Demonstration of the Mechanism of Transient Entrainment and Interruption of Ventricular Tachycardia With Rapid Atrial Pacing

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An unusual case is presented in which ventricular tachycardia at a rate of 141 beats/min was transiently entrained by rapid atrial pacing at rates of 150, 155 and 160 beats/min, and was interrupted by rapid atrial pacing at a rate of 165 beats/min. During each period of transient entrainment, constant ventricular fusion beats were present except for the last entrained beat, and progressive ventricular fusion (different fusion QRS complexes) was demonstrated when comparing QRS complex configurations during transient entrainment at each pacing rate. Interruption of the ventricular tachycardia was associated with localized conduction block to the right ventricular recording site, followed by activation of that site from a different direction and with a shorter conduction time by the subsequent pacing impulse. These data clearly distinguish transient entrainment of a tachycardia from overdrive suppression of a tachycardia, and strongly suggest that reentry was the underlying mechanism of the ventricular tachycardia.

Transient entrainment of a tachycardia with rapid pacing is an increase in the rate of the tachycardia to a faster pacing rate, with resumption of the intrinsic rate of the tachycardia on either abrupt cessation of pacing or slowing of the pacing rate below the intrinsic rate of the tachycardia (1,2). Previous studies of transient entrainment and subsequent interruption of atrial flutter during rapid atrial pacing (3) and of ventricular tachycardia during rapid right ventricular pacing (1) provided the first descriptions of transient entrainment of an arrhythmia and differentiated such entrainment from simple overdrive suppression of an arrhythmia. However, the mechanism of the transient entrainment and interruption of both atrial flutter and ventricular tachycardia was unclear.

We recently demonstrated (2) the mechanism of transient entrainment and interruption of atrioventricular (AV) bypass pathway type paroxysmal atrial tachycardia. Transient entrainment of this tachycardia was shown to be due to repeated early entrance of the wavefront from the pacing impulse into the spontaneous tachycardia's reentrant loop in both an antidromic and orthodromic direction. The antidromic wavefront from each pacing impulse was repeatedly blocked as it collided with the orthodromic wavefront of the previous beat and, in effect, extinguished the tachycardia. However, the early entrance of the orthodromic wavefront of each pacing impulse repeatedly reset the tachycardia. The result was that during transient entrainment, the tachycardia rate increased to the pacing rate. With termination of pacing after transient entrainment, the tachycardia resumed because the orthodromic wavefront of the last pacing impulse traveled around the reentrant loop unopposed, thereby resulting in continuation of the spontaneous tachycardia at its previous spontaneous rate. Interruption of the tachycardia occurred when rapid pacing produced a block within the reentrant loop of both the antidromic and orthodromic wavefronts of the same pacing impulse during the same beat.

On the basis of these data, we suggested that transient entrainment of AV bypass pathway type paroxysmal atrial tachycardia could be used as a model for identifying and understanding reentrant arrhythmias (2). We also suggested that the underlying mechanism of the arrhythmia can be best explained by reentry (2) if: 1) one can demonstrate constant fusion beats during transient entrainment of a tachycardia except for the last transiently entrained beat, 2) during transient entrainment of a tachycardia at two or more different pacing rates, one can demonstrate constant fusion at each of the different pacing rates but different degrees of fusion...
(progressive fusion) at the different rates, or 3) interruption of a tachyarrhythmia by rapid pacing is associated with localized conduction block to a site followed by activation of that site from a different direction and with shorter conduction time by the next pacing impulse.

The present report demonstrates all three of these phenomena during transient entrainment and interruption of ventricular tachycardia during rapid atrial pacing. This unique case is presented for several reasons: 1) for the first time, it illustrates the mechanism of transient entrainment and interruption of ventricular tachycardia; 2) it shows clearly the criteria required for establishing unquestionable presence of transient entrainment of ventricular tachycardia (as opposed to being unable to differentiate transient entrainment from simple overdrive suppression of a protected focus [1] or the like); 3) it fulfills all the criteria during transient entrainment and subsequent interruption of an arrhythmia that strongly suggest an underlying reentrant mechanism; and 4) it serves to highlight the underappreciated point that ventricular tachycardia may be interrupted by rapid atrial pacing if 1:1 AV conduction at a sufficiently rapid rate can be achieved.

