Idiopathic Sustained Ventricular Tachycardia Responsive to Verapamil: Clinical Electrocardiographic and Electrophysiologic Considerations

HIROSHI KASANUKI, M.D., SATOSHI OHNISHI, M.D.
ETSUKO TANAKA, M.D., AND KOSHIUCHI HIROSAWA, M.D.

Fifteen cases of idiopathic VT responsive to verapamil were studied to examine its clinical, electrocardiographic and electrophysiologic features. All patients were male, aged 15–49, average age 28. Initial onset of VT occurred at ages 9–48 (average 21). Time from onset of VT to first admission was 1–20 years (average 8.2 years), and patients had been followed for 17–40 months (average 27 months). 13 cases had palpitations, 5 had faintness, 1 had syncope, but no deaths were reported. ECG's at time of VT exhibited CRBBB + LAD pattern in 12 cases, CRBBB + RAD pattern in 1, and LBBB in 2. VT rate was 130–200 bpm (average 163 bpm), with QRS width of 0.11–0.16 sec (average 0.14 sec). ECG's during sinus rhythm revealed no ST/T abnormalities, although in 6 cases they were found post-VT. 5 cases had recognizable H waves during VT, and HV intervals were shorter than that during sinus rhythm.

VT could be induced by programmed electrical stimulation in 14 cases. VT or RVR could be induced by atrial pacing in 6 of 14, single RV extra-stimuli in 12 of 14, paired pulses in 5 of 12, RV overdrive pacing in 7 of 14, and burst pacing in 6 of 14 cases. VT could be terminated by RV burst pacing in 14 of the 15 cases, while single RV stimuli were effective in 5 out of 12 cases.

Among the 12 cases in which VT could be induced by single RV extra-stimuli, the relationship between changes in premature interval for the induction of VT and the echo interval of VT (extra-stimulus to first VT complex) was examined. 8 showed an inverse relationship, 3 showed a concordant relationship and 1 case could not be assessed. An inverse relationship was found between changes in paced cycle length and echo interval for the 2 cases in which VT could be induced by rapid pacing.

Verapamil terminated sustained VT in 12 out of 13 cases, and in another case had a pronounced decelerating effect. Prior to termination, VT rate was drastically reduced (from 163 ± 29 bpm to 128 ± 29 bpm). Verapamil was able to prevent the induction of VT in 6 out of 14 cases, while in 6 cases the VT zone was expanded and in 2 cases the VT zone was narrowed. Both minimum and maximum values of premature intervals for induction were significantly extended, from 278 ± 58 sec to 223 ± 82 msec and from 312 ± 93 msec to 629 ± 96 msec, respectively.

Reentry was considered as the primary mechanism for verapamil-responsive

Key Words:
Idiopathic sustained ventricular tachycardia
Verapamil
Electrophysiologic study
Reentry
Triggered activity

Department of Internal Medicine, The Heart Institute of Japan Tokyo Women's Medical College, Tokyo, Japan
Mailing address: Hiroshi Kasanuki, M.D., Department of Internal Medicine, The Heart Institute of Japan, Tokyo Women's Medical College, 10 Kawada-cho, Shinjuku-ku, Tokyo 162, Japan


NII-Electronic Library Service
induced VT among 10 cases examined in this study. However, in 3 cases triggered activity, and in one case enhanced automaticity, was suspected as well. Therefore, no one mechanism can be said to be responsible for this type of VT.

Although the efficacy of verapamil for the treatment of ventricular tachycardia (VT), has been reported by Gotsman et al.\(^1\) and Heng et al.\(^2\), it has generally been considered to be ineffective. However, recent reports, indicate that verapamil is effective against certain types of VT such as idiopathic VT with a morphologic pattern of RBBB and LAD in young patients\(^3\)–\(^12\); exercise-triggered VT with a morphologic pattern of LBBB and RAD\(^13\); multifrom accelerated idioventricular rhythm from acute myocardial infarction\(^14\); polymorphous VT in acute myocardial infarction\(^15\); torsades de pointes with normal QT and short coupling intervals\(^16\); and VT related to cardiomyopathy\(^17\). Belhassen et al.\(^1\) and Lin et al.\(^7\) suggest that sustained VT responsive to verapamil in young patients without obvious organic heart disease represents a distinct clinical entity with unique electrocardiographic and electrophysiologic properties. Slow inward currents are proposed as the mechanism for such VT, but details remain unclear. This study considers the clinical, electrocardiographic, and electrophysiologic findings in 15 cases of idiopathic sustained VT, and discusses suspected mechanisms.

