


RECURRENT VT: MECHANISMS/ Josephson et al.

1. Mechanisms

MARK E. JOSEPHSON, M.D., LEONARD N. HOROWITZ, M.D., ARDESHIR FARSHIDI, M.D., AND JOHN A. KASTOR, M.D.

SUMMARY The mechanism of recurrent sustained ventricular tachycardia (VT) was evaluated in 21 patients. Re-entry as the mechanism for VT was suggested by a) the reproducible initiation (19) and termination (15) of the arrhythmia by programmed stimulation. The rate, ventricle of origin, and stimulation site determined the method of termination. One VPD was usually required for VT rates less than 175/min and/or ventricle of origin ipsilateral to the stimulation site, while two VPDs were usually required for VT with faster rates originating in a contralateral ventricle. The proximal His-Purkinje system (HPS) was not required for initiation or maintenance of VT. Evidence localizing the site of re-entry to a small portion of the ventricles included: a) ventricular capture by ventricular premature depolarizations (VPDs) or pacing (VP) without terminating VT (5), b) sinus capture following VPDs and/or supraventricular fusions without terminating VT (12), and c) atrial pacing normalizing the QRS and H-V intervals without terminating VT (5).

THE MECHANISM OF RECURRENT SUSTAINED VENTRICULAR TACHYCARDIA is not established. Recent investigations utilizing intracardiac stimulation and recording techniques to evaluate this arrhythmia have yielded conflicting results. Studies by Wellens et al.4 suggested that most recurrent ventricular tachycardias could be reproducibly initiated and/or terminated by programmed stimulation, implicating re-entry as the underlying mechanism. Furthermore, it has been postulated that the bundle branches can be an integral part of the re-entrant circuit.1,6 8 In contrast, a recent publication by Denes et al.7 suggested that most ventricular tachycardias could not be predictably induced or terminated.

The present study was undertaken to investigate the mechanism of chronic sustained ventricular tachycardia and the role of the bundle branches and ventricular myocardium in its initiation and maintenance.

From the Electrophysiology Laboratory, Hospital of the University of Pennsylvania, Cardiovascular Section, Department of Medicine, University of Pennsylvania School of Medicine, Philadelphia, Pennsylvania. Dr. Horowitz is recipient of a Career Development Investigatorship, American Heart Association, Southeastern Pennsylvania Chapter.

Address for reprints: Mark E. Josephson, M.D., Director, Electrophysiology Laboratory, 660 White Bldg., Hospital of the University of Pennsylvania, 3400 Spruce Street, Philadelphia, Pennsylvania 19104.

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Table 1. Clinical Data

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*Q, 2, 3, F = with normal angiographic coronary arteries.

Abbreviations: ASMI = anteroseptal myocardial infarction; ASHD = atheroembolic heart disease; AVB = atrioventricular block; CAA = coronary artery anomaly; CL = cycle length; CM = cardiomyopathy; F = female; HCVD = hypertensive cardiovascular disease; IACD = intracranial conduction defect; ICRBBB = incomplete right bundle branch block; IHSS = idiopathic hypertrophic subaortic stenosis; Indet = indeterminate; IMI = inferior myocardial infarction; IVCD = intraventricular conduction defect; LAH = left anterior hemiblock; LBBB = left bundle branch block; LVH = left ventricular hypertrophy; M = male; NTMI = non-transmural myocardial infarction; RBBB = right bundle branch block; WNL = within normal limits; Vent An = ventricular aneurysm.

Table 2. Electrophysiologic Data

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a performed at a basic cycle length of 600 msec.

Abbreviations: BBR = bundle branch re-entry; CL = cycle length; ERP-LV = effective refractory period of left ventricle; ERP-RV = effective refractory period of right ventricle; Indet = indeterminate; LBBB = left bundle branch block; RBBB = right bundle branch block; VERP = ventricular effective refractory period.
None of the patients had specifically referred for diagnostic and/or therapeutic evaluation of their ventricular tachycardia. Electrophysiologic studies were performed in the non-sedated postabsorptive state after informed consent had been obtained. Three patients had ventricular tachycardia at the beginning of the study, the remainder were in sinus rhythm. Three to six electrode catheters were inserted either percutaneously or by cutdown and fluoroscopically positioned in the heart. The number of recording sites varied from patient to patient but usually included the right atrium, A-V junction at the His bundle site, right ventricle at the apex, septum, inflow and/or outflow tract; left ventricle, either at the apex, septum, lateral wall, and/or base, and coronary sinus. Quadrupolar electrode catheters were generally used when recording and stimulation from a given site were required; in these cases one pair of electrodes was used for recording and the other for stimulation. A tripolar catheter was used to obtain the His bundle and local ventricular electrogram at the A-V junction.

