = ventricular potential; A = atrial potential; H = His bundle potential; S = stimulus artifact. See text for discussion.
during an episode of paroxysmal atrial tachycardia at a rate of 177 beats/min in a patient with type A Wolff-Parkinson-White syndrome. Using a tripolar His bundle catheter, we recorded two potentials: one from the distal His bundle, which gave an excellent His bundle recording but a poor atrial electrogram, and one from the proximal pair of electrodes, which provided a satisfactory atrial complex when recorded at low gain. The sequence of cardiac activation during this patient’s tachycardia is illustrated in figure 1. The activation wave front passes orthodromically from the atria to the AV node where conduction is slow (AH interval = 152 msec), and then through the His-Purkinje system to the ventricles. Activation then proceeds from the ventricles retrogradely to the atria through the left-sided AV bypass pathway, so that the earliest atrial activation is in the region of the coronary sinus recording site, confirming the presence of a left-sided AV bypass pathway.

In this patient, rapid atrial pacing from the distal pair of the high right atrial electrode catheter at rates of 180, 185, and 190 beats/min transiently entrained the tachycardia. Figure 2B shows transient entrainment during pacing at 195 beats/min. During pacing at this rate, the beat-to-beat cycle length recorded at all atrial sites is identical to that for the pacing rate, fulfilling one criterion for transient entrainment: that during overdrive pacing, there is an increase in the rate of all tissue of the cardiac chamber being paced to the pacing rate. The AV nodal conduction time has prolonged to 207 msec. Figure 3A shows cessation of overdrive pacing at 195 beats/min, with prompt resumption of the paroxysmal atrial tachycardia at its previous spontaneous rate, fulfilling the other criterion for transient entrainment: that with abrupt cessation of pacing, the tachycardia resumes at its intrinsic rate. The last entrained atrial beat (asterisk) at the high right atrial site, the low lateral right atrial site, and the His bundle site immediately follows the last stimulus artifact, but the last entrained beat at the coronary sinus site is one cycle length beyond the last stimulus artifact. This interpretation is supported by two points. First, with cessation of pacing, there is a long pause at a cycle length that clearly exceeds the pacing cycle length at all recording sites except the coronary sinus site, where the cycle length one beat beyond the last pacing cycle is identical to the pacing cycle length. Not until the subsequent cycle does the coronary sinus cycle length exceed the pacing cycle length. Second, the morphology of the atrial complexes recorded at all sites during pacing is different from that during paroxysmal atrial tachycardia except at the coronary sinus site, where it is the same. This indicates that the coronary sinus site is being activated by a wave front coming from the same direction during pacing as during the spontaneous arrhythmia, whereas the other atrial recording sites are being activated from a different direction during pacing than during the spontaneous arrhythmia. Thus,

![Figure 3](http://circ.ahajournals.org/)

**Figure 3.** ECG lead II recorded simultaneously with electrograms from the same recording sites in the same patient as in figure 2. Termination of overdrive pacing of the tachycardia from the distal pair of electrodes from the high right atrial electrode catheter is shown both for a 308-msec cycle length (195 beats/min) (A) and 292-msec cycle length (rate of 205 beats/min) (B). In the recordings from the coronary sinus site, the arrows from each stimulus artifact indicate the atrial complexes which result from that stimulus. The asterisk indicates the last transiently entrained beat at each recording site. S = stimulus artifact. P_f = fusion P wave. See text for discussion.
both during spontaneous tachycardia (fig. 2A) and during pacing from the high right atrium (figs. 2B and 3A), the coronary sinus site is being activated in a retrograde direction via the AV bypass pathway, i.e., orthodromically in the reentry loop.

During the period of pacing from the high right atrium, there is fusion of atrial activation, part of the atria being activated in a retrograde direction via the AV bypass pathway and part activated directly by a wave front from the pacing site. However, with the last entrained beat in the region of the coronary sinus (third beat in fig. 3A), there is no longer atrial fusion, as the normal sequence of activation during the spontaneous tachycardia resumes. The absence of atrial fusion during the last entrained beat is further confirmed in the ECG: positive P waves immediately follow each QRS complex in figure 2B and the first and second QRS complexes in figure 3A, resulting from fusion of atrial activation, but disappear after the third QRS complex in figure 3A because the latter P wave, although entrained, results from retrograde atrial activation. Thus, figure 3A illustrates transient entrainment of paroxysmal atrial tachycardia that is associated with atrial fusion except for the last entrained beat. It also illustrates that a long pause recorded at several atrial sites immediately after cessation of atrial pacing does not reflect overdrive suppression. Furthermore, the presence of atrial fusion during transient entrainment clearly distinguishes transient entrainment from simple overdrive pacing of the heart.