**MATERIALS AND METHODS**

Materials: 15 cases of sustained VT for which verapamil had slowing, terminating, or preventive effects were studied. All were male, aged 15–49 yrs, average age 28. Both non-invasive exams (ECG, chest X-ray, echocardiogram, etc.) and invasive exams (cardiac catheterization, cardiac angiography, etc.) revealed no organic heart disease in any patient.

Methods: Electrophysiologic studies (EPS) were conducted on all patients. Our stimulation protocol consisted of rapid pacing, burst pacing, and single/double extrastimuli while pacing from the right atrium and right ventricle. Pharmacological protocol and definitions were as reported in an earlier study.\(^11\),\(^12\) 5–15 mg verapamil was administered intravenously 1 mg/min. Programmed stimulation was performed 5–30 min after completion of drug infusion. Results were compared using student’s t-test for paired observations.

**RESULTS**

(1) Clinical Findings (Table I)

During VT, 13 cases complained of palpitations; 5 had syncope, and 1 was asymptomatic. 1 case experienced congestive heart failure due to VT lasting for 3 days. The initial onset of VT ranged between ages 9–48, average age 21. Time from first VT to EP tests was 1–20 yrs, average time 8.2 yrs. Post-EPS followup was 17–40 months, average 27 months, with no deaths.

(2) ECG and His Bundle Electrogram Findings (Table I)

12 cases (80%) showed a CRBBB + LAD pattern on ECG during VT. LAD ranged from -60° to -120°, average -93°. 1 case exhibited a CRBBB + RAD pattern and 2 cases exhibited CLBBB patterns. VT rate ranged from 130 to 200 bpm, average 163 bpm. QRS width ranged

---

*Japanese Circulation Journal Vol. 50, January 1986*
TABLE 1  CLINICAL, ELECTROCARDIOGRAPHIC AND ELECTROPHYSIOLOGICAL FEATURES OF 15 PATIENTS

