Electrophysiological Studies in Patients with Chronic Recurrent Ventricular Tachycardia

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SUMMARY Seventeen consecutive patients with chronic recurrent ventricular tachycardia (VT) were studied in an attempt to delineate the reproducibility and mechanism of this arrhythmia. Six patients had nonsustained and 11 had sustained VT. The following electrophysiological techniques were utilized in an attempt to reproduce VT:
1) rapid atrial and ventricular pacing (17 pts); 2) atrial extrastimulus technique (17 pts); 3) ventricular extrastimulus technique (17 pts); 4) V1V2V3 stimulation technique (5 pts); 5) ventricular pacing from two or more sites (5 pts). Ventricular tachycardia was induced in six of 11 (54%) patients with sustained VT. However, in four there was only a single induction and only in the remaining two patients could VT be repetitively induced. In the latter two patients ventricular tachycardia was induced with both atrial and ventricular stimulation. Ventricular tachycardia could not be induced in any patient with nonsustained VT, although three had spontaneous episodes of ventricular tachycardia during study.

In conclusion, in the present series of patients with chronic recurrent VT, this rhythm could not be reproducibly induced in the majority of patients in the cardiac catheterization laboratory utilizing catheter stimulation techniques.

RECENT ELECTROPHYSIOLOGICAL STUDIES in patients with recurrent ventricular tachycardia have demonstrated duplication of the arrhythmia with critically timed electrical stimulation.1,8 All except one of the previous reports have been concerned with selected cases. Recently, Wells and Lie reported results of electrophysiological studies in a series of 36 patients with ventricular tachycardia, 20 of whom had chronic recurrent ventricular tachycardia. They noted that ventricular tachycardia could be induced with ventricular extrastimulus technique in 17 of the 20 patients with chronic recurrent ventricular tachycardia. Electrophysiological observations in these cases suggested that ventricular tachycardia reflected re-entrant mechanisms.

In the present study, we report the results of electrical stimulation utilizing several types of stimulation techniques in 17 consecutive cases of chronic recurrent ventricular tachycardia, in an attempt to further delineate the reproducibility of chronic recurrent ventricular tachycardia. We also attempted to further define mechanisms of this rhythm.

Methods
Patient Selection and Definitions

Seventeen consecutive patients undergoing electrophysiological study because of chronic recurrent ventricular tachy-
Ventricular Tachycardia

<table>
<thead>
<tr>
<th>QRS config during VT</th>
<th>Rates (beats/min)</th>
<th>Coupl. Int. (msec)</th>
<th>Duration</th>
<th>Type of VT</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 RBBB + LAD 180-210</td>
<td>N.O. 2 yrs</td>
<td>Sustained</td>
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<tr>
<td>2 RBBB + LAD 185</td>
<td>N.O. 2 yrs</td>
<td>Sustained</td>
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<tr>
<td>3 RBBB + RAD 185-250</td>
<td>N.O. 11 yrs</td>
<td>Sustained</td>
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<tr>
<td>4 RBBB + LAD 160</td>
<td>N.O. 1 mo</td>
<td>Sustained</td>
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<tr>
<td>5 RBBB + LAD 185</td>
<td>N.O. 4.2 yrs</td>
<td>Sustained</td>
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<tr>
<td>6 RBBB + LAD 130</td>
<td>N.O.</td>
<td>Sustained</td>
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<tr>
<td>7 RBBB + LAD 150-180</td>
<td>N.O. 3 yrs</td>
<td>Sustained</td>
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<tr>
<td>8 RBBB + LAD 185</td>
<td>N.O. 2 mo</td>
<td>Sustained</td>
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<tr>
<td>9 RBBB + LAD 130-180</td>
<td>N.O. 1 yr</td>
<td>Sustained</td>
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<tr>
<td>10 LBBB</td>
<td>130-180 N.O. 3 yrs</td>
<td>Sustained</td>
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<tr>
<td>11 LBBB</td>
<td>150 N.O. 3 yrs</td>
<td>Sustained</td>
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<tr>
<td>12 LBBB</td>
<td>150 480 3 yrs</td>
<td>Non-sustained</td>
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<tr>
<td>13 LBBB</td>
<td>130-170 420-480</td>
<td>Non-sustained</td>
<td></td>
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<tr>
<td>14 LBBB</td>
<td>210 360 1.5 yrs</td>
<td>Non-sustained</td>
<td></td>
<td></td>
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<tr>
<td>15 LBBB</td>
<td>150 480 1 yr</td>
<td>Non-sustained</td>
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<tr>
<td>16 RBBB + LAD 180</td>
<td>370 3 yrs</td>
<td>Non-sustained</td>
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<tr>
<td>17 LBBB</td>
<td>110-150 510-580 6 mo</td>
<td>Non-sustained</td>
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Abbreviations: Coupl. Int. = coupling interval of initiating beat; LBBB = Right bundle branch block; LAD = Left axis deviation; RAD = Right axis deviation; N.O. = Not observed; and VT = Ventricular tachycardia.