Case Report

Surgical Procedure and Postoperative Pacing

In 1978, a 38 year old man underwent open heart surgery to treat aortic regurgitation and a false aneurysm extending from near the right coronary cusp to the muscular interventricular septum. The aortic valve was replaced with a Carpentier-Edwards valve prosthesis, and the false aneurysm was oversewn. At the termination of surgery, but before closure of the chest wall, a pair of temporary epicardial wire electrodes were placed high in the right atrium and a single epicardial wire electrode was placed on the right ventricular epicardium. All electrodes were then brought out through the anterior chest wall for potential diagnostic and therapeutic use in the postoperative period (4,5).

In the immediate postoperative period, the patient had sinus rhythm. The electrocardiogram demonstrated first degree AV block, a right bundle branch block pattern and marked left axis deviation. Subsequently, the patient developed recurrent ventricular tachycardia with a left bundle branch block pattern (Fig. 1) that was resistant to antiarrhythmic drug therapy, which included procainamide, lidocaine and phenytoin.

Because the patient was hemodynamically stable and there was 1:1 ventriculoatrial (VA) conduction during the episodes of ventricular tachycardia, rapid atrial pacing was utilized in an effort to interrupt or control the arrhythmia. Atrial pacing was performed at selected rates using a Medtronic 1349A programmable stimulator. During the pacing procedures, electrocardiographic leads 1 and V1, either the bipolar right atrial electrogram or bipolar atrial pacing stimulus, and the unipolar right ventricular electrogram were recorded simultaneously using an Electronics for Medicine model DR12 switched beam oscilloscopic recorder. All data were also recorded on a Honeywell model S600C FM magnetic tape recorder for later playback and analysis.

Transient Entrainment of Ventricular Tachycardia

Pacing at 150 beats/min. Increase in ventricular rate during rapid pacing. Figure 2 illustrates leads I and V1 recorded simultaneously with the right atrial stimulus artifact and the unipolar ventricular electrogram during rapid atrial pacing at a rate of 150 beats/min (400 ms cycle length).

Figure 1. Leads I and V1 recorded simultaneously with a bipolar atrial electrogram (AEG) and a unipolar ventricular electrogram (VEG) during spontaneous ventricular tachycardia (VT) at a rate of 141 beats/min (425 ms cycle length). In this and subsequent figures, the circled number indicates the duration of each QRS complex in milliseconds and time lines are at 1 second intervals.

Figure 2. Leads I and V1 recorded simultaneously with the atrial pacing (A Pace) stimulus (S) artifact (Stim.) from the atrial pacing site and the unipolar ventricular electrogram (VEG) during atrial pacing at a rate of 150 beats/min (400 ms).
Note that the ventricular rate increases to the pacing rate, fulfilling one of the criteria for transient entrainment. Also, note that the configuration and duration of the QRS complexes in leads I and V₁ are constant, but have changed when compared with those during the spontaneous ventricular tachycardia. However, the configuration of the unipolar ventricular electrogram is unchanged compared with that during the spontaneous ventricular tachycardia.

Return of tachycardia to spontaneous rate on termination of pacing. Figure 3 illustrates the termination of atrial pacing at the rate of 150 beats/min and demonstrates that the ventricular tachycardia returns to its spontaneous rate promptly after termination of pacing, fulfilling another criterion of transient entrainment. Note, however, that the ventricular cycle length in all leads (I, V₁ and the ventricular electrogram) 1 beat beyond the last pacing stimulus is the same (400 ms) as the pacing cycle length. It is not until the subsequent cycle that the ventricular tachycardia returns to its previous spontaneous rate. Of special interest, the last transiently entrained beat (asterisk) has a different QRS configuration than during the previous period of transient entrainment, but this beat has the same QRS configuration as during the ventricular tachycardia. Also note that conduction time from the last stimulus artifact to the last transiently entrained beat at the right ventricular recording site is 620 ms.

