PATHOPHYSIOLOGY AND NATURAL HISTORY
VENTRICULAR ARRHYTHMIA

Idiopathic sustained left ventricular tachycardia: clinical and electrophysiologic characteristics

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ABSTRACT  Electrophysiologic studies were performed in 16 patients 11 to 45 years old (mean 33 years) with idiopathic sustained (lasting more than 5 min) ventricular tachycardia (VT) originating from the left ventricle. Endocardial mapping during VT showed that the earliest site of activation was at the apical inferior portion of the left ventricle in 14 patients whose QRS morphology during VT showed a right bundle branch block pattern and left-axis deviation, but at the apical anterosuperior portion of the left ventricle in two patients whose QRS morphology during VT showed a right bundle branch block and right-axis deviation. Single programmed ventricular stimulation induced VT in 13 patients, and rapid ventricular pacing induced VT in the remaining three patients. Rapid ventricular pacing terminated VT in all patients. The relationship between the coupling interval and the echo interval was inverse in all eight patients with a wide VT inducible zone. Entrainment was recognized in three of six patients. The initiation of VT by constant pacing depended on the number of pacing beats but not the duration of pacing in four patients tested. Intravenous verapamil terminated the VT in 13 of 14 patients. Long-term oral verapamil was also effective in all five patients who required long-term oral therapy for their symptoms associated with VT. In conclusion (1) idiopathic left ventricular tachycardia has unique electrocardiographic, electrophysiologic, and electropharmacological properties, (2) the electrophysiologic characteristics suggest that the mechanism is reentry, and (3) verapamil is effective in both the short- and long-term treatment of VT.


IDIOPATHIC sustained ventricular tachycardia (VT) has recently been characterized by its QRS morphology during VT (right bundle branch block and left axis deviation) and its responsiveness to verapamil. The QRS configuration of the tachycardia suggests that it originates from the Purkinje fiber network of the left posterior fascicle. However, extensive endocardial mapping has not been done to confirm the origin of this VT.

Reentry has been postulated as the mechanism for the VT on the basis that it can be induced and terminated by extrastimuli. Recent electrophysiologic studies in animals have shown that tachycardia due to triggered activity can also be induced and terminated by extrastimuli. Thus, further evidence is required to clarify its mechanism.

The purpose of this study was to investigate in consecutive patients with sustained VT originating in the left ventricle and without overt evidence of organic heart disease (1) the origin of VT, (2) the electrophysiologic characteristics of VT, (3) the efficacy of antiarrhythmic drugs, and (4) long-term clinical follow-up.

Methods

From January 1979 through December 1986, 16 patients with recurrent sustained VT of left ventricular origin, but without clinical evidence of heart disease, were studied at the National Cardiovascular Center of Osaka, Japan. The mean age of the patients at the time of our evaluation was 31.3 ± 14.0 years. Sustained VT was defined as protracted paroxysmal runs of VT lasting longer than 5 min and usually requiring pharmacologic or electrical intervention. The absence of organic heart disease was diagnosed by: (1) normal cardiac examination, (2) normal resting electrocardiogram, (3) normal chest x-ray, (4) lack of significant ST depression or ST elevation during or after a submaximal treadmill exercise test, (5) a normal echocardiogram (no structural cardiac abnormalities, no enlargement of the cardiac chambers, normal left ventricular wall thickness, and normal left ventricular wall motion), and (6) a normal radio-nuclide angiogram (no enlargement of the cardiac chambers and normal left and right ejection fraction). In 10 patients, left ventricular angiography, right ventricular angiography, and coronary arteriography were performed to establish that no struc-
tural heart disease was present. The left origin of the VT was
determined by ventricular endocardial mapping during VT.

**Electrophysiologic studies.** After giving informed written
consent patients underwent electrophysiologic studies per-
formed while they were in the postabsorptive state. Antiarrhyth-
mic medications were discontinued at least 3 days before the
studies. His bundle electrograms were recorded by a standard
technique. A bipolar electrode catheter was passed into the high
right atrium for stimulation of the atrium. A second bipolar
electrode catheter with an interelectrode distance of 10 mm was
introduced into the right ventricle for stimulation or recording
from the right ventricle. A third bipolar electocatheter with a
10 mm interelectrode distance of introduced into the left ven-
tricle for stimulation of or recording from the left ventricle.
Surface electrophysiologic leads (I, II, V1, and V3) and intra-
cardiac electrograms were simultaneously recorded on a mul-
tichannel photographic recorder (Simens-Elma Mingograf 82)
at a paper speed of 100 mm/sec. A His potential during VT was
confirmed by recording the potential just before the initia-
tion and immediately after the termination of VT while fixing
the electrode catheter. Electrical stimuli of 2 msec duration and
approximately twice the diastolic threshold were provided by a
programmable digital stimulator (Fukuda Denshi Cardiac Stim-
ulator; BC-A).