Stimulation was performed using a specially designed programmable stimulator and an isolated constant current source. The stimuli were rectangular pulses 1–1.5 msec in duration at twice diastolic threshold (0.75–2 mA).

Definition of Terms

S₁ – S₁ = basic paced cycle length.
S₂ and S₃ = first and second premature stimuli delivered during an S₁ – S₁.
S₁ – S₂ = coupling interval from eighth beat of a basic drive cycle to the first premature stimulus.
S₁ – S₃ = coupling interval between first and second premature stimuli.
V₁, V₂, V₃ = ventricular depolarizations produced by S₁, S₂ and S₃ respectively.
Effective refractory period of the ventricles = the longest S₁ – S₂ which fails to evoke a ventricular depolarization.
Tachycardia zone = range of S₁ – S₂ or S₂ – S₃ coupling intervals which results in ventricular tachycardia.

Bundle branch re-entry = the appearance of an extra, nonstimulated ventricular depolarization in response to S₂ or S₃ which is dependent upon attaining a critical degree of retrograde His-Purkinje delay (V-H prolongation). The QRS of the re-entrant response usually has a configuration similar to the paced complex and is preceded by a His potential with an H-V interval equal to or greater than that of a normally conducted antegrade impulse.

Ventricle of origin of the tachycardia was tentatively classified according to the earliest recorded ventricular electrogram and by the QRS morphology of the tachycardia.

The following protocol of programmed stimulation was used:

1) Atrial pacing at incremental rates (120–220 beats/min);
2) Premature atrial stimuli during sinus rhythm and/or atrial pacing;
3) Ventricular pacing at incremental rates (80–250 beats/min);
4) Premature ventricular stimuli during sinus rhythm and/or ventricular pacing. If one premature ventricular stimulus (S₂) scanning from late diastole to ventricular refractoriness did not initiate ventricular tachycardia, scanning with a second premature stimulus (S₃) was then employed. Double premature stimuli (S₂, S₃) were introduced starting at an S₁–S₂ 50 to 100 msec longer than the ventricular effective refractory period, and S₂–S₃ equal to the S₁–S₂ interval. S₂–S₃ was shortened, and when S₂ failed to evoke a V₃, S₃–S₂ was decreased until S₃ could evoke a response or ventricular tachycardia. This method was used until both S₂ and S₃ reached refractoriness. Stimulation was performed at two or more basic ventricular cycle lengths in 16 patients. Stimulation was routinely performed from the right ventricular apex (21 patients) and from multiple right ventricular sites in six patients. In 12 patients left ventricular stimulation was also used.
5) After ventricular tachycardia was induced, programmed single, double, or triple ventricular stimuli were introduced in an attempt to terminate the arrhythmia. The extrastimuli were introduced from multiple sites including the left ventricle in ten patients.

In the three patients in whom ventricular tachycardia was present at the onset of the study, the protocol began at step 5 and was repeated beginning at step 1 after conversion to sinus rhythm.

Intracardiac recordings from several sites (usually the right atrium, right ventricular apex, A-V junction, and in 17 patients additional sites within the right ventricle, left ventricle, and/or coronary sinus) were simultaneously recorded with two or three surface electrocardiographic leads and time lines. Intracardiac electrograms were filtered at 40 to 500 Hz. The data were stored on magnetic tape and later retrieved on photographic paper at speeds of 150–400 mm/sec.

Results

Initiation of Ventricular Tachycardia

In 19 of 21 patients (90%) ventricular tachycardia could be initiated by the introduction of one or two ventricular stimuli or by rapid ventricular pacing (table 3). Twenty-seven morphologically distinct ventricular tachycardias were induced. The rates and morphology of the induced tachycardias were similar to those noted during spontaneous episodes.