Nonfusion of last entrained beat. As per our previous observations of similar phenomena during transient entrainment of AV bypass pathway type paroxysmal atrial tachycardia (2), the data in Figures 2 and 3 indicate that during transient entrainment, there was fusion of ventricular activation resulting from collision of the antidromic wavefront with the orthodromic wavefront of each previous beat. However, because the orthodromic wavefront of the last pacing impulse had no antidromic wavefront from a subsequent beat with which to collide, this last entrained beat was not fused despite occurring at the pacing cycle (Fig. 3). These observations, therefore, fulfill one of the suggested criteria for identifying a reentrant rhythm, namely, the presence of constant fusion beats during transient entrainment except for the last transiently entrained beat (2). Finally, the unchanged configuration of the ventricular electrogram during transient entrainment and after termination of pacing indicates that this electrode recording site is being activated from the same direction, orthodromically, both during rapid atrial pacing and during the spontaneous ventricular tachycardia. Figure 4 diagrammatically illustrates ventricular activation consistent with the data in Figures 1 to 3.

Pacing at 155 beats/min. Figure 5 demonstrates the termination of atrial pacing at a rate of 155 beats/min (387 ms cycle length). Note that during the period of rapid atrial pacing, the ventricular rate again increases to the pacing rate. Note also that the configuration and duration of the QRS complexes in leads I and V₁ are constant during the period of pacing, but have changed further when compared with those during both the spontaneous ventricular tachycardia and the previous rapid atrial pacing at 150 beats/min (that is, there is progressive fusion). However, the configuration of the right ventricular electrogram again remains unchanged during pacing compared with that during the spontaneous tachycardia. With the termination of atrial pacing (open circle), the ventricular tachycardia promptly resumes at its previous rate. Once again, the last transiently entrained beat (asterisk) occurs one cycle length beyond the last pacing stimulus and the QRS complexes in leads I and V₁ of this last entrained beat are not fused but rather have the same configuration as during the spontaneous ventricular tachycardia. Note also that conduction time from the last stimulus artifact to the last transiently entrained beat at the right ventricular recording site is 626 ms.

Progressive fusion. Figure 6 diagrammatically depicts ventricular activation consistent with the data recorded in Figure 5; activation is identical to that for the events diagrammatically depicted in Figure 4, except that with the shorter cycle length present during the faster pacing rate, the antidromic wavefront can penetrate further into the reentrant loop. Therefore, each antidromic wavefront collides with the orthodromic wavefront of each preceding beat at a different ventricular location than at the previous slower pacing rate, resulting in different degrees of fusion (progressive fusion) at the two pacing rates. The fact that the
Figure 4. A, Left panel, Diagrammatic representation of the reentrant loop during spontaneous ventricular tachycardia (VT) using a model suggested from the work of Wit et al. (6) and El-Sherif et al. (7). The X represents the orthodromic wavefronts of the reentrant rhythm. In this and subsequent diagrams, the arrows indicate the direction of spread of the impulse, the box represents an area of slow conduction, the serpentine line indicates slow conduction of the impulse in the latter area, the dot represents the right ventricular electrogram (VEG) recording site and the large arrow indicates the wavefront from the pacing impulse entering into the ventricular tachycardia reentrant loop, where it is conducted orthodromically (ortho) and antidromically (anti). Middle panel, Diagrammatic representation of the introduction of the first pacing impulse (X + 1) during atrial pacing at a rate of 150 beats/min during the spontaneous ventricular tachycardia. The antidromic wavefronts from the pacing impulse (X + 1) collide with the orthodromic wavefronts from the previous spontaneous beat (X), resulting in fusion of ventricular activation which, in effect, interrupts the tachycardia. However, the orthodromic wavefront from the pacing impulse (X + 1) continues the ventricular tachycardia, resetting it to the pacing rate. Right panel, Diagrammatic representation of the introduction of the second pacing impulse (X + 2) during atrial pacing at a rate of 150 beats/min during the spontaneous ventricular tachycardia. The antidromic wavefronts (X + 2) collide with the orthodromic wavefronts from the previous paced beat (X + 1), again resulting in ventricular fusion, which, again, in effect interrupts the ventricular tachycardia. However, once again the orthodromic wavefront (X + 2) from the pacing impulse continues the ventricular tachycardia, resetting it to the pacing rate. Note that during the spontaneous rhythm and during the period of pacing, the right ventricular electrogram (VEG) recording site is always activated by an orthodromic wavefront. B, Diagrammatic representation of the termination of atrial pacing illustrated in Figure 3. Left panel, The large arrow indicates the wavefront from the last pacing impulse delivered at a rate of 150 beats/min entering into the reentrant loop of the ventricular tachycardia, where it is conducted orthodromically and antidromically. The antidromic wavefronts (X, a) collide with the orthodromic wavefronts (X, [a]) of the previous beat (X, − 1) resulting in fusion of ventricular activation, which, in effect, interrupts the tachycardia, but the orthodromic wavefront from the last pacing impulse continues, and, in fact, resets it. Right panel, The orthodromic wavefronts from the last pacing impulse are now unopposed by antidromic wavefronts from a subsequent pacing impulse, so that no fusion of ventricular activation occurs despite the presence of transient entrainment. This last entrained beat restarts the tachycardia (dashed lines) which continues spontaneously. See text for discussion.