Figure 4 illustrates the cardiac activation sequence consistent with the data during high right atrial pacing at a rate of 195 beats/min. The wave front produced by each pacing impulse enters and preexcites the reentry loop orthodromically, but also enters and preexcites the reentry loop antidromically. The antidromic wave front from the pacing impulse collides in the atria with the orthodromic wave front of the preceding beat, resulting in extinction of the reentrant wave front and producing fusion of atrial activation. However, the pacing impulse that enters and preexcites the reentry loop in the orthodromic direction continues, and, in fact, resets the tachycardia. This sequence is repeated with each pacing impulse, with a consequent increase in the rate of the paroxysmal atrial tachycardia to the pacing rate.

Figure 4 also illustrates the cardiac activation sequence consistent with the data after abrupt cessation of pacing at 195 beats/min. The last pacing impulse enters the reentry loop antidromically and orthodromically, as just described. However, this time, when the orthodromic impulse reenters the atria in a retrograde direction, it is unopposed by any antidromic wave front because pacing has ceased. This unopposed orthodromic wave front then continues the paroxysmal atrial tachycardia, but now at its spontaneous rate. Also, because there is no antidromic wave front, there is no atrial fusion associated with this last orthodromically entrained beat. We believe that these observations — the demonstration of fusion during transient entrainment except during the last entrained beat when there is no fusion — may be a hallmark for establishing reentrant rhythms.

Figure 3B illustrates cessation of rapid atrial pacing at a rate of 205 beats/min, followed once again by resumption of the paroxysmal atrial tachycardia. An asterisk denotes the last entrained beat at each site. During pacing at this still faster rate, the coronary sinus is now activated in an antidromic direction. This is apparent from two observations. First, the morphology of the atrial complex from the coronary sinus, although superimposed on the ventricular complex, is clearly different from that seen during retrograde activation of this site. In fact, it is the same as that seen during overdrive pacing of sinus rhythm when pacing from the same high right atrial site and, parenthetically, makes transient entrainment during atrial pacing at this rate virtually indistinguishable from simple overdrive capture of the heart. Second, the cycle length one beat beyond the last paced beat is longer than the pacing cycle length. However, because the last paced beat is conducted orthodromically to the ventricles and then retrogradely to the atria via the AV bypass pathway, it also activates the atria in the region of the

![Figure 3A](http://circ.ahajournals.org/)

*Figure 3A.* By guest on September 15, 2017
coronary sinus orthodromically, with consequent continuation of the paroxysmal atrial tachycardia. As in figure 3A, the long pause at the several atrial recording sites, this time including the coronary sinus site, that immediately follows cessation of rapid atrial pacing mimics, but does not reflect, overdrive suppression. The change from orthodromic activation of the coronary sinus at a pacing rate of 195 beats/min to antidromic activation at a rate of 205 beats/min is associated with an increase in the AH interval from 207 msec (fig. 3A) to 264 msec (fig. 3B), i.e., is associated with a further prolongation of conduction time through the area of slow conduction within the reentry loop.

Figure 5 illustrates the cardiac activation sequence consistent with the data during atrial pacing at 205 beats/min. Because at this faster pacing rate the pacing cycle length is shorter than before, the wave front from each pacing impulse enters the reentry loop even earlier, and AV nodal conduction time is even longer. (As noted in figure 3, the AH interval increased by 57 msec when the pacing rate was increased from 195 to 205 beats/min.) These factors permit the antidromic wave front of each pacing impulse to reach the region of the coronary sinus before the orthodromic wave front of the previous beat. Because the antidromic wave front from the pacing impulse \((X + 1)\) prevents the orthodromic wave front of the previous beat \((X)\) from activating the atria, there is no longer atrial fusion during rapid atrial pacing. However, as shown in figure 5, with cessation of pacing, the orthodromic wave front from the last paced beat comes around the reentry loop, but this time is unopposed when it reaches the AV bypass pathway. The result is retrograde conduction to the atria with continuation of the paroxysmal atrial tachycardia.

