


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 with 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" 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. In contrast, a recent publication by Denes et al. 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.

Recurrent Sustained Ventricular Tachycardia

1. Mechanisms

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Patients and Methods

Twenty-one patients with chronic sustained ventricular tachycardia were studied (tables 1 and 2). The patients included 4 females and 17 males ranging in age from 8 to 73 years. Seven of the 21 patients had no heart disease documented at cardiac catheterization. The most common cardiac abnormality noted in the remainder was coronary artery disease. Eight of the ten patients with coronary artery disease had clinical, roentgenographic, and/or angiographic evidence of left ventricular aneurysm.

The diagnosis of ventricular tachycardia was made using standard electrocardiographic criteria and confirmed in all cases by intracardiac recordings during the arrhythmia which demonstrated the absence of His deflections consistently preceding ventricular depolarization with H-V intervals equal to or greater than that of normal sinus rhythm. All patients had recurrent spontaneous episodes of ventricular tachycardia that were typically sustained for minutes to days and usually required pharmacologic or electrical termination. Each patient had at least four spontaneous episodes of their tachycardia(s). The resting electrocardiogram in sinus rhythm was normal in five patients and showed old myocardial infarction in ten patients. Intraventricular conduction disturbances were present in eight patients. No patient demonstrated a prolonged Q-T interval or pre-excitation during sinus rhythm and/or atrial pacing.
### Table 1. Clinical Data

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

Abbreviations: ASMI = anteroapical myocardial infarction; ASHD = atherosclerotic heart disease; AVB = atrioventricular block; CAA = coronary artery anomaly; CL = cycle length; CM = cardiomyopathy; F = female; HCVD = hypertensive cardiovascular disease; IACD = intracardiac 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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<th>Case</th>
<th>AH (msec)</th>
<th>HV (msec)</th>
<th>ERP-RV* (msec)</th>
<th>ERP-LV* (msec)</th>
<th>BBR (msec)</th>
<th>CL (mean) (msec)</th>
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<th>Axis°</th>
<th>Induced ventricular tachycardia</th>
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<tr>
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<tr>
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<td>185</td>
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<td>420</td>
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<td>150 (RV)</td>
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<td>80</td>
<td>55</td>
<td>260</td>
<td>210</td>
<td>+</td>
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<td>LBBB</td>
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<td>21</td>
<td>65</td>
<td>40</td>
<td>180</td>
<td>-</td>
<td>-</td>
<td>240</td>
<td>RBBB</td>
<td>-240</td>
<td>220 (RV)</td>
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</table>

* 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 sustained a myocardial infarction
within the three weeks prior to the study. All patients were
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
rhymth. 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. Quadripolar 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 tripollar
catheter was used to obtain the His bundle and local ven-
tricular 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**

\[ S_1 - S_1 = \text{basic paced cycle length.} \]

\[ S_1 - S_2 = \text{first and second premature stimuli delivered} \]

\[ S_1 - S_2 = \text{coupling interval from eighth beat of a basic} \]

\[ S_1 - S_2 = \text{drive cycle to the first premature stimulus.} \]

\[ V_1, V_2, V_3 = \text{ventricular depolarizations produced by} \]

\[ S_1, S_2, \text{and } S_3 \text{ respectively.} \]

Effective refractory period of the ventricles = the longest

\[ S_1 - S_2 \text{ which fails to evoke a ventricular depolariza-} \]

Tachycardia zone = range of \[ S_1 - S_2 \text{ or } S_2 - S_3 \text{ cou-} \]

pling intervals which results in ventricular tachycardia.

Bundle branch re-entry = the appearance of an extra,
nonstimulated ventricular depolarization in response to
\[ S_2 \text{ or } S_3 \text{ which is dependent upon attaining a} \]

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 ventricu-
lar 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 \[ S_1 - S_2 = 50 \text{ to 100 msec longer} \]

than the ventricular effective refractory period, and \[ S_2 - S_3 \text{ equal to the} \]

\[ S_1 - S_2 \text{ interval. } S_2 = S_3 \text{ was shortened, and when} \]

\[ S_3 \text{ failed to evoke a } V_3, S_1 - S_2 \text{ was decreased until} \]

\[ S_3 \text{ could evoke a response or ventricular tachycardia.} \]

This method was used until both \[ S_2 \text{ and } S_3 \text{ reached} \]

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 in-
troduced in an attempt to terminate the arrhythmia. The
extrastimuli were introduced from multiple sites in-
cluding 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 ventri-
cle, 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 tachycar-
dias 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 tachycar-
dia 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-
TABLE 3. Initiation and Termination of Ventricular Tachycardia

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<th>Mode of Termination</th>
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<tr>
<td>21</td>
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</tr>
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</table>

+ = successful. Patients in whom both 1 and 2 VPD are + had different tachycardias with different modes of initiation.

*Termination protocol not completed due to hemodynamic deterioration requiring cardioversion.