Atrial pacing, programmed ventricular extrastimulation, and
ventricular pacing were performed for induction of VT. Atrial
pacing was performed from the right atrium. The duration of
stimulation was 30 sec. Stimulation was performed selecting an
initial rate just above the spontaneous sinus rate. The rate was
increased by 10 beats/min until atrioventricular block occurred.

During programmed ventricular extrastimulation, the ventri-
cle was paced at two different cycle lengths (600 and 400 msec).
If a single ventricular extrastimuli failed to elicit VT, timed
double ventricular extrastimuli were delivered until VT was
provoked or until all extrastimuli failed to evoke ventricular
responses. If VT was not induced with the extrastimuli, incre-
mental ventricular pacing at cycle lengths of 400 to 240 msec
for periods of 5 to 30 sec was performed. Stimulation was first
performed at the right ventricular apex, but when VT was not
induced, the same stimulation protocols were repeated at the
right ventricular outflow tract. When VT was induced neither by
stimulation at the right ventricular apex nor the right ventricular
outflow tract, the protocols were repeated at the left ventricular
apex. The tachycardia was considered inducible only when the
induced ventricular tachycardia replicated the spontaneous
ventricular tachycardia in both morphologic characteristics and
rates.

The site of the earliest ventricular activation of the VT was
determined by endocardial mapping during VT of eight sites in
the right ventricle and 12 in the left ventricle by the technique
of Josephson et al.5 Pace-mapping was also performed from the
sites of earliest activation in eight patients. As to the nomen-
clature of left ventricular subdivisions, we followed the rec-
ommendations of the Committee on Nomenclature of Myocar-
dial Wall Segments of the International Society of Computerized
Electrocardiography.6

The relationship between the coupling interval (the interval
between the last beat of basic cycle length and the premature
beat) and the echo interval (the interval between the premature
beat and the first beat of the VT) was determined in eight patients
in whom a wide range of single ventricular stimuli initiated the
VT (patients 1, 4, 6, 8, 9, 11, 14, and 16). The presence of
progressive fusion was assessed by pacing from the right ven-
tricle during VT in six patients (patients 1, 8, 9, 14, 15, and 16).
The presence of fragmentation or delayed potentials during
normal sinus rhythm was examined by recording the electrogram
from eight sites in the right ventricle and 12 sites in the left
ventricle. In four patients (patients 1, 3, 4, and 5) in whom rapid
pacing consistently induced VT the relationship between the
number of pacing beats and the induction of VT was examined.
A pacing cycle length that consistently induced VT was used.
The pacing was started at 2 beats and was increased in incre-
ments of 1 beat up to 14 beats and repeated several times to
determine whether the induction of VT was dependent on the
number of pacing beats.

**Antiarrhythmic drugs.** The termination of VT with drugs was
attempted by the intravenous administration of one or more of
the following agents on different occasions during spontaneous
or induced VT. The interval between testing various drugs was
at least 72 hr to avoid overlap of pharmacologic effects. (1)
ajmaline, 50 mg, over 5 min; (2) lidocaine, 100 mg, over 30 sec;
(3) propranolol, 10 mg, over 5 min; and (4) verapamil, 10 mg,
over 5 min.

Antiarrhythmic drugs were considered effective when they
terminated sustained VT. Five patients (patients 4, 5, 6, 9, and
12) were placed on long-term oral verapamil, 240 mg/day, for
troublesome symptoms associated with VT. The efficacy of
long-term oral therapy was assessed by clinical history and Holter
monitoring.

**Results**

**Characteristics of VT.** The clinical and electrophys-
ologic characteristics of all patients are presented in
table 1. The QRS morphology during VT was a right
bundle branch block pattern, defined as having the
wide terminal R or R' in V1, in all patients. The
electrical axis during VT was leftward in 14 patients
(figure 1, A) and rightward in two patients (figure 1, B).