Cardiac were subjects of the present study. All patients were seen between March 1973 and October 1975. Ventricular tachycardia was diagnosed using standard electrocardiographic criteria, including the presence of wide QRS tachycardia (three or more beats), usually with A-V dissociation, and often with captures and fusions. The diagnosis of ventricular tachycardia was confirmed in all equivocal cases with His bundle recordings. "Recurrent" ventricular tachycardia, as defined in this study, reflected multiple episodes (at least three discrete episodes). In almost all cases, the number of episodes far exceeded this minimum number. "Chronicity" was defined as the occurrence of multiple episodes for at least one month. Patients with multifocal ventricular tachycardia and ventricular tachycardia associated with acute myocardial infarction or drug intoxication were excluded.

 Characteristics of Ventricular Tachycardia (table 1)

QRS morphology of ventricular tachycardia was of right bundle branch block pattern (lead V) in nine patients, suggesting left ventricular tachycardia and left bundle
branch block pattern in seven patients, suggesting right ventricular tachycardia. One patient had right and left ventricular tachycardia on different occasions. The observed rates of ventricular tachycardia varied from 110 to 250 beats/min. Initiation of ventricular tachycardia during sinus rhythm was repetitively observed in six patients. In all, the QRS morphology of the initiating premature beats was identical to subsequent beats of the tachycardia. The coupling interval of the first beat was relatively fixed in four and varied in two (table I). The onset of ventricular tachycardia was not observed in 11 patients.

Two types of chronic recurrent ventricular tachycardia could be distinguished. One type was characterized by frequent, short, self-terminating episodes with frequent premature beats between episodes of ventricular tachycardia. Occasionally the episodes were prolonged and interrupted by sinus beats only at long intervals. This type of ventricular tachycardia resembled the type 2 tachycardia of Gallavardin and is classified in the present study as “non-sustained” ventricular tachycardia. The other type was characterized by prolonged paroxysms of sustained ventricular tachycardia usually necessitating pharmacologic or electrical termination. In these patients, episodes of tachycardia were separated by periods of sinus rhythm as long as six months. This type of paroxysmal ventricular tachycardia was similar to the type 4 tachycardia of Gallavardin and is classified in the present study as “sustained” ventricular tachycardia.

### Characteristics of Patients (table 2)

Patients ages ranged from 17 to 67 with a mean of 42 years. There were 11 males and 6 females. Individual ages and diagnosis are listed in table 2. Ten patients complained of palpitations, four had dyspnea, two had syncope, and one had dizziness. The resting electrocardiogram showed old myocardial infarction in five, intraventricular conduction defects in eight, and was normal in three.

One patient (8) had intermittent second degree A-V block and a demand right ventricular pacemaker. Nine of the patients were specifically referred to our laboratory from outside physicians for evaluation of ventricular tachycardia. The remainder were detected in our inpatient or outpatient services.

### Electrophysiological Studies

By use of the Seldinger technique, three electrode catheters were passed under local anesthesia and positioned in the heart chambers. A quadripolar catheter was placed in the high right atrium for atrial pacing (distal poles) and record-

<table>
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<th>Table 3. Electrophysiological Studies</th>
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<tr>
<td>AP (beats/ min.)</td>
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</tr>
<tr>
<td>1</td>
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<td>2</td>
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<td>17</td>
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*Sinus rhythm.