Figure 6 shows cessation of pacing at a rate of 225 beats/min. The last entrained beat at each recording site is indicated by an asterisk. The coronary sinus site is activated only in an antidromic direction, because this time the last paced atrial beat is not conducted to the ventricles. With the last paced beat not only blocked antidromically in the region of the AV bypass pathway but also blocked orthodromically in the AV node, the paroxysmal atrial tachycardia is interrupted. In fact, it is necessary to achieve block of both the antidromic and orthodromic wave fronts from the same pacing impulse within the reentry loop in order to interrupt this reentrant tachycardia. Had the pacing been stopped after an atrial beat that had not been blocked in the orthodromic direction, the tachycardia would have resumed. This is illustrated in figure 7, which shows cessation of atrial pacing at the same rate of 225 beats/min. The orthodromic wave front from the last paced beat is not blocked, with consequent resumption of the paroxysmal atrial tachycardia. In this patient, overdrive atrial pacing of the paroxysmal atrial tachycardia at a rate of 215 beats/min resulted in second-degree AV block and, as expected, if pacing was stopped after an atrial beat that was blocked orthodromically in the AV node, the tachyarrhythmia was interrupted.

**Fusion During Transient Entrainment**

Atrial fusion occurred in nine patients and always resulted from intraatrial collision of the antidromic wave front of the pacing impulse with the orthodromic wave front from the preceding beat. The atrial fusion was critically related to conduction time from the atrial pacing site to the atrial recording sites and to slow conduction within the reentry loop, i.e., to slow conduction in the AV node (figs. 3–5).

Some additional observations regarding fusion during transient entrainment of AV bypass pathway paroxysmal atrial tachycardia should be noted. First, only atrial fusion was encountered. In the four patients with AV bypass pathways capable of retrograde conduction...
only, this is not surprising. However, one might expect to see some examples of ventricular fusion during transient entrainment in patients with the Wolff-Parkinson-White syndrome, particularly those whose AV bypass pathways were capable of conducting impulses from the atria to the ventricles at a very rapid rate. No such ventricular fusion was observed during our studies, including three of our patients with left sided AV bypass pathways who demonstrated 1:1 AV conduction over the AV bypass pathway during overdrive pacing of sinus rhythm at rates of 240 beats/min or more. The lack of ventricular fusion was attributable in all but the latter three patients to antegrade refractoriness of the AV bypass pathway due either to an intrinsic property of the AV bypass pathway or to retrograde conduction from the orthodromic wave front of each preceding beat, the latter being either manifest (demonstrated by intraatrial fusion) or concealed. The absence of ventricular fusion in the three patients whose AV bypass pathway was capable of very rapid antegrade AV conduction is explained by the development of second-degree AV nodal block. Second-degree AV nodal block always occurred before an overdrive atrial pacing rate was reached that was rapid enough to permit the antidromic wave front to be conducted through the AV bypass pathway to the ventricles before the orthodromic wave front from the preceding beat could prevent it. Since the development of second-degree AV nodal block in these patients always resulted in interruption of the paroxysmal atrial tachycardia (fig. 8), no ventricular fusion beats were seen during transient entrainment in these three patients.

Atrial fusion was seen only in patients with a left-sided AV bypass pathway, and then only during pacing from the high right atrium. This was due to a combination of factors, but primarily AV nodal conduction time during the period of atrial pacing, and conduction time from the atrial pacing site to the various atrial portions of the reentry loop (fig. 3). Thus, the AV nodal conduction time was too long and conduction time from the selected atrial pacing sites to the atrial portion of the reentry loop too short to permit atrial fusion during transient entrainment of right-sided AV bypass pathway type of paroxysmal atrial tachycardia.

**Modes of Interruption of the AV Bypass Pathway Type of Paroxysmal Atrial Tachycardia**

Interruption of this arrhythmia by overdrive pacing was always associated with the development of block of both the antidromic and orthodromic wave fronts from the same pacing impulse within the reentry loop. Two types of such block were seen. Three patients had block at the AV bypass pathway of both the antidromic and orthodromic wave fronts from the same pacing impulse. The antegrade block of the antidromic wave front was manifest by a normal, narrow QRS complex during the atrial pacing. The retrograde block of the orthodromic wave front was manifest by failure of retrograde atrial activation after termination of atrial pacing although a normal, narrow QRS complex did

**Figure 6.** ECG lead II recorded simultaneously with electrograms from the same recording sites in the same patient as in figure 2 at the termination of overdrive pacing of the paroxysmal atrial tachycardia from the distal pair of electrodes from the high right atrial electrode catheter at a 267-msec cycle length (rate of 225 beats/min). Upon termination of pacing, the tachycardia is interrupted. The asterisk indicates the last transiently entrained beat at each recording site. S = stimulus artifact. Time lines are at 1-second intervals. See text for discussion.
follow the orthodromic wave front of the last atrial pacing impulse.