**Endocardial mapping.** During normal sinus rhythm,
fragmentation or delayed potentials were recognized in
none of the 16 patients. The site of earliest ventricular
activation of the VT could be determined by endocar-
dial mapping in all 16 patients (figure 2). Fragmentation preceded ventricular activation in none of the patients and the earliest ventricular activation during VT coincided with or was within 10 msec of QRS onset in all patients (figure 3). The earliest activations were at the apical inferior site of the left ventricle in 14 patients whose QRS morphology during VT showed left-axis deviation (patients 1 to 13 and 16) and at the apical anterosuperior site in two patients whose QRS morphology during VT showed right axis deviation (patients 14 and 15). Pacing from the site of earliest activation, performed in eight patients, produced an identical QRS configuration to that of VT in all of them (figure 4). A His potential was recorded during VT in 10 patients. It occurred 20 to 30 msec after the earliest ventricular activation in all of them (figure 5). Ventriculoatrial conduction was present during tachycardia in two patients (patients 8 and 13), but it could be interrupted by carotid sinus pressure without terminating the tachycardia in both of them.

**Electrophysiologic characteristics of VT.** The electrophysiologic characteristics of VT in all patients are presented in table 2. Atrial pacing induced VT in four of 16 patients. A single ventricular extrastimulus induced VT in 13 of 16 patients. Rapid ventricular pacing was required to induce VT in three patients; this was accomplished from the right ventricular apex in one patient and from the left ventricle in two patients. Nonclinical VT that was not electrocardiographically identical to that occurring spontaneously was not observed in any patient.

Of the 13 patients in whom the single ventricular extrastimuli induced VT, eight patients had a wide zone of coupling intervals during which VT could be induced. In these patients, the relationship between the premature coupling interval (the interval between the last beat of the basic cycle length and the premature beat) and the echo interval (the interval between the premature beat and the first beat of the VT) was determined. The relationship was inverse in all of them. A typical example is shown in figure 6 (patient 9).

The relationship between the number of pacing beats and the induction of VT was examined in four of 16 patients (Nos. 1, 3, 4, and 5). In patient 1, the VT was induced with a greater frequency by 4, 7, 8, 12, and 13 pacing beats than by 3, 5, 6, 9, or 10 beats (figures 7 and 8). Similarly, the induction of VT was closely related to a certain number of pacing beats at a critical

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**FIGURE 1.** A, Electrocardiogram obtained during VT in patient 1. Note that the QRS configuration is a right bundle branch block pattern with left-axis deviation. B, Electrocardiograms obtained during VT in patient 14. Note that the QRS configuration is a right bundle branch block pattern with right-axis deviation.
FIGURE 2. The site of the earliest ventricular activation of VT (indicated by closed circles) in all patients. The left ventricle is subdivided into four portions: anteroseptal, anterosuperior, inferior, and posterolateral. The left ventricle is further subdivided into three regions from apex to base: apical, middle, and basal. Note that the VT originated from the apical inferior region in 14 patients, and from the apical anterosuperior region in two patients.

pacing rate in the remaining three patients examined.

Progressive fusion was demonstrated in three of six patients. A representative example is shown in figure 9, (patient 16).

Rapid pacing from the right ventricular apex terminated VT in all 16 patients.

Effective drugs for the termination of VT. The drugs found to be effective in terminating VT in all patients are listed in table 3. Lidocaine terminated VT in two patients. Ajmaline terminated VT in 13 of 15 patients. Verapamil terminated VT in 13 of 14 patients.

Long-term prophylactic effects of verapamil. Of 13 patients in whom verapamil effectively terminated VT, five (Nos. 4, 5, 6, 9, and 12) required long-term oral therapy for their troublesome symptoms associated with VT. They were placed on 240 mg/day oral verapamil and were followed for 1 to 6 years (mean 2.8 years). One patient (patient 4) has been free of symptomatic VT. In the remaining four patients, the VT became nonsustained and patients improved clinically.

Discussion

Localization of VT. Most VT originating in the left ventricle in adults is associated with ischemic heart disease, in contrast to VT originating in the right ventricle, which has been recognized in patients with right ventricular dysplasia, postsurgical congenital heart disease, and sometimes in patients without organic heart disease. However, recurrent sustained VT originating in the left ventricle in patients without detectable heart disease has been recognized for several years. Usually, this idiopathic left ventricular tachycardia is characterized by the configuration of left-axis deviation in addition to right bundle branch block. Belhassen et al.1 and Denes et al.8 have reported VT with such morphologic characteristics. German et al.3 reported a similar VT in 10 patients without apparent organic heart disease and found that the earliest ventricular activity during VT occurred at the region of the left ventricle.