Twelve ventricular tachycardias were initiated by a single ventricular premature depolarization (VPD) and 14 required two VPDs. In four patients (cases 2, 12, 14 and 18) in whom two different tachycardias were induced, two ventricular stimuli were required to induce one of the tachycardias while one stimulus initiated the other tachycardia. The tachycardia could be initiated by VPDs introduced during sinus rhythm in only three patients (cases 13, 14 and 20). In each of the remaining cases the ventricular tachycardia could only be initiated by stimulation while the ventricles were being paced. In patients in whom ventricular tachycardia could be induced during both sinus rhythm and ventricular pac-
In our study, we observed that the rate and morphology of the tachycardias were similar (fig. 1). In each case a reproducible tachycardia zone of 10 to 200 msec was present. The width of the zone was unrelated to the rate or morphology of the tachycardia. An inverse relationship of the coupling interval of the initiating VP and the interval to the first ventricular complex of the tachycardia was observed in 11 patients.

In eight patients the ventricular tachycardia could be reproducibly initiated by ventricular pacing at rates of 150 to 250 beats/min. In each case pacing was initiated late in diastole, at a coupling interval at which single premature stimuli could not induce the tachycardia. There was no relationship between the rate of the tachycardia and the number of premature stimuli or pacing rate required to initiate it. In one patient (case 11) rapid ventricular pacing was the only method of induction. In another patient (case 18) rapid atrial pacing initiated the tachycardia which was also capable of being induced by single ventricular extrastimuli or rapid ventricular pacing.

Role of Ventricular Conduction Delay and Pacing Site in the Initiation of Ventricular Tachycardia

Local conduction delay (latency) due to encroachment on local ventricular refractoriness was not a prerequisite for the development of ventricular tachycardia. In most patients the ventricular tachycardia was initiated in the absence of measurable latency (fig. 1).

Stimulation at one or more right ventricular sites and/or from the left ventricle was capable of initiating the tachycardia (fig. 2), although the zones of initiation varied somewhat from site to site. However, right ventricular stimulation was capable of inducing arrhythmia in each case in which the tachycardia could be initiated.

Role of Proximal His-Purkinje System to the Initiation and Maintenance of Ventricular Tachycardia

The initiation of ventricular tachycardia was independent of the presence or degree of retrograde His-Purkinje (HPS) conduction delay (Vf - Hf prolongation) and frequently occurred in its absence. Furthermore, the initiation of the tachycardia was unrelated to the development of bundle branch re-entry which was noted in 13 patients. When bundle branch re-entry did occur and was followed by ventricular tachycardia, the morphology of the beat due to bundle branch re-entry resembled that of the paced complex, while the morphology of the ventricular tachycardia was always different.

In no case was a consistent His bundle deflection noted at the onset of or during the ventricular tachycardia; however, dissociated His deflections were seen in ten patients during the tachycardia. In one patient a His bundle electrogram was recorded several times throughout the study appearing to be fixed at two different H-V intervals, while at other

![Figure 1](https://circ.ahajournals.org/)

**Figure 1.** Initiation of ventricular tachycardia during sinus rhythm and ventricular pacing (case 14). Analog records are organized from top to bottom: ECG leads I, aVR, V1, and electrogram from the high right atrium (HRA). His bundle (HBE) and right ventricle (RV). In panel A ventricular tachycardia is initiated during sinus rhythm with one ventricular stimulus (S1, arrow). In panel B, the same tachycardia is initiated during ventricular pacing (S1-S2) by a single premature stimulus (S1). **T** = time lines.
times no His deflections were present despite a constant QRS morphology and tachycardia rate (fig. 3). Furthermore, when A-V dissociation was present during the tachycardia, infra-His block of the supraventricular impulse (6 patients) and fusion beats due to partial antegrade ventricular depolarization (12 patients) were observed (fig. 4).

**Stimulation During Ventricular Tachycardia**

In 15 of 17 patients (88%) the tachycardia could be reproducibly terminated by programmed single or double ventricular stimuli or by ventricular pacing (table 3). In the remaining four patients hemodynamic deterioration occurred too rapidly to allow completion of the termination protocol and DC countershock was required; in each of these cases the tachycardia rate exceeded 200 beats/min. In two cases (cases 19 and 21), the tachycardia could not be terminated by any method of stimulation.

In no case could atrial pacing terminate the ventricular tachycardia. In four cases rapid atrial pacing during ventricular tachycardia resulted in supraventricular capture, documented by normalization of the sequence of intracardiac electrograms (3 cases) and QRS morphology (4 cases) without terminating the arrhythmia (fig. 5). In each of these four patients the ventricular tachycardia could be terminated by programmed ventricular depolarizations or by rapid ventricular pacing.