right ventricular electrogram has the same configuration during rapid atrial pacing as during the spontaneous rhythm indicates that this recording site is being activated orthodromically (that is, from the same direction as during the spontaneous ventricular tachycardia).

Figures 3 and 5 illustrate all the criteria necessary to establish the presence of transient entrainment: 1) increase in the rate of the tachycardia to the pacing rate, 2) resumption of the intrinsic rate of the tachycardia on cessation of pacing, 3) constant fusion during the period of transient entrainment except for the last transiently entrained beat, and 4) progressive fusion. The latter two criteria also have been suggested as demonstrating a reentrant rhythm (2).

Pacing at 160 beats/min. Figure 7 illustrates termination of atrial pacing at a rate of 160 beats/min (375 ms cycle length). Again, all the phenomena of transient entrainment are clearly illustrated, namely, constant ventricular fusion except for the last transiently entrained beat (asterisk); progressive fusion, as the QRS complexes in leads I and V1 during rapid atrial pacing at a rate of 160 beats/min show

Figure 5. Leads I and V1 recorded simultaneously with either the atrial pacing stimulus (S) artifact (Stim) or atrial electrogram (AEG) and unipolar ventricular electrogram (VEG) at the termination of atrial pacing at a rate of 155 beats/min (387 ms). All intervals are in milliseconds.
Figure 6. Diagrammatic representation of the termination of atrial pacing illustrated in Figure 5. **Left panel.** Identical to the description of the left panel in Figure 4B, except that the pacing impulse was delivered at a rate of 155 beats/min. **Right panel.** Identical to the description of the right panel in Figure 4B. Note that the right ventricular electrogram (VEG) recording site is activated only by an orthodromic wavefront from the pacing impulse. See text for discussion.

Figure 8. Diagrammatic representation of the termination of atrial pacing illustrated in Figure 7. **Left panel.** Identical to the description of the left panel in Figure 4B, except that the last pacing impulse was delivered at a rate of 160 beats/min. **Right panel.** Identical to the description of the right panel in Figure 4B. Note again that the right ventricular electrogram (VEG) recording site is activated only by an orthodromic wavefront from the pacing impulse. See text for discussion.

Figure 7. Leads I and V1 recorded simultaneously with the atrial pacing stimulus (Stim) artifact (Stim), and the unipolar ventricular electrogram (VEG) during atrial pacing at a rate of 160 beats/min (364 ms cycle length). The **circled stars** denote an abrupt change in configuration of the recorded QRS complexes in both leads and in the unipolar ventricular electrographic complex. In the ventricular electrographic tracing, each **arrow** points to the resulting ventricular electrogram. Before the change in configuration of the QRS complex and ventricular electrogram, the stimulus to ventricular electrogram interval is 364 ms. Then, after the localized block to the ventricular electrographic recording site, the stimulus to ventricular electrogram interval becomes 305 ms. Note also that this localized conduction block is associated with a one cycle increase in the beat to beat cycle length localized to the ventricular electrographic recording site (from 364 to 425 and then back to 364 ms). In the lead I tracing, the **dashed arrows** represent the antidromic wavefronts and the associated **solid arrows** represent the orthodromic wavefronts from the 5th and 6th pacing impulses. After the block of both the antidromic and the orthodromic impulses of the 6th pacing impulse in the reentrant loop of the ventricular tachycardia, the ventricles are activated by the 7th pacing impulse, as expected, during overdrive atrial pacing of a sinus rhythm. All intervals are in milliseconds.