The other and most frequent type (12 patients) was the development of block of the antidromic and orthodromic wave fronts of the same pacing impulse at two separate sites: block of the antidromic wave front at or in the AV bypass pathway and block of the orthodromic wave front at or in the AV node. There were two subsets of this type. In one subset, consisting of nine patients, it was critical to terminate atrial pacing immediately after block of the orthodromic wave front in the AV node, or the tachycardia would not be interrupted (figs. 6 and 7). This is because the orthodromic wave front from the next pacing impulse simply restarted the tachycardia. In the other subset, consisting of three patients, termination of pacing at any time after block of the orthodromic wave front in the AV node resulted in successful interruption of the tachycardia (fig. 8).

Several important points become apparent upon analysis of the latter subset. After the development of block of the antidromic and orthodromic wave fronts from the same pacing impulse as just described above, the antidromic wave front of the next pacing impulse was now free to conduct to the ventricles via the AV bypass pathway (fig. 8). This was because the orthodromic wave front from the previous beat that had been colliding with the antidromic wave front of the pacing impulse was no longer present (it had been blocked in the AV node), and confirms our explanation that during the period of transient entrainment, the antidromic wave front of each pacing impulse collided with the orthodromic wave front of the preceding beat. Furthermore, for all subsequent pacing impulses, antegrade conduction to the ventricles via the AV bypass pathway continued (fig. 8), resulting in continued block of the antidromic and orthodromic wave fronts of the same pacing impulse, either in the ventricles or within the specialized AV conduction system. Thus, for this subset, as soon as second degree AV block developed, the tachycardia remained interrupted, even if antegrade conduction subsequently occurred over the AV node during pacing at the same rate.

Figure 8 illustrates another point that we believe may become a marker of reentry: the demonstration that interruption of the tachycardia is associated with localized block of one beat of the pacing impulse to a site (in this case the ventricles) followed by activation of that site by the next pacing impulse, but from a different direction than previously (in this case via the AV bypass pathway) and with a shorter conduction time than previously (in this case, 152 msec decreased from 379 msec).

**Discussion**

These data demonstrate that during transient en-
transient of AV bypass pathway–type paroxysmal atrial tachycardia, the pacing impulse enters and preexcites the reentry loop in both an orthodromic and an antidromic direction. Each antidromic wave front collides with the orthodromic wave front from the previous beat, in effect extinguishing the tachycardia, but each orthodromic wave front from the pacing impulse continues the tachycardia and, in fact, resets it to the pacing rate. Therefore, transient entrainment of AV bypass pathway–type paroxysmal atrial tachycardia is a repeated stopping and resetting of the tachycardia, which results in an increase of the tachycardia rate to the overdrive pacing rate. Interruption of the tachycardia with overdrive pacing requires production of block of both the antidromic and orthodromic wave fronts of the same pacing impulse within the reentry loop. Therefore, a critically rapid pacing rate must be achieved that will produce this type of block. Other-