A single ventricular depolarization was able to terminate the tachycardia in nine patients. Termination of the arrhythmia with one VPD usually required either a slow tachycardia rate (≤ 175 beats/min) or stimulation at a site ipsilateral to the site of origin (e.g., right ventricular stimulation during right ventricular tachycardia). Two ventricular premature depolarizations or ventricular pacing at rates greater than that of the tachycardia were required to terminate the arrhythmia in six patients — a finding that tended to occur when the tachycardia rate was rapid (i.e., greater than 175 beats/min) or when the ventricular tachycardia appeared to originate in the ventricle contralateral to the site of stimulation. The inability of a single
VPD to terminate the tachycardia correlated with failure of the VPD to depolarize the opposite ventricle. Depolarization of the contralateral ventricle by the second VPD (V₂) terminated the arrhythmia (fig. 6).

In several patients apparent biventricular capture, defined by recording earlier than expected right and left ventricular electrograms in response to premature stimuli, occurred without termination of the tachycardia. The tachycardia was either unaltered or a less than compensatory pause resulted. In one patient simultaneous stimulation of both ventricles resulted in capture without termination, followed by full compensatory pauses (fig. 7). Full compensatory pauses in the presence of biventricular capture suggest that the re-entrant circuit was localized and relatively protected. This contention was substantiated by moving the left ventricular catheter into an apical aneurysm which demonstrated continuing activity despite apparent biventricular capture by double ventricular stimulation (fig. 8).

Ventricular pacing terminated the ventricular tachycardia in 12 patients. In six patients pacing at rates slower than the tachycardia (underdrive) could terminate the arrhythmia. This was always due to a random stimulus occurring at a coupling interval previously shown to be effective for a single stimulus to terminate the arrhythmia. In the remaining six patients pacing at rates faster than the tachycardia (overdrive) was required to terminate the arrhythmia. In each of these cases double ventricular stimuli had been required for termination.

In 11 patients ventricular pacing resulting in ventricular capture failed to terminate the arrhythmia (fig. 9). In 5 of these patients ventricular pacing could not initiate ventricular tachycardia; therefore, termination with subsequent reinitiation could not explain this observation.

In five cases the tachycardia persisted despite spontaneous sinus captures or sinus beats occurring during the pause produced by stimulated ventricular premature depolarizations. The possibility of termination and immediate reinitiation of the arrhythmia by the sinus beat was excluded.

**Figure 4.** Antegrade depolarization of the His-Purkinje system during ventricular tachycardia (case 1). Antegrade fusion beats are seen after the second and fifth complexes. These fusions also demonstrate premature capture of both the RV and LV recorded in the CS. The H-V intervals of the fusion complexes are 40 and 38 msec.

**Figure 5.** Failure of supraventricular capture during atrial pacing to terminate ventricular tachycardia (case 1). The panels are continuous and are organized in a fashion similar to previous records. During ventricular tachycardia (first four beats) atrial pacing from the coronary sinus (CS) is begun (arrows) at a cycle length of 350 msec. Starting with the sixth complex, antegrade capture is present as shown by a QRS morphology and A-H and H-V intervals identical to those seen during antegrade pacing at an identical rate in the absence of ventricular tachycardia. Pacing is terminated in the bottom panel but the tachycardia continues. Since atrial pacing could not induce the tachycardia, one would assume the tachycardia was continuing during atrial pacing.
by the fact that atrial premature depolarizations were previously shown to be incapable of inducing the tachycardia.

Discussion

Data Supporting a Re-entrant Mechanism

Predictable and reproducible initiation and termination of a tachyarrhythmia by programmed stimulation has been considered the sine qua non of a re-entrant mechanism. Utilizing standard intracardiac recording and stimulation techniques, we evaluated the mechanisms of chronic recurrent ventricular tachycardia in 21 patients. We are able to initiate and/or terminate the arrhythmia in a predictable fashion in 19 of 21 patients independent of the underlying cardiac pathology. Although many ventricular tachycardias were initiated by single VPDs (12/27), more required double premature stimuli for initiation (14/27).