<table>
<thead>
<tr>
<th>Case</th>
<th>Age (yrs)</th>
<th>Age of VT-onset (yrs)</th>
<th>Symptom</th>
<th>Sinus-rhythm</th>
<th>QRS pattern</th>
<th>QRS axis</th>
<th>QRS width (sec)</th>
<th>VT rate (min)</th>
<th>&quot;H&quot; wave</th>
<th>ST-T change</th>
<th>Induction of VT, RVR</th>
<th>Termination of VT from RV</th>
<th>Follow-up (month)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>33</td>
<td>30</td>
<td>Palp</td>
<td>iRBBB</td>
<td>RBBB</td>
<td>-110°</td>
<td>0.12</td>
<td>182</td>
<td>+</td>
<td>O</td>
<td>S</td>
<td>S.B</td>
<td>35</td>
</tr>
<tr>
<td>2</td>
<td>27</td>
<td>16</td>
<td>Palp. CHF</td>
<td>normal</td>
<td>RBBB</td>
<td>-100°</td>
<td>0.12</td>
<td>168</td>
<td>+</td>
<td>S</td>
<td>S</td>
<td>B</td>
<td>36</td>
</tr>
<tr>
<td>3</td>
<td>30</td>
<td>15</td>
<td>Palp</td>
<td>normal</td>
<td>RBBB</td>
<td>-100°</td>
<td>0.11</td>
<td>200</td>
<td>+</td>
<td>O</td>
<td>S.D.O</td>
<td>S.B</td>
<td>24</td>
</tr>
<tr>
<td>4</td>
<td>16</td>
<td>10</td>
<td>Palp. Faint</td>
<td>tall R in V1</td>
<td>RBBB</td>
<td>-100°</td>
<td>0.16</td>
<td>130</td>
<td>+</td>
<td>O</td>
<td>S.O.B</td>
<td>S.B</td>
<td>27</td>
</tr>
<tr>
<td>5</td>
<td>20</td>
<td>16</td>
<td>Palp</td>
<td>QS in V1, V2</td>
<td>RBBB</td>
<td>-120°</td>
<td>0.16</td>
<td>150</td>
<td>+</td>
<td>S.D</td>
<td>B</td>
<td>17</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>45</td>
<td>35</td>
<td>Palp. Faint</td>
<td>iRBBB</td>
<td>RBBB</td>
<td>-60°</td>
<td>0.14</td>
<td>150</td>
<td>+</td>
<td>S.O</td>
<td>S.O.B</td>
<td>B</td>
<td>40</td>
</tr>
<tr>
<td>7</td>
<td>33</td>
<td>32</td>
<td>Palp. Faint</td>
<td>normal</td>
<td>RBBB</td>
<td>-60°</td>
<td>0.15</td>
<td>130</td>
<td></td>
<td></td>
<td>S.O.B</td>
<td>S.B</td>
<td>29</td>
</tr>
<tr>
<td>8</td>
<td>15</td>
<td>11</td>
<td>Palp. Faint</td>
<td>normal</td>
<td>RBBB</td>
<td>-90°</td>
<td>0.13</td>
<td>183</td>
<td>+</td>
<td>S.D</td>
<td>B</td>
<td>26</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>14</td>
<td>10</td>
<td>Palp. Faint</td>
<td>normal</td>
<td>RBBB</td>
<td>-90°</td>
<td>0.12</td>
<td>150</td>
<td>+</td>
<td>S.O.B</td>
<td>S.B</td>
<td>30</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>44</td>
<td>35</td>
<td>—</td>
<td>LAD</td>
<td>RBBB</td>
<td>-90°</td>
<td>0.14</td>
<td>150</td>
<td></td>
<td>O</td>
<td>O</td>
<td>S.B</td>
<td>17</td>
</tr>
<tr>
<td>11</td>
<td>20</td>
<td>14</td>
<td>Palp</td>
<td>normal</td>
<td>RBBB</td>
<td>-110°</td>
<td>0.14</td>
<td>180</td>
<td>+</td>
<td>S.O.B</td>
<td>B</td>
<td>25</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>22</td>
<td>12</td>
<td>Palp</td>
<td>normal</td>
<td>RBBB</td>
<td>-90°</td>
<td>0.16</td>
<td>140</td>
<td></td>
<td>S.O.B</td>
<td>S.O.B</td>
<td>S.B</td>
<td>18</td>
</tr>
<tr>
<td>13</td>
<td>18</td>
<td>9</td>
<td>Palp</td>
<td>normal</td>
<td>RBBB</td>
<td>+110°</td>
<td>0.13</td>
<td>150</td>
<td>+</td>
<td>S</td>
<td>B</td>
<td>19</td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>35</td>
<td>15</td>
<td>Palp</td>
<td>normal</td>
<td>LBBB</td>
<td>-120°</td>
<td>0.16</td>
<td>194</td>
<td></td>
<td>S</td>
<td>B</td>
<td>32</td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>49</td>
<td>48</td>
<td>Syncope</td>
<td>normal</td>
<td>LBBB</td>
<td>+60°</td>
<td>0.16</td>
<td>187</td>
<td>+</td>
<td>O</td>
<td>S.D.B</td>
<td>B</td>
<td>34</td>
</tr>
</tbody>
</table>

Palp = palpitation; CHF = congestive heart failure; faint = faintness; O = overdrive pacing; S = single ventricular or atrial extrastimuli; D = double ventricular extrastimulus; B = burst pacing
from 0.11 to 0.16 sec, average 0.14 sec. Among those exhibiting a CRBBB pattern, 3 cases were r s R type, 4 were q R type, 5 were R type. During normal sinus rhythm ECG's, 2 cases showed incomplete right bundle branch block, and 1 showed LAD. In 6 cases transient ST/T abnormalities, located in the inferior lateral posterior wall, were found after VT termination.

His bundle electrograms revealed no abnormalities in PA, AH, and HV intervals during sinus rhythm. During VT, 5 cases had H waves immediately preceding the QRS. HV intervals were 10–30 msec, and were shorter during sinus rhythm. According to surface potential mapping recorded in 8 cases, VT originated in the left ventricular apex in 3 cases, posterior left ventricular septum in 4 cases, and right ventricular outflow tract in 1 case.

(3) Induction and termination of VT (Table I)

Sustained VT could be induced by pacing in 14 cases. One case (case 7) could not be evaluated, because VT was present before pacing. In one case VT could be induced by rapid pacing after intravenous administration of 1 mg/min isoproterenol (case 10). VT or repetitive ventricular response (RVR) was inducible by rapid pacing in the right atrium in 6 cases; single extrastimuli in the right atrium in 1 case; single extrastimuli in the right ventricle in 12 cases (of which 5 cases could also be induced by double extrastimuli); rapid right ventricular pacing in 7 cases; and burst pacing in 6 cases. Of the 12 cases responsive to single extrastimuli in the right ventricle, in 5 cases only RVR with a morphology similar to VT could be induced.