FIGURE 3. Endocardial mapping during VT in patient 1. Surface leads I, II, V1, and V6 are recorded with multiple time-aligned bipolar endocardial recordings. Note that the earliest endocardial activity is observed at the apical inferior site of the left ventricle.
configuration of right bundle branch block and right-axis deviation originates within the Purkinje network of the anterior division of the left bundle block.

Electrophysiologic characteristics of VT. Both reentry and triggered activity have been postulated as possible mechanisms for the chronic sustained VT in patients without apparent organic heart disease. German et al. suggested that the mechanism responsible for the VT was reentry and that a reentrant circuit was located in the Purkinje fiber network of the left posterior fascicle, thus explaining its characteristic QRS configuration and its property of induction and termination by extrastimuli. Lin et al. reported three patients with similar VT and came to the same conclusion regarding the mechanism of the VT. Zipes et al. reported three patients with similar VT, but they suggest that triggered activity might have been responsible for the condition in one of them. The assumption that the VT is a reentrant tachycardia has mainly depended on the observation that it could both be induced and terminated by extrastimuli. However, tachycardia due to triggered

ventricle supplied by the posterior division of left posterior fascicle. In the present study, the configuration of VT showed right bundle branch and left-axis deviation in 14 patients (figure 1, A), and right bundle branch block and right-axis deviation in two patients (figure 1, B). Endocardial mapping during VT showed that the earliest ventricular activation was at the apical inferior region of the left ventricle in all 14 patients in whom the QRS configuration of the VT showed right bundle branch block and left-axis deviation, while it was at the apical anterosuperior region of the left ventricle in two patients in whom VT showed right bundle branch block and right-axis deviation (figure 2). The fact that pacing from the site of the earliest ventricular activation produced very similar QRS complexes to the VT suggests that the earliest activation site indicates the origin of VT in these patients (figure 4). Endocardial mapping also showed that the His bundle potential during VT occurred 10 to 20 msec after the earliest ventricular activation, suggesting early retrograde conduction to the His bundle (figure 5). The results of endocardial mapping and pace-mapping suggest that VT with a QRS configuration of right bundle branch block and left-axis deviation originates within the Purkinje network of the posterior division and that the QRS

![FIGURE 4. Pacing from the site of earliest ventricular activation during VT (apical inferior site of the left ventricle) in patient 16. Note that pacing this site produces QRS complexes very similar to the spontaneous VT.](http://circ.ahajournals.org/content/circulation/102/11/564.full)

**TABLE 2**

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Mode of initiation</th>
<th>VT zone (msec)</th>
<th>Coupling interval vs echo interval</th>
<th>Progressive fusion</th>
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<tbody>
<tr>
<td>1</td>
<td>S, A</td>
<td>280-240</td>
<td>1</td>
<td>–</td>
</tr>
<tr>
<td>2</td>
<td>S</td>
<td>300-280</td>
<td>?</td>
<td>NT</td>
</tr>
<tr>
<td>3</td>
<td>S</td>
<td>290-280</td>
<td>?</td>
<td>NT</td>
</tr>
<tr>
<td>4</td>
<td>S</td>
<td>300-270</td>
<td>I</td>
<td>NT</td>
</tr>
<tr>
<td>5</td>
<td>S, A</td>
<td>300</td>
<td>?</td>
<td>NT</td>
</tr>
<tr>
<td>6</td>
<td>S</td>
<td>260-200</td>
<td>I</td>
<td>NT</td>
</tr>
<tr>
<td>7</td>
<td>B, A</td>
<td>—</td>
<td>?</td>
<td>NT</td>
</tr>
<tr>
<td>8</td>
<td>S</td>
<td>300-220</td>
<td>I</td>
<td>–</td>
</tr>
<tr>
<td>9</td>
<td>S, A</td>
<td>320-280</td>
<td>I</td>
<td>+</td>
</tr>
<tr>
<td>10</td>
<td>B</td>
<td>—</td>
<td>?</td>
<td>NT</td>
</tr>
<tr>
<td>11</td>
<td>S</td>
<td>320-290</td>
<td>I</td>
<td>NT</td>
</tr>
<tr>
<td>12</td>
<td>S</td>
<td>300</td>
<td>?</td>
<td>NT</td>
</tr>
<tr>
<td>13</td>
<td>B</td>
<td>—</td>
<td>?</td>
<td>NT</td>
</tr>
<tr>
<td>14</td>
<td>S</td>
<td>270-230</td>
<td>I</td>
<td>+</td>
</tr>
<tr>
<td>15</td>
<td>S</td>
<td>310-300</td>
<td>?</td>
<td>–</td>
</tr>
<tr>
<td>16</td>
<td>S</td>
<td>430-360</td>
<td>I</td>
<td>+</td>
</tr>
</tbody>
</table>