Mechanism of Initiation

Ventricular tachycardia could be initiated by programmed ventricular stimulation during sinus rhythm in only three patients, whereas ventricular pacing with or without premature stimuli induced the tachycardia in 19. Although our data do not provide an explanation for this observation, possibilities include: a) shortening of ventricular refractoriness by ventricular pacing as compared to supraventricular rhythm; b) a direct effect of the electrical current on local dispersion of refractoriness; and c) the altered sequence of depolarization and hence inhomogeneity of repolarization produced by ventricular pacing; all of which facilitate the ability of a premature stimulus to cause re-entrant rhythms.

An inverse relationship of the coupling interval which resulted in ventricular tachycardia (V1-Vs or V2-Vt) to the interval from the initiating VPD to the first complex of the ventricular tachycardia (V2 or Vt-Vf) was frequently observed. This finding is analogous to the findings in patients with A-V nodal re-entrant tachycardia and supports re-entry as the mechanism of these ventricular tachycardias.

In seven patients ventricular tachycardia could be induced by rapid ventricular pacing. A single example of this phenomenon in man has been recently published but its mechanism was not discussed. In vitro studies have demonstrated that rapid pacing can result in rate-dependent block in depressed segments of Purkinje tissue which provides the two substrates for re-entry, slow conduction and unidirectional block. In experimental myocardial infarction, pacing-induced fragmentation of depolarization wavefronts within focal, but electrically heterogeneous areas has been demonstrated and such fragmentation has led to re-entrant ventricular arrhythmias. Recent studies in excised human ventricular aneurysms suggest similar mechanisms may be operative in man.

![Figure 6. Termination of ventricular tachycardia by two ventricular stimuli (case 17). The analog records are organized from top to bottom: ECG leads I, aV<sub>R</sub>, V<sub>L</sub>, and electrograms from the coronary sinus (CS), His bundle (HBE), right ventricular apex (RVA), and lateral wall of the left ventricle (LV-lat). Ventricular tachycardia is present at a cycle length of 435 msec. A premature right ventricular stimulus, S<sub>2</sub>, is introduced at a coupling interval of 270 msec. This captures the right ventricle but not the left (note LV-lat electrogram occurs as expected). When a second right ventricular stimulus, S<sub>3</sub>, is introduced at the identical coupling interval (S<sub>2</sub> - S<sub>3</sub> = 270 msec), both ventricles are captured and the tachycardia is terminated after another ventricular complex. The mechanism of this last complex is uncertain.](http://circ.ahajournals.org/content/115/15/437/F6)

![Figure 7. Ventricular capture without terminating ventricular tachycardia (case 16). Analog records of the electrocardiogram (I, aV<sub>R</sub>, V<sub>L</sub>, and electrograms from coronary sinus (CS), atrioventricular junction (AVJ), right ventricular apex (RVA), and lateral wall of the left ventricle (LV-lat). After the second beat of ventricular tachycardia, ventricular stimuli from both the RVA and LV-lat are introduced at a coupling interval of 345 msec without terminating the arrhythmia. After two more beats of ventricular tachycardia biventricular stimuli are introduced at a shorter coupling interval of 265 msec but the tachycardia persists. Full compensatory pauses are noted after each pair of premature stimuli.](http://circ.ahajournals.org/content/115/15/437/F7)
As a note of caution it has been recently shown that under appropriate experimental conditions, automatic rhythms due to enhanced afterpotentials can be triggered by pacing or premature stimulation. The relevance of these fascinating findings to human ventricular arrhythmias is uncertain. If triggerable automaticity is operative in the intact human heart, differentiation of this phenomena from re-entry by standard stimulation techniques may be impossible. The use of drugs, however, may help distinguish these mechanisms. Wellens has recently demonstrated the ineffectiveness of verapamil, an agent previously shown to abolish triggered automaticity, on chronic sustained ventricular tachycardia in a group of patients similar to ours. This observation provides additional support for re-entry as mechanism for this arrhythmia.

The rate and morphology of the induced ventricular tachycardia was independent of the method of initiation or refractory periods at pacing sites. Furthermore, the onset of ventricular tachycardia was unrelated to local ventricular muscle conduction delay at the site of stimulation. Delays in conduction at sites distant from the site of stimulation but in proximity to the re-entrant circuit may have been required; but this hypothesis is not directly verified in most cases. Moreover, the initiation of the tachycardia by atrial pacing and the fact that right ventricular stimulation could induce ventricular tachycardia in each patient, regardless of site of origin, suggest that local factors at the site of stimulation were not responsible for the arrhythmia. Our data suggest that left ventricular stimulation appears unnecessary for initiation of ventricular tachycardia and need not be routinely performed for this purpose.