**Figure 9.** Leads I and V1 recorded simultaneously with the atrial pacing stimulus (S) artifact (Stim), and the unipolar ventricular electrogram (VEG) during atrial pacing at a rate of 165 beats/min (364 ms cycle length). The **circled stars** denote an abrupt change in configuration of the recorded QRS complexes in both leads and in the unipolar ventricular electrographic complex. In the ventricular electrographic tracing, each **arrow** points to the resulting ventricular electrogram. Before the change in configuration of the QRS complex and ventricular electrogram, the stimulus to ventricular electrogram interval is 640 ms. Then, after the localized block to the ventricular electrographic recording site, the stimulus to ventricular electrogram interval becomes 305 ms. Note also that this localized conduction block is associated with a one cycle increase in the beat to beat cycle length localized to the ventricular electrographic recording site (from 364 to 425 and then back to 364 ms). In the lead I tracing, the **dashed arrows** represent the antidromic wavefronts and the associated **solid arrows** represent the orthodromic wavefronts from the 5th and 6th pacing impulses. After the block of both the antidromic and the orthodromic impulses of the 6th pacing impulse in the reentrant loop of the ventricular tachycardia, the ventricles are activated by the 7th pacing impulse, as expected, during overdrive atrial pacing of a sinus rhythm. All intervals are in milliseconds.

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has increased to the atrial pacing rate, and a fusion QRS configuration in leads I and V₁ is present (representing further progressive fusion). Initially, the right ventricular electrogram maintains its same configuration as during the spontaneous tachycardia, indicating a period of transient entrainment. However, the QRS configuration suddenly changes to a right bundle branch block pattern (star), the same configuration recorded during previous periods of sinus rhythm. The right ventricular electrographic configuration also changes (star), indicating that the right ventricular recording site is activated from a different direction than during both the period of spontaneous ventricular tachycardia and the previous periods of transient entrainment. In fact, it is the same ventricular electrographic configuration that was recorded during previous sinus rhythm. Figure 10 illustrates the termination of atrial pacing at 165 beats/min recorded several seconds after the last beat in Figure 9. Note that the ventricular tachycardia has been interrupted after termination of pacing. Figure 11 shows that sinus rhythm resumes about 3 seconds after termination of pacing.

Localized conduction block. Analysis of the sequence of events in Figures 9 and 10 and events during the termination of pacing in Figures 3, 5 and 7 demonstrates that the abrupt change in configuration of the right ventricular electrogram and simultaneous appearance of the right bundle branch block pattern in the electrocardiographic leads (Fig. 9) are associated with localized block of one pacing impulse to the right ventricular recording site. During transient entrainment of the ventricular tachycardia at atrial pacing rates of 150 (Fig. 3), 155 (Fig. 5) and 160 beats/min (Fig. 7), conduction times from the atrial pacing site to the right ventricular recording site were 620, 626 and 632 ms, respectively, considerably longer than each respective pacing cycle length. This remarkably prolonged conduction time is most easily appreciated by examining the interval from the last atrial pacing stimulus to the last entrained beat at the right ventricular electrographic recording site (Fig. 3, 5 and 7). This very long conduction time resulted in the presence of a skip phenomenon; that is, the atrial pacing stimulus and its resulting right ventricular electrogram were always temporally separated by a right ventricular electrogram caused by a previous atrial pacing stimulus.