VT was terminated by burst pacing in 14 of 15 cases (93%), and by single right ventricular extrastimuli in 5 of 12 cases (42%). Overdrive suppression and warming up were observed in the one case unresponsive to termination by electrical stimulation. In the 12 cases in which VT could be induced by single right ventricular extrastimuli, the relationship between the premature interval of the paced extrastimulus and the echo interval of VT (extrastimulus to first VT complex) was examined. In the 2 cases inducible by rapid pacing alone, the relationship between pacing cycle length and echo interval of VT was examined. In 9 cases, shortening of the
premature interval caused an inverse lengthening of the echo interval (inverse relationship). Conversely, in 2 cases shortening of the premature interval showed a corresponding decrease in echo interval (concordant relationship). No clear relationship could be discerned in the remaining 3 cases. The echo zone for induction of VT or RVR by extrastimuli ranged from 10 to 140 msec.

(4) Termination Effect of Verapamil
Verapamil was able to affect VT in 13 cases. Of these, 12 showed complete termination and 1 showed noticeable prolongation of VT cycle length. In 2 remaining cases, the termination effect of verapamil could not be evaluated, because verapamil was not administered during VT. There were no cases in which verapamil caused no change or deterioration. VT rate prior to termination showed marked slowing from 163 ± 29 bpm to 128 ± 29 bpm, p < 0.001 (Fig. 1). As for the manner of termination, VT was decelerated, fusion or capture beats appeared followed by further deceleration, and sinus rhythm was returned. As indicated in Fig. 2, after conversion to sinus rhythm with 5 mg verapamil, continued administration of an additional 5 mg caused premature ventricular beats to appear with QRS morphologies identical to VT. Further administration of verapamil did not abolish these premature beats.
Mechanisms of Paroxysmal Tachyarrhythmias

one way or the other, the rate of induced VT was slower after the administration of verapamil, and in 2 cases VT ceased spontaneously within 10 beats. In 5 cases there was no change in the relationship (inverse/concordant) between premature interval and echo interval before and after the administration of verapamil. Of the 3 cases in which no clear relationship could be discerned previously, 1 showed a concordant change and one an inverse change after administration of verapamil (Figs. 4, 5).

Figure 6 shows a case in which verapamil caused marked prolongation of the VT zone. The effect of verapamil dosage on VT zone and VT rate is also indicated. Increasing the dosage from 5 mg to 10 mg widened the VT zone and increased the minimum and maximum interval for the premature interval while slowing the VT rate.

DISCUSSION

Recently much interest has focused on idiopathic sustained VT responsive to verapamil but studies in which EP tests were conducted are few. We report the clinical, electrocardiographic, His bundle electrographic, and electrophysiologic characteristics of 15 cases examined at our institution, and consider the effect of verapamil as well as the mechanism of VT responsive to verapamil.

(1) Clinical Findings

In previous reports, the age distribution of patients with idiopathic VT responsive to verapamil has been 7–51 yrs. The fact that they center on a young population, average age 20–33, is a noteworthy characteristic. The average age of onset in our study (21 yrs) further substantiates this trait. However, the inclusion of 3 cases in which onset occurred after age 35 suggests this kind of VT is not necessarily limited to the young. As for symptoms, Ward et al. reported palpitations in all. 13 cases in this study had palpitations, though relatively light in degree. The possibility of syncope and congestive heart failure due to prolonged VT must be noted, however. Belhassen et al. and Lin et al. reported cases monitored for 12 years. Our study includes one patient with VT for 20 years, and no deaths have been reported over an average followup period of 27 months, indicating good prognosis.

As for gender, Belhassen et al. and Lin et al.
Klein et al. did not ascribe any significance to it. In our experience idiopathic sustained VT has been more prevalent among males, and idiopathic non-sustained VT more prevalent among females, but further investigation is necessary.

(2) ECG Findings

Belhassen et al. considered the appearance of a CRBBB + LAD pattern to be one of the most distinguishing traits of this VT. A total of 16 of 17 cases (94%) in studies by Belhassen et al., Lin et al., Klein et al., and Ward et al. were of this type. Our study found 12 of 15 cases (80%) to be of this type, while 1 case exhibited a CRBBB + RAD pattern and 2 cases showed a LBBB pattern. Wu et al. reported 3 out of 3 cases of exercise-triggered idiopathic VT.
VT responsive to verapamil to be of the LBBB type. Among the 7 cases exhibiting a CRBBB + LAD pattern, surface potential mapping revealed the left ventricular apex and the proximal portion of the left posterior fascicle to be the origin of VT. Belhassen et al. determined the origin to be in the lower left ventricular apex and postero-septal area by endocardial mapping. A pattern of CRBBB + LAD, then, may not necessarily be essential in the pathogenesis of verapamil-responsive VT. QRS width during VT was extended (0.11–0.16 sec.), and VT rates were within 130–200 bpm, in line with the reports of others.