Mechanism of Termination

In 15 of 17 patients in whom the termination protocol could be applied, the rate and morphology of the tachycardia determined the mechanism of its termination. If the tachycardia rate was less than 175 beats/min, a single ventricular stimulus, or underdrive pacing, could frequently terminate the arrhythmia, particularly a right ventricular tachycardia during right ventricular stimulation. If the rate of the tachycardia exceeded 175 beats/min, particularly if the tachycardia was left ventricular in origin and stimulation was performed from the right ventricle, two closely coupled ventricular stimuli, or overdrive ventricular pacing, were required for termination. This suggests that at a rapid tachycardia rate, a single ventricular depolarization, which conducts slowly due to its prematurity, cannot reach and/or

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**Figure 8.** Concealed perpetuation of ventricular tachycardia within a ventricular aneurysm (case 16). The records are organized similarly to figure 7; two ECG leads (2, V) are shown and the left ventricular catheter has been moved to within the left ventricular aneurysm (LV-An). During ventricular tachycardia, two right ventricular stimuli (STS) are introduced at coupling intervals of 130 and 190 msec respectively. These result in capture of the electrograms from the right ventricle and from the left ventricle as recorded in the coronary sinus without terminating the arrhythmia. The explanation for this phenomenon is apparent in the LV-An electrogram which demonstrates continuing and unaltered activity at this site. Thus concealed perpetuation of the tachycardia is due to localized and protected activity within the aneurysm.

**Figure 9.** Ventricular pacing resulting in "capture" without termination (case 14). The panels are continuous and are organized similarly to figure 1. Ventricular tachycardia is present at a cycle length of 430 msec in the first four beats. Right ventricular pacing(s) at a cycle length of 400 msec is begun late in diastole and results in apparent ventricular capture. Termination of pacing (bottom panel) reveals persistence of ventricular tachycardia. Since ventricular pacing was incapable of initiating the ventricular tachycardia, we must assume persistence of the arrhythmia during ventricular pacing.
penetrate the re-entrant circuit in time to terminate the tachycardia. When two ventricular premature depolarizations are introduced, the first shortens or "peels back" the ventricular refractory period allowing the second ventricular premature depolarization to reach the re-entrant circuit in time to terminate the arrhythmia. This was demonstrated in several patients in whom simultaneous right and left ventricular electrograms revealed that termination of the tachycardia was dependent upon the ability of the right ventricular stimulus to depolarize (capture) the left ventricle. In these cases only when double stimuli were delivered could the left ventricle be "captured" by right ventricular stimulation. These results are consistent with the factors determining the ability of a programmed stimulus to terminate the arrhythmia which include: 1) distance of stimulation site from the re-entrant circuit; 2) refractoriness of intervening tissue; and 3) conduction velocity of the stimulated wave front. These findings explain the failure of VPDs to terminate the arrhythmia despite "apparent capture," and stress the frequent requirement of multiple extrastimuli in the investigation and therapy of this arrhythmia.

Components of Re-entrant Circuit

Some investigators have suggested that the specialized conduction system, including the bundle branches, forms part of the re-entrant circuit of the ventricular tachycardia. Our data do not support this contention.

Observations at the Initiation of Ventricular Tachycardia

The initiation of ventricular tachycardia was not dependent on the development of His-Purkinje conduction delay or bundle branch re-entry in any of our patients. Thus bundle branch re-entry, which may occur in 40 to 60% of patients during ventricular stimulation, does not signify a propensity toward the development of ventricular tachycardia.

Observations During Ventricular Tachycardia

In no case were His bundle potentials causally related to ventricular depolarization during the tachycardia. If macro re-entry involving the proximal conduction system were the mechanism of this arrhythmia, consistent His bundle electrograms with a normal or prolonged antegrade conduction time should be observed. Thus, no role for the proximal His-Purkinje system in the genesis or maintenance of ventricular tachycardia could be demonstrated.

Both ventricles could be prematurely depolarized without terminating the tachycardia. Since full compensatory pauses may result following the introduction of VPDs during the tachycardia, entrance block to the re-entrant circuit must be present, assuming the impulses have reached the re-entrant site. This assumption was confirmed in one case in which we were able to record persistent activity within a ventricular aneurysm which was unaffected by stimulation.