However, as is clear from analysis of the data in Figure 10, at the termination of atrial pacing at a rate of 165 beats/min, there is no skip phenomenon. Rather, the last atrial pacing stimulus is immediately followed by its resulting right ventricular electrogram. Furthermore, as one works back from the last atrial pacing stimulus and its associated ventricular electrogram to the point where the morphologic changes occur in the right ventricular electrogram and the QRS complexes in leads I and V₁ (Fig. 9 and 10), it becomes clear that the skip phenomenon was present before those changes. However, with the occurrence of those changes, there was no longer any skip phenomenon. Thus, there had to be localized block of one pacing impulse to the right ventricular recording site. The presence of localized block is further supported by the localized prolongation of the cycle length from 364 to 425 ms immediately preceding the change in ventricular electrographic configuration. After the localized block, the right ventricular recording site was activated by the next pacing impulse, but from a different direction (supported by the change in electrographic configuration recorded from this fixed electrode) and with a shorter conduction time (305 ms compared with the immediately preceding 640 ms) (Fig. 9). And again, the cycle length at this site was the same as the pacing cycle length. The demonstration of localized conduction block to a site followed by activation of that site from a different direction and with a shorter conduction time is another suggested criterion for demonstrating a reentrant rhythm (2).
Presence of an Area of Slow Ventricular Conduction

Analysis of conduction times from the atrial pacing stimulus to each associated right ventricular electrogram during atrial pacing is of some interest. The stimulus to onset of the QRS complex interval (equivalent to a PR interval) varied from 235 ms at the slowest atrial pacing rate (150 beats/min) to 255 ms at the fastest atrial pacing rate (165 beats/min), these intervals being long as a result of prolongation of the AV nodal conduction time associated with rapid atrial pacing (8). The stimulus to right ventricular electrogram interval varied from 620 ms during transient entrainment at the atrial pacing rate of 150 beats/min to 640 ms during transient entrainment at the atrial pacing rate of 165 beats/min. The 20 ms increase in this interval is also undoubtedly explained largely by prolongation of AV nodal conduction time at the faster pacing rate (8). However, the enormous difference (385 ms) between the stimulus to onset of ventricular activation interval and the stimulus to right ventricular electrogram interval, present during transient entrainment at each pacing rate, strongly supports the presence of an area in the ventricular tachycardia reentrant loop of slow conduction through which the orthodromic wavefront of each pacing impulse travels before it reaches the right ventricular electrographic recording site. This, of course, is completely consistent with the present understanding of the requirements to obtain and maintain a reentrant rhythm (9).

Of further interest, conduction through this region must take place during the interval between inscription of the QRS complexes, yet no indication of ventricular activation interval and the stimulus to right ventricular electrogram interval, present during transient entrainment at each pacing rate, strongly supports the presence of an area of slow conduction in the ventricular tachycardia reentrant loop of slow conduction through which the orthodromic wavefront of each pacing impulse travels before it reaches the right ventricular electrographic recording site. This, of course, is completely consistent with the present understanding of the requirements to obtain and maintain a reentrant rhythm (9). Of further interest, conduction through this region must take place during the interval between inscription of the QRS complexes, yet no indication of ventricular activation appears in the electrocardiograms during this interval.

Finally, whether slow conduction in this area of the ventricles persists after interruption of the ventricular tachycardia is uncertain. However, the fact that immediately after interruption of the ventricular tachycardia, the stimulus to right ventricular electrogram interval decreased by 335 ms and the QRS complex configuration returned to “normal” suggests that this area of slow conduction in the ventricle was only functionally present during the ventricular tachycardia.

Causes of electrographic changes during pacing at 165 beats/min. The changes in configuration of the QRS complexes in electrocardiographic leads I and V1 and in configuration of the right ventricular electrogram that occurred during atrial pacing at 165 beats/min can be explained by the following sequence (Fig. 12): During the initial period of transient entrainment at the atrial pacing rate of 165 beats/min, each wavefront from the pacing impulse is conducted antidromically and orthodromically into the reentrant loop. Thus, initially the ventricular electrographic configuration is unchanged from that during the spontaneous ventricular tachycardia and during pacing at the previous slower rates, because this site is still activated by an orthodromic wavefront. The antidromic wavefronts from each pacing impulse continue to collide with the orthodromic wavefronts from the previous beat, resulting in a fusion QRS configuration. Abruptly, however, not only are the antidromic wavefronts of the subsequent pacing impulse blocked by the orthodromic wavefronts of the preceding beat, but the orthodromic wavefront of that same pacing impulse is also blocked during the same beat. This results in interruption of the ventricular tachycardia. There being no more functional reentrant loop, the wavefront from the next atrial pacing impulse is conducted to and activates the ventricles as if one were overdrive pacing a sinus rhythm, resulting in the usual QRS configuration for a normally conducted supraventricular beat in this patient. Therefore, the electrocardiographic configuration of the right ventricular epicardium is activated from a different direction (explaining the resulting change in configuration of the right ventricular electrogram) and with a shorter conduction time (because the activation wave-
front to this site no longer has to enter the reentrant loop orthodromically and travel through the loop and its presumed area of slow conduction to the recording site).