Abbreviations: AP = atrial pacing; VP = ventricular pacing; AEST = atrial extrastimulus technique; VEST = ventricular extra-stimulus technique; CL = cycle length; RVO = right ventricular output; RVI = right ventricular inflow; RVA = right ventricular age; LVA = left ventricular anterior; LVP = left ventricular posterior; VF = ventricular tachycardia; rep = reproducible; obs = observed.
ing (proximal poles). A quadripolar catheter was placed in the right ventricle for ventricular pacing and sensing. In five patients, the ventricular catheter was positioned under fluoroscopic observation at three different right ventricular sites: 1) right ventricular apex (RVA); 2) right ventricular inflow tract (RVIL); 3) right ventricular outflow tract (ROV). In one patient a pacing catheter was placed in the left ventricle, through a brachial artery cutdown and positioned in the anterior and posterior left ventricle. A tripolar catheter was positioned across the tricuspid valve for His bundle recording. Four electrocardiographic leads (I, II, III and V,), a high right atrial electrogram, and His bundle electrograms were recorded. All oral medication was discontinued 48–72 hours prior to the study. Ten of the patients were not on any antiarrhythmic drug therapy for at least a week prior to study. Informed consent was obtained in all patients.

The following protocol of investigation was utilized in an attempt to replicate ventricular tachycardia (fig. 1 and table 3): Incremental atrial pacing (maximum rates of 130 to 200 beats/min) with sudden cessation in all 17 patients (fig. 1A). Incremental ventricular pacing with sudden cessation (maximal rates of 110 to 200 beats/min) in 16 of 17 patients (fig. 1B). Atrial extrastimulus testing in all 17 patients (in four at only a single cycle length and in 13 at two or more cycle lengths [fig. 2C]). Ventricular extrastimulus testing in all 17 patients (in nine patients at only a single cycle length, in eight patients at two or more cycle lengths) and in five patients at multiple ventricular sites, including the left ventricle in one (fig. 1D). V1V2V3 stimulation, utilizing a V1-V2 just greater than the ventricular refractory period, and scanning with V3, was utilized in 5 of the 17 patients (fig. 1E).

Results

Intervals and Refractory Periods

Atrioventricular nodal (A-H) and His Purkinje (H-V) conduction times were normal in 13 of the 17 patients. Four patients had prolonged H-V intervals (> 55 msec) and one had a prolonged A-H interval (> 140 msec). Atrioventricular nodal functional refractory periods varied from 350 to 505 msec and were normal in all. Ventricular refractory periods varied from 200 to 430 msec. Shortening of cycle length decreased ventricular refractory periods appropriately.

Induction of Ventricular Tachycardia (table 3)

Ventricular tachycardia could be repetitively induced in only two of the 17 patients. In the first patient (9) ventricular tachycardia was induced following cessation of atrial pacing at 130 beats/min, following cessation of ventricular pacing at 150/min (fig. 2A), with atrial extrastimulus technique (cycle length of 600 msec) at V1-V2 intervals between 380 and 360 msec (fig. 2B), and with ventricular coupled pacing (cycle length of 600 msec) at V1-V2 intervals between 400 and 220 msec (fig. 2, panel C). The effective refractory period of the ventricular muscle was the inner limit of the echo zone. Ventricular tachycardia was not induced following cessation of atrial pacing at rates below 130 beats/min or following cessation of ventricular pacing at rates less than 150 beats/min. The coupling interval of the first beat of ventricular tachycardia varied from 465 to 575 msec. This variation could not be explained and did not relate to the site of stimulation (atrial or ventricular), the cycle length, or the prematurity of the pacing stimuli.

In the second patient (10) ventricular tachycardia was induced following cessation of atrial pacing at 130 beats/min, following cessation of ventricular pacing at 150/min, and with ventricular extrastimulus testing (cycle length of 600 msec) at V1-V2 coupling intervals between 250 and 230 msec, and with V1V2V3 technique.