Furthermore, VPDs resulting in biventricular capture followed by intervening sinus captures, and atrial pacing resulting in supraventricular complexes frequently failed to terminate ventricular tachycardia. The continuation of the tachycardia under these circumstances could be termed "concealed perpetuation." Supraventricular capture with continuation of the arrhythmia suggests that a small, electrocardiographically silent area of the ventricles forms the re-entrant circuit. If the re-entrant circuit were composed of a large area of the ventricles, supraventricular captures with identical morphology to sinus complexes would be impossible without terminating the arrhythmia. This implies that depolarization of the major part of the ventricles as well as the proximal conduction system should be considered a consequence of a more circumscribed process. The re-entrant circuit must therefore be localized to a very small area within the ventricles which is relatively protected.

Relationship to Prior Investigations

Our findings confirm those of Wells et al. that in almost all of our patients with recurrent sustained ventricular tachycardia, the arrhythmia could be reproducibly initiated and/or terminated. The failure of Denes et al. to initiate and terminate similarly defined ventricular tachycardia with reproducibility is not readily explained but may be due to differences in patient population or techniques of stimulation. Our studies show that the use of multiple intra-ventricular electrodes may allow one to analyze more accurately the mechanisms of initiation and termination of the ventricular tachycardia.

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Recurrent Sustained Ventricular Tachycardia

2. Endocardial Mapping

MARK E. JOSEPHSON, M.D., LEONARD N. HOROWITZ, M.D., ARDESHIR FARSHIDI, M.D., JOSEPH F. SPEAR, PH.D., JOHN A. KASTOR, M.D., AND E. NEIL MOORE, D.V.M., PH.D.

SUMMARY

Endocardial ventricular mapping of 21 ventricular tachycardias (VT) in 17 patients was performed using electrode catheters. Activation at multiple left and right ventricular sites was utilized to determine the site of origin of the VT. Eleven VT had a left bundle branch block pattern (VT-LBBBB) and 10 VT had right bundle branch block pattern (VT-RBBBB). In all VT-RBBBB the earliest site of activation was in the LV or septum. In VT-LBBBB the earliest site was RV (4/11), LV (5/11) and septum (2/11). All ventricular tachycardias with QRS < 140 msec arose in the septum. In patients with an aneurysm, the site of origin of ventricular tachycardia was always in the aneurysm. All VT-LBBBB arising from the left ventricle originated in an aneurysm involving the septum. QRS changes during ventricular tachycardia were associated with alterations in the pattern of ventricular activation without alteration of the site of origin. In three patients the site of origin predicted by endocardial ventricular mapping was confirmed intraoperatively by epicardial and/or endocardial mapping.

We conclude that endocardial ventricular mapping demonstrates the limitations of the surface electrocardiogram in localizing the site of origin of ventricular tachycardia. The method may provide important data upon which the surgical therapy of ventricular tachycardia is based.

THE SURGICAL APPROACH TO THE THERAPY of medically resistant ventricular tachycardia has continued to evolve over the past ten years. However, the success of ventricular aneurysmectomy and/or coronary artery bypass grafting in terminating this arrhythmia varies, and in many instances may be accompanied by a high surgical mortality.1-4 Recently, intraoperative epicardial mapping has been used as a surgical guide to localize more accurately the site of origin of the arrhythmia.5-7 In spite of this technique these surgical interventions for ventricular tachycardia are still not universally successful. The present report concerns the development of a new technique, ventricular endocardial mapping, which provides useful data that may improve the efficiency of these surgical interventions.

Methods and Materials

Seventeen patients with sustained recurrent ventricular tachycardia underwent ventricular endocardial mapping as a

From the Electrophysiology Laboratory, Hospital of the University of Pennsylvania, Cardiovascular Section, Department of Medicine, University of Pennsylvania School of Medicine, Philadelphia, Pennsylvania. Dr. Horowitz is the recipient of a Career Development Investigatorship award from the American Heart Association, Southeastern Pennsylvania Chapter.

Address for reprints: Mark E. Josephson, M.D., Director, Electrophysiology Laboratory, 666 White Building, Hospital of the University of Pennsylvania, 3400 Spruce Street, Philadelphia, Pennsylvania 19104.

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M E Josephson, L N Horowitz, A Farshidi and J A Kastor

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