†Indicates more than one VT with similar initiation characteristics. Abbreviations: NA = not attempted; VPD = ventricular premature depolarization; VP = ventricular pacing.

ing, 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 VPD 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 refactoriness 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 (V_H2 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. Initiation of ventricular tachycardia during sinus rhythm and ventricular pacing (case 14). Analog records are organized from top to bottom: ECG leads 1, aVF, V_1, and electrogram from the right atrium (HRA). His bundle (HBE) and right ventricle (RV). In panel A ventricular tachycardia is initiated during sinus rhythm with one ventricular stimulus (S_1). T = time lines.](http://circ.ahajournals.org/)

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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

![Figure 2](http://circ.ahajournals.org/)

**Figure 2. Initiation of ventricular tachycardia by right or left ventricular stimulation (case 15).** Both panels are organized from top to bottom: ECG leads I, aVF, V1, and electrograms from the high right atrium (HRA), His bundle (HBE), right ventricular apex (RVA), left ventricular apex (LVA). In panel A, two right ventricular premature stimuli (S1, S2) are introduced after the eighth RV paced complex (S0), resulting in ventricular tachycardia. In panel B, two left ventricular stimuli (S3, S4) are delivered after the eighth LV paced complex (S0), resulting in ventricular tachycardia. Note that the coupling intervals of the premature stimuli are identical. Stimulus artifacts are indicated by small arrows.

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

**Figure 3. Dissociated His bundle deflections during ventricular tachycardia (case 2).** Both panels are organized similarly to previous figures. Ventricular tachycardia is present at a cycle length of 290 msec in both panels. On the left His potentials (H) appear simultaneous with the onset of the QRS and are therefore retrograde. On the right, recorded 10 sec later, no His potentials are noted.
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 (V₄) 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.
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.\textsuperscript{12, 13} 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;\textsuperscript{14} b) a direct effect of the electrical current on local dispersion of refractoriness;\textsuperscript{15} 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 (V\textsubscript{1}−V\textsubscript{5} or V\textsubscript{2}−V\textsubscript{5}) to the interval from the initiating VPD to the first complex of the ventricular tachycardia (V\textsubscript{2} or V\textsubscript{5}−V\textsubscript{7}) was frequently observed. This finding is analogous to the findings in patients with A-V nodal re-entrant tachycardia,\textsuperscript{16} 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\textsuperscript{17} 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.\textsuperscript{17} 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.\textsuperscript{18, 19} Recent studies in excised human ventricular aneurysms suggest similar mechanisms may be operative in man.\textsuperscript{20}

**Figure 6.** Termination of ventricular tachycardia by two ventricular stimuli (case 17). The analog records are organized from top to bottom: ECG leads 1, aVF, V\textsubscript{1}, 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\textsubscript{i}, 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\textsubscript{2}, is introduced at the identical coupling interval (S\textsubscript{i}−S\textsubscript{2} = 270 msec), both ventricles are captured and the tachycardia is terminated after another ventricular complex. The mechanism of this last complex is uncertain.

**Figure 7.** Ventricular capture without terminating ventricular tachycardia (case 16). Analog records of the electrocardiogram (1, aVF, and V\textsubscript{1}) 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.
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 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
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\(^1\)\(^2\)\(^,\)\(^3\) 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,\(^8\)\(^9\) 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.\(^8\) 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 resulted 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 Wellens\(^1\)\(^4\) 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.\(^2\) 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

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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-LBBB) and 10 VT had right bundle branch block pattern (VT-RBBB). In all VT-RBBB the earliest site of activation was in the LV or septum. In VT-LBBB 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-LBBB 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 aneurectomy 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.2,3 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 part of the electrophysiologic evaluation of their arrhythmia. The clinical data for these patients are listed in table 1. Eight patients had ischemic heart disease and seven of these patients had a clinical angiographically documented ventricular aneurysm. Twenty-one morphologically distinct ventricular tachycardias were studied in the 17 patients. Four patients demonstrated two ventricular tachycardia morphologies. Ten ventricular tachycardias showed a right bundle branch block morphology (R, qR, rsR', Rs in V1) and 11 demonstrated a left bundle branch block pattern (QS or rS in V1).

In each patient three to six electrode catheters were percutaneously introduced and positioned at the following endocardial sites: 1) high right atrium; 2) atrioventricular (A-V) junction at the point where the His bundle was recorded; 3) right ventricular apex; 4) coronary sinus; 5) right ventricular outflow tract; and 6) left ventricle. The left ventricular catheter was positioned using the retrograde arterial approach. The A-V junction and coronary sinus catheters, when used, remained fixed so that right ventricle activation at the atrioventricular junction and left ventricular activation along the atrioventricular groove were continuously recorded. In addition, the catheter in the right ventricular apex was left in a stable position whenever possible. One or more ventricular catheters were used as exploring electrodes. The mapping sites included the fixed sites and the midseptum, anterior wall, outflow tract, lateral inflow tract.
Recurrent sustained ventricular tachycardia. 1. Mechanisms.
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