In four patients, (2,3,7,8) only a single episode of ventricular tachycardia could be induced, this induction depending upon a fortuitous set of circumstances. In one of these patients (2), induction of ventricular tachycardia occurred during atrial pacing at 170 beats/min (fig. 3A and B). Induction of ventricular tachycardia seemed to be dependent upon the occurrence of functional right bundle branch block with left anterior hemiblock, which only occurred once during the study (fig. 3B). Aberrant conduction occurred in the second conducted beat following a pause due to block above H (not shown). This aberrantly conducted beat showed right bundle branch morphology with normal QRS axis. The subsequently conducted paced beat also showed similar functional block. The third conducted beat following the pause showed additional left axis deviation similar to the QRS complexes shown in figure 3, panel B. Following cessation of pacing, the ventricular tachycardia became manifest with QRS configuration of right bundle branch block and left axis deviation. The H-V interval of the last two conducted paced beats was slightly shorter as compared to the H-V during normal conduction (panels 3A and 3B) and the width of the QRS showing right bundle branch block of conducted beats was narrower than those of the tachycardia. These findings suggested that the paced beats reflected fusion QRS complexes between an induced left ventricular tachycardia and the conducted supraventricular beats. Repeated atrial pacing at 170 beats/min without right bundle branch block and left anterior hemiblock aberrancy failed to induce the tachycardia (fig. 3A). Ventricular pacing at 170 beats/min was not performed. The re-entrant pathways for this tachycardia could have involved the divisions of the left bundle branch. However, the occurrence of re-entry within the specialized conduction system was not proven by the above findings. With coupled ventricular pacing, a zone of ventricular echoes with QRS configuration similar to the ventricular tachycardia was also induced (fig. 3C).

In the second patient (8), a paced premature ventricular beat induced a short run of ventricular tachycardia with QRS configuration similar to the paced beat, which was then followed by a short run of ventricular tachycardia (4 beats) with QRS configuration identical to the patient's spontaneous tachycardia. The third patient (3) had ventricular tachycardia induced during ventricular pacing at a cycle length of 580 msec, when a pacing spike fell on a spontaneous premature beat. In this patient coupled ventricular pacing at cycle length of 550 msec and V1V2V3 technique subsequently during the study failed to induce ventricular tachycardia. In the fourth patient (7) a single episode of ventricular tachycardia was induced during ventricular pacing, the induction not being recorded.

In 11 patients, ventricular tachycardia could not be induced by any of the electrophysiological interventions (table 3). It is noteworthy that three of these 11, (12, 13, 17) had spontaneous self-induced episodes of ventricular tachy-
Ventricular tachycardia during study (fig. 4), despite our inability to induce ventricular tachycardia with electrophysiological testing. In these patients, the initiating beat of the tachycardia was identical to subsequent beats (fig. 4). In eight patients, those who had no spontaneous onset or electrical induction, the tachycardia was not documented during the study. The following interventions were unsuccessful in these 11 patients: incremental atrial pacing in all; incremental ventricular pacing in ten of 11; atrial extrastimulus testing in all; ventricular extrastimulus testing in all; \( V_1-V_2V_3 \) testing in three; and ventricular extrastimulus testing at multiple ventricular sites in three (table 3).

A ventricular echo zone (one or more beats) with QRS configuration similar to the paced beats was found in three patients (4, 8, 13). A zone of ventricular echoes with QRS configuration similar to previous documented ventricular tachycardias was found in cases 2, 8, 9, 10, in all of whom ventricular tachycardias could be electrically induced.

**Termination of Ventricular Tachycardia**

In six of the nine patients who had ventricular tachycardia during the study, the termination of the tachycardia was spontaneous. In one patient (3) the tachycardia was terminated by rapid ventricular pacing. In another patient (9) the tachycardia could be terminated by two timed ventricular beats in sequence (fig. 2D). In the third patient (10) ven-
tachycardia could be interrupted by single premature ventricular beat.

Comparison of Patients with Nonsustained and Sustained Ventricular Tachycardia

In the group of patients with nonsustained ventricular tachycardia (cases 12 to 17), the arrhythmia could not be reproduced in any of the cases. In the group of patients with sustained ventricular tachycardia, (cases 1 to 11), ventricular tachycardia could be induced in the catheterization laboratory in six of the 11 patients (54%). The difference between groups in regard to inducibility of ventricular tachycardia could not be explained by noting the etiology of heart disease, the site of origin of ventricular tachycardia, or the stimulation techniques utilized during electrophysiological studies (tables 1, 2, 3).