All patients’ VT terminated with pacing, and none showed fragmentation.

A = VT induced by atrial pacing; S = VT induced by a single ventricular stimulus; B = VT induced by burst ventricular pacing; VT zone = a range of coupling intervals that induced VT during a single extrastimulus method; VT zone (−) = VT not induced by a single extrastimulus; I = the relationship between the coupling interval and the echo interval was inverse; = the relationship could not be determined; = progressive fusion not present; + = progressive fusion present; NT = progressive fusion not tried.

*The site of stimulation was the right ventricular apex in all but patients 10 and 13, in whom it was the left ventricular apex.
FIGURE 5. His bundle recording during VT in patient 13. Surface leads (I, II, III, V₁, V₃, and V₆) are recorded with the intracardiac recordings, which were as follows: right ventricular mid septum (RV mid sep), apical inferior portion of the left ventricle (LV apical inf), high right atrium (HRA), and His bundle (HBE) electrogram. Note that the onset of the QRS coincides with the activity of the left ventricular apical inferior region, and precedes the His bundle activity by 20 msec.

FIGURE 6. The relationship between the premature coupling interval (S₁-S₂) and the echo interval (S₂-V₁) at a basic cycle length of 600 msec (S₁-S₂) in patient 9. Note that as (S₁-S₂) becomes shorter (320 to 285 msec), (S₂-V₁) becomes longer (340 to 365 msec), suggesting an inverse relationship.
That the initiation of tachycardia by constant pacing depends on the basic cycle length is characteristic of both reentry and triggered activity. Also, the ability to initiate tachycardia is closely related to the number of pacing beats in both kinds of tachycardia. However, in triggered activity, the longer the period of fixed pacing, the greater the chance of induction. In contrast, in reentrant tachycardia, certain numbers of pacing beats are more likely to induce tachycardia at a critical pacing rate. This may be caused by a Wenckebach type of conduction in one of the two pathways in addition to the presence of unidirectional block. The Wenckebach type of conduction delay is important for the initiation of reentrant tachycardia, and has been observed in patients with atrioventricular nodal reentrant tachycardia during constant ventricular pacing at a rate at which type I second-degree ventriculoatrial block occurs. In the present study, the initiation of VT was closely related to the number of pacing beats but not to the duration of pacing. In patient 1, VT was initiated with greater frequency by 4, 7, 8, 12, and 13 pacing beats, and less frequency by 3, 5, 6, 9, and 10 beats at a pacing cycle length of 300 msec (figures 7 and 8). This suggests that conduction of a Wenckebach type in the reentrant pathway was present at a cycle length of 300 msec.

The presence of progressive fusion as defined by Waldo et al. is one of the characteristics of reentry. The fact that it was recognized in only three of six patients in the present study is not inconsistent with reentry, since the ability to satisfy this criterion provides strong evidence confirming the mechanism of

![FIGURE 7. The relationship between the number of pacing beats at a cycle length of 300 msec and the initiation of VT in patient 1. Note that 4, 8, and 12 pacing beats induced VT, but 3, 5, and 9 pacing beats did not.](image)

activity may also be started or stopped by a single premature stimulus. Thus, in the present study, three other electrophysiologic characteristics were examined: (1) the relationship between the premature coupling interval and the echo interval, (2) the relationship between the number of pacing beats and the induction of VT, and (3) the presence of progressive fusion.

In reentrant tachycardia, such as atrioventricular reentrant tachycardia, atrioventricular nodal reentrant tachycardia, and VT associated with old myocardial infarction, the relationship between the premature coupling interval and the echo interval is usually inverse. In contrast, in triggered tachyarrhythmias, the relationship is typically direct. The fact that the relationship was consistently inverse in all our patients suggests that its mechanism was reentry.