Figure 8. ECG lead V1 recorded with bipolar electrograms from selected sites at the termination of overdrive pacing of paroxysmal atrial tachycardia (spontaneous cycle length 319 msec, rate 188 beats/min) from the distal pair of electrodes from the high right atrial electrode catheter (HRAh) at a 270-msec cycle length (rate 222 beats/min). The electrode recording sites include the His bundle position (HB), the proximal pair of the electrode catheter placed in the high right atrium (HRAh), and the distal (CSd) and proximal (CSp) pairs of the electrode catheter placed in the coronary sinus. The arrows from each stimulus artifact in the HRAh tracing point to the atrial and ventricular complexes in the His bundle electrogram that results from that stimulus. Although the interval from the stimulus artifact to the low atrial electrogram remains constant, the interval to the onset of ventricular activation (measured from the stimulus artifact to the onset of the QRS complex in the ECG) prolongs with each beat until atrioventricular (AV) block occurs after the sixth beat (star). The seventh and subsequent paced beats are again conducted to the ventricles, but primarily antegrade over the AV bypass pathway; the last three QRS complexes are probably entirely the result of that conduction. Upon termination of pacing, sinus rhythm resumes. Note that the time line partially obscures the of the QRS complex of the seventh beat. In the recordings from both coronary sinus sites, the arrows from each stimulus artifact point to an atrial complex that results from that stimulus. The first conduction interval from the stimulus artifact to atrial activation at each coronary sinus site is long, and it prolongs with the next stimulus. Both of these intervals reflect activation of the coronary sinus region by the orthodromic wave front of the pacing impulse, and indicate the presence of atrial fusion. The prolongation of this interval (stimulus to coronary sinus) results from prolongation of conduction of the orthodromic wave front of the pacing impulse through the area of slow conduction (the AV node). The orthodromic wave front of the third pacing impulse fails to activate the atrial tissue at this recording site (indicated by the blocked arrows). This is because AV nodal conduction became so prolonged that the antidromic wave front of the fourth pacing impulse reached the atrial tissue at the coronary sinus sites earlier than the orthodromic wave front of the third pacing impulse. This change in activation sequence is also reflected by a clear change in the morphology of the atrial complex recorded at this site. Subsequent pacing impulses continue to activate the atrial tissue at the coronary sinus site from the same “antidromic” direction. S = stimulus artifact. Time lines are at 1-second intervals. All numbers indicate msec. See text for discussion.
wise, overdrive pacing will simply transiently entrain the paroxysmal atrial tachycardia.

**Explanation of Events During Transient Entrainment and Interruption of Other Tachyarrhythmias**

We have previously demonstrated transient entrainment and interruption of classic (type I) atrial flutter and some types of ventricular tachycardia and ectopic atrial tachycardia.\(^5\)\(^-\)\(^12\) Although these other arrhythmias differ from AV bypass pathway—type tachycardia, the basic nature of transient entrainment and interruption of these tachyarrhythmias by overdrive cardiac pacing is probably similar. Thus, extrapolation from the observations of the present study to those during transient entrainment and interruption of other tachyarrhythmias helps to explain several observations:

1. The critically rapid pacing rate required for overdrive interruption of type I atrial flutter and for some types of ventricular tachycardia and ectopic atrial tachycardia\(^5\)\(^-\)\(^9\)\(^,\)\(^20\)\(^,\)\(^21\) is apparently required to achieve block of both the antiodromic and orthodromic wave fronts of the same pacing impulse within the reentry loop. (2) The critical duration of pacing required at the critically rapid pacing rate, particularly evident for interruption of type I atrial flutter,\(^5\)\(^-\)\(^9\)\(^,\)\(^20\)\(^,\)\(^21\) appears to be explained in part by the time required to develop block of the orthodromic wave front from the pacing impulse (fig. 8). (3) The localized conduction block to the inferior left atrium of the orthodromic wave front of one pacing impulse with the subsequent activation of this site from a different direction and with a shorter conduction time and with the appearance of a positive P wave in ECG lead II, all associated with the interruption of type I atrial flutter during overdrive pacing from the high right atrium,\(^5\)\(^-\)\(^9\)\(^,\)\(^20\)\(^,\)\(^21\) appears to be explained by the example of the interruption of the tachycardia shown in figure 8. (4) The presence of fusion beats during transient entrainment of other tachyarrhythmias\(^5\)\(^-\)\(^12\)\(^,\)\(^20\)\(^-\)\(^23\) is explained by the collision of the antiodromic wave front from the pacing impulse with the orthodromic wave front of the preceding beat. Although the degree of fusion at each pacing rate is constant, providing conduction times during pacing are constant, different degrees of fusion at different overdrive pacing rates, i.e., progressive fusion, are explained by differences in conduction times during the reentry loop by the antiodromic wave front of the pacing impulse at the different pacing rates (the faster the overdrive pacing rate, the greater the antiodromic penetration).