Discussion

Recent studies in patients with known ventricular tachycardia have demonstrated that this arrhythmia could be reproduced in the catheterization laboratory with electrical stimulation of the heart. Wellens et al. published a series of articles on ventricular tachycardia studied by electrical stimulation. Their experience has been summarized in a recent publication. Since January 1971, they studied 36 patients with ventricular tachycardia. These patients were divided into four categories, those with chronic recurrent ventricular tachycardia (group 1), those with ventricular

FIGURE 3. Induction of ventricular tachycardia (patient 2). Panel A) Following cessation of atrial pacing at 170 beats/min ventricular tachycardia is not induced. Note that the atrial paced beats have narrow QRS complexes. Panel B) Atrial pacing at 170 beats/min in the presence with functional right bundle branch block and left anterior hemiblock. The H potential of the conducted beats is obliterated by the stimulus artifact except for the last two beats. Ventricular tachycardia is induced during atrial pacing. Panel C) Induction of ventricular echoes, with QRS configuration identical to the QRS of the ventricular tachycardia, with ventricular extrastimulus testing.

FIGURE 4. Spontaneous onset of ventricular tachycardia. Note that the initiating beat is similar to the subsequent beats. The H potential is buried in the ventricular complex.
tachycardia developing within 24 hours following an acute myocardial infarction (group 2), and those who developed ventricular tachycardia two to four weeks after an acute myocardial infarction (group 3) and finally patients with Q-T prolongation syndrome (group 4). There were 20 patients in group 1. In 17 of these 20, ventricular tachycardia could be initiated with the ventricular extrastimulus technique. In 14 patients, this depended upon a single premature beat and in three, two ventricular premature beats. In the patients of the remaining groups 2, 3 and 4 the tachycardia could not be initiated with ventricular pacing. Wellens concluded that ventricular tachycardia was probably re-entrant in the group with chronic recurrent tachycardia while in the group with acute and recent myocardial infarction, abnormal automaticity was the most likely mechanism responsible for arrhythmias. The site of re-entry could not be defined in their study.

Fontaine et al.6 studied two patients with known recurrent ventricular tachycardia in the operating room. The tachycardia could be induced and terminated by single timed ventricular extrastimuli in one case, and by short bursts of stimuli at 300 beats/min in the second patient. Epicardial mapping in one of the patients showed a delayed epicardial potential. A single ventriculotomy based on the epicardial map was successful in terminating the attacks of ventricular tachycardia. Both the stimulation and epicardial mapping studies favored re-entry as a mechanism of tachycardia, although the exact site of re-entry within the ventricle was not defined. Spurrell et al.6,7 reported six patients with ventricular tachycardia. In four of these patients, the tachycardia could be induced by single timed ventricular premature beats; in one patient, by two premature ventricular beats in sequence; and in one, electrical stimulation could not be studied since the tachycardia was reinitiated repeatedly by spontaneously occurring premature ventricular beats. In three of these patients, timed ventricular paced beats introduced during the tachycardia produced marked changes in QRS morphology (change in axis and/or bundle branch block pattern) accompanied by changes in the rate of the tachycardia. These findings suggested that the tachycardia was re-entrant, utilizing the bundle branches and/or fascicles for re-entry. Two other patients underwent epicardial mapping studies during sinus rhythm, both patients showing areas of slow activation in the epicardial region where the earliest ventricular activation was found during ventricular tachycardia. From the epicardial mapping studies during the tachycardia, they concluded that part of re-entrant circuit probably involved the bundle branches.6 Guerot et al.8 reported a case where the initiation of ventricular tachycardia by a single atrial premature beat was dependent upon the occurrence of the functional right bundle branch block of the conducted atrial beat, suggesting re-entry within bundle branches. Recently, Touboul et al.8 reported a patient in whom ventricular tachycardia could be induced by rapid atrial pacing and single timed atrial and ventricular premature beats. Aberrant conduction of the supraventricular beats was not a necessary condition for induction of ventricular tachycardia. It was suggested that the mechanism of the tachycardia was re-entrant, although the site could not be defined.

In the present series, we report 17 consecutive patients with chronic recurrent ventricular tachycardia. These patients had electrophysiological studies to determine the reproducibility of ventricular tachycardia in the catheterization laboratory. In none of six patients with non-sustained chronic recurrent ventricular tachycardia, could tachycardia be induced with electrical stimulation despite spontaneous episodes of tachycardia occurring during the study in three of the six patients. This finding suggested that a mechanism other than re-entry was responsible for ventricular tachycardia in these patients.