### TABLE 3

<table>
<thead>
<tr>
<th>Patient</th>
<th>Lidocaine</th>
<th>Ajmaline</th>
<th>Verapamil</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>−</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>2</td>
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<td>+</td>
<td>+</td>
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<tr>
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<td>+</td>
<td>+</td>
</tr>
<tr>
<td>5</td>
<td>−</td>
<td>+</td>
<td>+</td>
</tr>
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<td>+</td>
<td>+</td>
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</tr>
<tr>
<td>15</td>
<td>−</td>
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</tr>
<tr>
<td>16</td>
<td>−</td>
<td>+</td>
<td>NT</td>
</tr>
</tbody>
</table>

+ = terminated VT; − = failed to terminate VT; NT = not tested.
FIGURE 8. The relationship between the number of pacing beats at a cycle length of 300 msec and the initiation of ventricular tachycardia in patient 1. The percentage of times that VT was initiated is indicated on the vertical axis, and the number of pacing beats is indicated on the horizontal axis. The denominators represent the number of trials; the numerators represent the number of trials in which VT was initiated. Note that VT was initiated with greater frequency by 4, 7, 8, 12, and 13 pacing beats and with less frequency by 3, 5, 6, 9, or 10 beats.

FIGURE 9. Progressive fusion in patient 16. The first trace shows electrocardiographic lead II during VT (cycle length 315 msec). Second, third, fourth traces show electrocardiographic lead II during periods of transient entrainment pacing from the right ventricular septum at cycle lengths of 310 (second trace), 300 (third trace), and 255 msec (forth trace). The bottom trace shows electrocardiographic leads II after interruption of pacing from the right ventricular septum at a cycle length of 250 msec. The top trace shows the morphology of ventricular complexes during VT. The morphologies of the ventricular complexes in the second through the fourth traces represents different degrees of ventricular fusion (progressive fusion) during transient entrainment at the respective pacing rates. The morphologies of the ventricular complexes on the bottom trace represent ventricular activation by pacing from the right ventricular septum.
reentry, but the absence of progressive fusion does not exclude reentry. In fact, the demonstration of progressive fusion requires the presence of an excitable gap in part of the reentrant loop.

Fragmentation is thought to represent an area of slow conduction that is a prerequisite for reentry.\textsuperscript{15} It is usually recorded before the onset of the QRS complex during VT in patients with associated organic heart disease. However, the fact that fragmentation could not be recorded during normal sinus rhythm or during VT in any of the patients in the present study is not inconsistent with reentry. It is possible that very slow conduction at a relatively small area produces enough delay for reentry without slowing fragmentation, as is seen in conduction within the atrioventricular node.

In summary, these electrophysiologic characteristics, in addition to the fact that the VT could be induced and terminated by pacing, strongly suggest that the mechanism of the VT was reentry.

**Antiarrhythmic drugs and patients follow-up.** One of the characteristics of idiopathic VT with a configuration of right bundle branch block and left-axis deviation is that it is responsive to verapamil but not responsive to lidocaine or propranolol. The present study shows that not only idiopathic VT with the configuration of right bundle branch block and left-axis deviation but also VT with right bundle branch block and right-axis deviation had the same electropharmacologic characteristics. The mechanism by which verapamil is effective in terminating this VT remains to be elucidated, but it is possible, as German et al.\textsuperscript{3} have suggested, that part of the circuit of the VT contains a calcium channel-mediated pathway. This may explain the facts that the VT could be terminated by verapamil and that no recognizable organic change could be demonstrated, suggesting that the area responsible for slow conduction was relatively small.

The present study confirms that intravenous verapamil is very effective in terminating VT and also shows that long-term oral verapamil is effective clinically. One patient placed on oral verapamil became asymptomatic, and all the remaining patients on verapamil improved symptomatically as the VT became nonsustained. The reason that oral verapamil did not suppress VT completely is probably related to the dose since 240 mg/day is a relatively small dose for adult patients.

We drew the following conclusions from this study: (1) Chronic sustained VT originating from left ventricle in patients without apparent organic heart disease has peculiar electrocardiographic, electrophysiologic, and electropharmacologic characteristics. (2) The electrophysiologic characteristics of the VT suggest that the mechanism is reentry. (3) Verapamil is effective for both short- and long-term treatment of this VT.

**References**

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T Ohe, K Shimomura, N Aihara, S Kamakura, M Matsuhisa, I Sato, H Nakagawa and A Shimizu

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