As for ECG at rest, Belhassen et al. reported no abnormalities, whereas Lin et al. reported T-wave inversion and ST abnormalities. In our study, no ST/T abnormalities were found. Belhassen et al. also emphasized transient T-wave abnormalities in the inferolateral wall post-VT. We only saw this in 40%, and consider it to be a nonspecific change due to factors such as prolonged durations of VT.

(3) His-Bundle Electrogram Findings
His bundle potentials were observed during VT in 5 of the 15 cases (33%). Lin et al. reported an absence of H waves in all 4 cases studied, while Ward et al. found H waves in all 5 of his patients. German et al. reported H waves in 8 of 10 cases in which VT could be induced by atrial pacing. In all studies including this one, those with H waves had a H-V interval of less than 35 msec. This implies that some form of macro reentry in the bundle branches is an unlikely cause for the VT.

(4) Response to Programmed Electrical Stimulation
Among a total of 30 cases with idiopathic VT responsive to verapamil examined by Belhassen et al. and others, VT could be induced by programmed stimulation in 26. In our study sustained VT could be induced in all cases except one, which was in VT even before pacing. The induction rates for atrial and ventricular pacing are reported as 0–40% and 0–100%, respectively, and in this study the corresponding figures were 43% and 93%. In 42% of the cases right ventricular premature stimulation did not induce sustained VT but caused RVR of similar morphology to the VT. On the other hand, rapid pacing induced sustained VT in all. The difference in induction mechanism is unknown.

VT could be terminated by burst pacing in 14 of the 15 cases. The one case that did not respond to burst pacing exhibited an overdrive suppression and a warming-up phenomenon, causing abnormal automaticity to be suspected.

Reentry and triggered activity due to late afterpotentials are considered as causes for VT that can be induced/terminated by programmed stimulation. Differentiation between the two causes is based on observation of induction/termination and of the effects of timed premature beats and overdriving during sustained VT. The relationship between premature interval of pacing and echo interval of VT is considered especially significant. An inverse relationship is thought to indicate reentry while a concordant relationship is thought to indicate triggered activity. Lin et al. found an inverse relationship in 3 of 4 cases, while Belhassen et al. found an inverse relationship in 1 of 3 cases. Lin et al., Ward et al., and Klein et al. suspected reentry as the mechanism of verapamil-responsive VT for the following reasons, among others: VT can be induced/terminated by programmed stimulation, especially premature stimulation; the duration and rate of induced tachycardia does not depend on the mode of induction; and no overdrive acceleration or warming up has been observed.

On the other hand, Wellens et al. suspected triggered activity because a concordant relationship between premature and echo intervals was noted in one of their cases. Sung et al. suspected triggered activity due to the following: sinus rhythm, atrial pacing, and ventricular pacing beyond a certain rate could all induce VT; premature ventricular stimulation could not induce VT; the echo interval was 0–80 msec shorter than the preceding cycle length; and shortening the pacing cycle length also caused shortening of the echo interval. Sung et al. also considered VT induced by premature ventricular stimulation to be caused by reentry, and reported that verapamil was ineffective in all cases.

In our experience, an inverse relationship between the premature pacing and VT echo intervals was found in 7 cases, and a concordant relationship in only 2. Of 3 cases in which no pattern could be discerned before administration of verapamil, one exhibited an inverse relationship and one a concordant relationship after administration. Reentry is suspected as the primary cause for cases exhibiting an inverse relationship. However, in the 3 cases which
exhibited a concordant relationship, VT could be induced by single ventricular extrastimuli, making determination of reentry or triggered activity difficult. In this respect our results differ from those of Sung et al.6

In this study there were 2 cases where VT could not be induced by premature stimulation but was induced by rapid pacing. In one of these cases, which showed an inverse relationship, VT could only be induced by atrial pacing, whereas left and right ventricular pacing at similar rates was ineffective. It is thought that in this case atrial pacing caused decremental conduction in the abnormal portion of the conduction system, causing a reentry similar to that described by Klein et al.8. The other case in which VT could not be induced by premature stimulation was that in which VT could be induced after administration of isoproterenol. Isoproterenol enabled induction of VT at a constant pacing rate, and the case showed an inverse relationship between cycle length and echo interval.