**Importance of Fusion Beats for the Demonstration of Transient Entrainment**

Transient entrainment without any demonstrable fusion beats, in some cases even at any overdrive pacing rate, was demonstrated in the present study and was related to conduction time through the various portions of the reentry loop (particularly through the area of slow conduction), to conduction time from the pacing site to the various portions of the reentry loop, and to conduction block at a site in the reentry loop other than the area of slow conduction. One might also expect transient entrainment of a tachyarrhythmia without any demonstrable fusion beats if one paces just distal to the area of slow conduction and unidirectional block, the latter, of course, being in the pathway of the antiodromic wave front. The inability to demonstrate fusion beats during overdrive pacing may make transient entrainment difficult, if not impossible, to distinguish from overdrive suppression of an automatic focus or a protected arrhythmogenic focus due to any mechanism (automatic, triggered, or reentrant). However, subsequent interruption of the arrhythmia by overdrive pacing at a faster rate is inconsistent with an automatic mechanism.\(^24\)

**Transient Entrainment and Interruption of AV Bypass Pathway—Type Paroxysmal Atrial Tachycardia as a Model for Understanding the Mechanism of Other Tachycardias**

Since rhythms due to classic automaticity can neither be initiated nor terminated by cardiac pacing, the ability to initiate or terminate an arrhythmia with a premature beat or with overdrive pacing identified was thought to be due to reentry;\(^25\) but this no longer holds because of the recent important demonstration that such pacing can also initiate and terminate arrhythmias due to triggered activity.\(^26\)\(^-\)\(^30\) However, our studies suggest that if one can demonstrate transient entrainment and subsequent interruption of a tachyarrhythmia, it is best explained by reentry with an excitable gap in the reentry loop. Therefore, we suggest the following hypothesis: (1) Before the interruption of a tachyarrhythmia with overdrive pacing, if one can demonstrate constant fusion beats during a period of transient entrainment of the tachyarrhythmia except during the last entrained beat which is not fused, or if during a period of transient entrainment of the tachyarrhythmia at two or more different pacing rates, one can demonstrate constant fusion beats at each of the different pacing rates, but different degrees of fusion (progressive fusion) at the different rates. (2) Associated with the interruption of a tachyarrhythmia with overdrive pacing, if one can demonstrate localized block to a site that is then activated by the next pacing impulse from a different direction and with a shorter conduction time than before the localized block, then the underlying mechanism of the arrhythmia must be reentry. These responses to overdrive pacing cannot be explained by tachyarrhythmias due to other known mechanisms, such as normal or abnormal automaticity,\(^24\) triggered activity,\(^26\)\(^-\)\(^30\) or a parasystolic focus\(^31\)\(^,\)\(^32\) whose intrinsic rhythm is generated by any mechanism.

The inability to demonstrate any of the components of the hypothesis during overdrive pacing of a tachyarrhythmia does not, however, mean that the tachycardia is not due to a reentry mechanism or that no transient entrainment took place during overdrive pacing. Neither transient entrainment nor interruption of a tachyarrhythmia may be possible if the tachyarrhythmia is due to the leading circle type of reentry.\(^33\)\(^-\)\(^35\) in
which there is essentially no excitable gap in the reentry loop. Furthermore, transient entrainment can occur without any fusion beats. In the latter circumstance, it may be impossible to differentiate transient entrainment of a reentrant arrhythmia from overdrive suppression of a protected arrhythmogenic focus.\(^1\) Finally, we have shown that overdrive pacing interruption of an arrhythmia may occur without the demonstration of localized block to a site. Thus, we believe that application of our narrowly drawn focus, with the awareness of the above limitations, will be a reliable means of identifying reentrant tachyarrhythmias.

**Acknowledgments**

The authors thank Drs. Pedro Brugada, Richard W. Henthorn, Michael R. Rosen, Heim J.J., Wellens, Andrew L. Wit, and Samuel L. Zimmerman for their critical review of our manuscript and also the above mentioned as Drs. Penelope Boyden, Paul F. Cranefield, and Brian F. Hoffman for their critical assessment of the concept of transient entrainment.

**References**

21. Plumb VJ, MacLean WAH, Cooper TB, James TN, Waldo AL: Atrial events during entrainment and interruption of atrial flutter by rapid atrial pacing. (abstr) Circulation 60 (suppl II): II-64, 1979
Transient entrainment and interruption of the atrioventricular bypass pathway type of paroxysmal atrial tachycardia. A model for understanding and identifying reentrant arrhythmias.
A L Waldo, V J Plumb, J G Arciniegas, W A MacLean, T B Cooper, M F Priest and T N James

_Circulation_. 1983;67:73-83
doi: 10.1161/01.CIR.67.1.73
_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1983 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/67/1/73.citation

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/