Discussion

Reentrant mechanism of transient entrainment and interruption of tachycardia. Our case is a clear and dramatic representation of transient entrainment and ultimate interruption of ventricular tachycardia with rapid atrial pacing. Extrapolating from our understanding of the mechanism of transient entrainment and interruption of atrioventricular (AV) bypass pathway type and paroxysmal atrial tachycardia (2), the data from this case are best explained by an underlying reentrant mechanism. As in the case of AV bypass pathway type paroxysmal atrial tachycardia, transient entrainment of the ventricular tachycardia by rapid atrial pacing is explained by early entrance of the wavefront from each pacing impulse into the excitable gap of a reentrant loop in both an antidromic and orthodromic direction. Each antidromic wavefront from each impulse collides with the orthodromic wavefront of the preceding beat resulting in a constant fusion QRS configuration. Each orthodromic wavefront from each pacing impulse resets the ventricular tachycardia, resulting in an increase of the ventricular tachycardia to the pacing rate. With termination of pacing because the orthodromic wavefront of the last pacing impulse is unopposed by an antidromic wavefront of a subsequent pacing impulse, the last entrained beat shows no fusion QRS configuration.

During transient entrainment at progressively faster pacing rates (150, 155 and 160 beats/min), the shorter pacing cycle lengths associated with these rates resulted in earlier entrance into the reentrant loop by the wavefronts from each pacing impulse. Therefore, the antidromic wavefront of each pacing impulse was able to penetrate the reentrant loop to a greater degree with each increment in pacing rate. Thus, while the place of collision of each antidromic wavefront with the preceding orthodromic wavefront was constant at each pacing rate, it was altered with each change in pacing, resulting in constant QRS fusion configuration at each pacing rate but progressive fusion of QRS configuration when comparing pacing at the different pacing rates.

Interruption of the ventricular tachycardia with rapid pacing only occurred when the antidromic and orthodromic wavefronts of the same pacing impulse were blocked during the same beat. This was identified by localized conduction block to a portion of the ventricles followed by activation of this same portion of the ventricles by wavefronts from subsequent pacing stimuli. These latter wavefronts activated the "blocked" sites from a different direction and with a shorter conduction time because these sites were no longer being activated by an orthodromic wavefront from the reentrant loop, but rather as one would expect when overdrive pacing a sinus rhythm from that pacing site.

The demonstration of these responses of ventricular tachycardia to rapid pacing are best explained by a reentrant mechanism with the reentrant loop of the tachycardia having an excitable gap (2). They cannot suitably be explained by other known mechanisms such as normal or abnormal automaticity (10), triggered activity (11–15) or a parasystolic focus (16,17) in which intrinsic rhythm is generated by any mechanism.

Implications for treatment of ventricular tachycardia with rapid pacing. These studies also highlight two other matters of practical importance for the treatment of ventricular tachycardia with rapid pacing. First, as we have demonstrated previously (1), in order to interrupt ventricular tachycardia with rapid pacing techniques, a critically rapid rate must be achieved. Pacing at rates faster than that of the spontaneous ventricular tachycardia but slower than the critically rapid rate will only transiently entrain the tachycardia. These observations are similar to those previously demonstrated for atrial flutter (3,18–20), AV bypass pathway type paroxysmal atrial tachycardia (2) and ectopic atrial tachycardia (21). Second, atrial pacing at sufficiently rapid rates can interrupt ventricular tachycardia provided 1:1 AV conduction at the critically rapid rate can be achieved.

References

18. 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 1979;60(suppl II):II–64.