There are recent reports indicating that the relationship between premature pacing interval or rapid pacing cycle length and VT echo interval can be concordant even for VT caused by reentry, and conversely, inverse in relationship even for VT caused by triggered activity.19,20 Therefore, the relationship between the two is not necessarily a decisive indication of one mechanism or the other. The cases of verapamil-responsive idiopathic sustained VT examined by us showed varying responses to programmed electrical stimulation. Consequently, it is also difficult to determine the mechanism of VT from these results alone.

(5) Termination and Preventive Effects of Verapamil

Verapamil was observed to suppress VT in 12 out 13 cases, but in one case even 10 mg of intravenous administration only had a slowing effect. Lin et al.7 and Ward et al.10 have also reported such single cases. One characteristic of verapamil’s termination of VT is a marked deceleration of the tachycardia before its abolition. VT rate is drastically reduced, followed by repetitive form before resolving into sinus rhythm. It is postulated that this is due to slow conduction in the reentry circuit, slowing of the discharge rate of the autonomic focus, or progression of exit block. However, the two cases in which continued administration of verapamil after suppression of VT caused ventricular premature beats cause us to suspect reentry over enhanced automaticity.

Although considerable attention has been focused on verapamil’s ability to suppress VT, little is reported about its influence on the inducibility of VT by programmed stimulation (indicative of preventive effect). Among 4 cases studied by Lin et al.7 one case was completely noninducible, in 2 cases non-sustained VT or RVR could be induced, and in one case sustained VT could be induced. Ward et al.10 also reported a case where VT could be induced after suppression by verapamil, where the induced VT ceased spontaneously thereafter. Mason et al.8 reported 5 non-inducible cases among 7 in which verapamil was able to terminate VT, and noted that VT ceased spontaneously in the 2 inducible cases within 5 seconds.

In our study VT became non-inducible in only 6 of 14 cases, while in another 6 the VT zone was expanded and in 2 the VT zone contracted. Of special interest is the fact that both maximum and minimum values for the premature interval that could induce VT were extended, and the extension was proportional to the verapamil dosage. This suggests the possibility that verapamil extended the refractory period of a portion of the reentry circuit. In previous studies we have stressed the importance of evaluating the termination effects and preventive effects of a medication separately11,12. It is not rare for verapamil to suppress VT without affecting its prevention. When verapamil was administered chronically to patients in whom it extended the VT zone, the frequency of VT episodes either remained unchanged or increased in 3 out of 4 cases, but each episode ceased spontaneously in a short while. German et al.4 also gave chronic oral administration of verapamil to 3 patients in which it had a termination effect, and in 2 cases observed cessation of sustained VT along with an appearance of non-sustained VT.

(6) Mechanism of VT

Verapamil is a powerful, potent inhibitor of slow channel inward currents. However, it also possesses cardiovascular and additional electrophysiologic effects. In other words, it alters outward currents21 and its (+) isomer blocks the sodium channel of cardiac tissue.22 It also decreases left ventricular preload and afterload, decreases cardiac contractility, and has a dilatory effect on coronary vessels. However, if a patient has no organic heart disease, the cardiovascular
effect can not be of much significance. It is also difficult to attribute verapamil’s significant effect on VT to its additional electrophysiological effects. Therefore, slow inward currents are suspected to play a role against VT, in which case both reentry with slow response action potentials in parts of the circuit as well as triggered activity due to late afterpotentials may be viable mechanisms. The recent discovery in human cardiac tissue of slow response action potentials responsive to verapamil, non-uniform refractory periods, unidirectional block, slow conduction, and triggered activity due to late potentials makes the above even more plausible.

In 15 cases of idiopathic VT responsive to verapamil, we assessed the response to programmed electrical stimulation and verapamil in order to differentiate between VT caused by reentry and that caused by triggered activity. However, we conclude that it is not possible to differentiate the two based on our present knowledge. Though cases due to reentry seem prevalent, there are also cases in which triggered activity is suspected. Furthermore, one case was suspected to be due to enhanced automaticity. Therefore no one mechanism can be ascribed to VT that responds to treatment by verapamil.

REFERENCES


Japanese Circulation Journal Vol. 50, January 1986