In the six of 11 (54%) patients with sustained ventricular tachycardia, tachycardia could be induced during electrophysiological study. In two patients, initiation of ventricular tachycardia could be reproducibly induced with rapid atrial and ventricular as well as timed single atrial and ventricular pacing, suggesting re-entrant mechanism. The exact site of re-entry within the ventricle could not be defined.

In most patients with ventricular tachycardia, due to limitations of recording techniques, re-entrant pathways cannot be localized nor can critical conduction delays be defined. The diagnosis of re-entry is only suggested and not conclusively proved, when ventricular tachycardia is initiated and terminated by single premature stimuli. Furthermore, it has been recently shown in canine Purkinje fibers exposed to sodium-free solution and ouabain that automaticity could also be initiated by a single propagated action potential.10 These findings, if applicable to human cardiac tissues under physiological conditions, would further complicate the diagnosis of re-entry with electrical stimulation studies.

Our two patients with repetitive induction and termination of tachycardia were similar to previously described cases, where tachycardia was felt to be re-entrant. It is interesting to note that in both of our patients, ventricular tachycardia could be initiated with both atrial and ventricular stimulation. This is an unusual occurrence, since A-V nodal delay usually prevents the achievement of appropriately short ventricular coupling intervals. Induction of ventricular tachycardia was dependent upon achievement of a critical V-V (QRS to QRS), interval which was relatively long in these two cases. This finding is consistent with the concept of re-entry since both unidirectional block and slow conduction could occur during rapid pacing, thus making the induction of re-entry dependent on a critical cycle length. Clinical counterpart to these findings could be cases of ventricular tachycardia that are "self induced" and are dependent on a critical V-V interval that is achieved during sinus rhythm.

It should also be emphasized that re-entrant ventricular tachycardia might relate to critical slow or fast rates, since either could predispose to re-entry by potentiating areas of unidirectional block.10,11 This could not be systematically examined in the present study since rates slower than sinus rhythm could not be tested, and fast rates were limited to maximum rates between 110 and 200 beats/minute.

In four of our patients, only a single episode of ventricular tachycardia was induced during the study, depending upon fortuitous circumstances. In one of these, tachycardia induction seemed to be dependent upon development of a functional intraventricular block, suggesting fascicular re-entry.

In five of our eleven patients with sustained ventricular tachycardia, tachycardia could not be reproduced during electrophysiological studies. Several hypotheses have to be
considered in order to explain this failure of induction. If spontaneous ventricular tachycardia was not re-entrant in these cases, then the failure of induction would be expected. If ventricular tachycardia was re-entrant, the lack of reproduction could reflect inadequate stimulation techniques. In the latter case, several factors singly or in combination could be involved. For example, we may not have tested at the critical cycle length at which unidirectional block of a premature impulse with achievement of critical conduction delay could occur. With \( V_1V_2V_3 \) stimulation, utilization of different \( V_1-V_3 \) intervals might allow replication of ventricular tachycardia with \( V_3 \). It is also possible that we failed to achieve a critical coupling interval because of distance from the site of re-entry, and that stimulation at sites closer to the origin of ventricular tachycardia would have allowed reproduction. \( V_1 \) It is also possible that the electrophysiological characteristics or even the presence of an area of unidirectional block and conduction delay are dependent upon local conditions (\( \text{pH} \), potassium, anoxia, etc.) which could change from day to day.

Although we have attempted to circumvent some of these difficulties by utilizing several cycle lengths and multiple pacing sites in these patients, the possibility that the above factors influences our results is likely.

In conclusion, our results suggest that most chronic recurrent ventricular tachycardias are not reproducible in the catheterization laboratory, utilizing the techniques in this study. We recognize that this conclusion is based on limited data and differs from the recent report of Wellens and Lie. The few cases that were reproducible came from the group of patients with sustained chronic recurrent ventricular tachycardia. In the group of patients with chronic recurrent nonsustained tachycardia this could not be reproduced with electrical stimulation. Similar inability to reproduce ventricular tachycardia has been previously described in patients with acute or recent myocardial infarction as well as in those with prolonged Q-T interval.

Our results also contrast with similar studies of patients with paroxysmal supraventricular tachycardia, where reproducibility is usually the case, and electrophysiological observations strongly suggest one or another type of re-entry. We believe that our study indicates a considerable lack of knowledge concerning the electrophysiological mechanisms of ventricular tachycardia in man.

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