Efficacy of Procainamide and Lidocaine in Terminating Sustained Monomorphic Ventricular Tachycardia
– Retrospective Case Series –

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**Background:** The efficacy of antiarrhythmic drugs in terminating sustained monomorphic ventricular tachycardia (SMVT) was assessed in a retrospective manner to provide a basis for recommending their use.

**Methods and Results:** The 90 patients were included in this study to evaluate the efficacy to terminate SMVT using procainamide or lidocaine. All patients were alert and responsive. The mean systolic blood pressure was 91±25 mmHg (range, 40–150 mmHg). SMVT was diagnosed from ECG recordings and later in an electrophysiological study. VTs with a cycle length of 329±55 and 324±61 ms were treated with the mean doses of 358±50 mg and 81±30 mg of procainamide and lidocaine and were terminated in 53/70 (75.7%) and in 7/20 (35.0%) respectively. The drugs were discontinued if there was no rise in blood pressure after slowing of the tachycardia rate or if there were signs of impending deterioration in consciousness. Though procainamide was effective, blood pressure was often low and DC shock should be available at all times during administration of the drug.

**Conclusions:** Procainamide, the relatively older drug, was more effective than lidocaine in terminating SMVT associated with structural heart diseases. This is a retrospective analysis but can form the basis for formulating guidelines for initial management of SMVT. (Circ J 2010; 74: 864–869)

**Key Words:** Antiarrhythmic agents; Arrhythmia; Ventricular tachycardia

The association of ventricular tachyarrhythmia with sudden cardiac death can be seen from the results of electrophysiologic studies (EPS) wherein sustained ventricular tachyarrhythmia can be repeatedly induced. In patients who die suddenly during recording of ambulatory ECG, approximately 80% are found have died from ventricular tachycardia (VT) or fibrillation (VF). VF requires immediate termination before arrival at hospital, and basic life support and advanced cardiovascular life support are essential for rescue. Sustained monomorphic VT (SMVT) of a rapid rate requires prompt termination, but, occasionally, SMVT may show stable hemodynamics and in such cases antiarrhythmic drugs can be administered to terminate the arrhythmia.

Procainamide is recommended as the initial treatment and is often effective for terminating SMVT. On the other hand, lidocaine had been widely used to terminate VT but in several studies its efficacy seemed to be limited. Lidocaine is now recommended as an alternative to amiodarone or for SMVT associated with acute myocardial ischemia or infarction. Recently, intravenous amiodarone became available in Japan and its indication for treatment of tachyarrhythmia seems to be expanding. It is indicated for SMVT that is hemodynamically unstable, refractory to conversion with countershocks or recurrent despite procainamide or other agents, and comparative studies of the drugs are still necessary for correct management of SMVT. In fact, the efficacy of amiodarone for terminating stable SMVT is relatively low. Another class III drug, nifekalant, is indicated in Japan for refractory ventricular tachyarrhythmia, and it is effective but associated with excessive QT prolongation and a risk of developing torsades de pointes.

We conducted a retrospective survey of the efficacy of procainamide in terminating SMVT and in the present study we discuss our results with those of previous studies. The efficacy of lidocaine in SMVT cases experienced during the same time period was also investigated.

**Methods**

**Patients**
From the records of consecutive patients with SMVT ad-
Table 1. Clinical Characteristics of the Patients Treated With Procainamide or Lidocaine

<table>
<thead>
<tr>
<th>Patients</th>
<th>Procainamide</th>
<th>Lidocaine</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>70</td>
<td>20</td>
<td>–</td>
<td></td>
</tr>
<tr>
<td>M/F</td>
<td>50/20</td>
<td>13/7</td>
<td>NS</td>
</tr>
<tr>
<td>Mean age</td>
<td>57±26</td>
<td>61±12</td>
<td>NS</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Underlying heart disease</th>
<th>Procainamide</th>
<th>Lidocaine</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Previous MI</td>
<td>18</td>
<td>4</td>
<td>NS</td>
</tr>
<tr>
<td>IDCM</td>
<td>10</td>
<td>3</td>
<td>NS</td>
</tr>
<tr>
<td>HCM</td>
<td>4</td>
<td>1</td>
<td>NS</td>
</tr>
<tr>
<td>ARVC</td>
<td>9</td>
<td>3</td>
<td>NS</td>
</tr>
<tr>
<td>Cardiac sarcoidosis</td>
<td>5</td>
<td>2</td>
<td>NS</td>
</tr>
<tr>
<td>LV aneurysm</td>
<td>4</td>
<td>1</td>
<td>NS</td>
</tr>
<tr>
<td>Postcardiac surgery</td>
<td>4</td>
<td>2</td>
<td>NS</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>16</td>
<td>4</td>
<td>NS</td>
</tr>
</tbody>
</table>

MI, myocardial infarction; IDCM, idiopathic dilated cardiomyopathy; HCM, hypertrophic cardiomyopathy; ARVC, arrhythmogenic right ventricular dysplasia; LV aneurysm, arrhythmogenic left ventricular aneurysm of unknown cause.

Table 2. Echo- and Electrocardiographic Characteristics of the Patients Treated With Procainamide or Lidocaine

<table>
<thead>
<tr>
<th>SMVT</th>
<th>Procainamide (n=70)</th>
<th>Lidocaine (n=20)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>LA dimension</td>
<td>42±8</td>
<td>40±8</td>
<td>NS</td>
</tr>
<tr>
<td>LV diastolic dimension</td>
<td>54±10</td>
<td>54±8</td>
<td>NS</td>
</tr>
<tr>
<td>LV ejection fraction</td>
<td>50±16</td>
<td>46±15</td>
<td>NS</td>
</tr>
<tr>
<td>SMVT Morphology (LBBB/RBBB)</td>
<td>32/38</td>
<td>32/61</td>
<td>NS</td>
</tr>
<tr>
<td>Cycle length (ms)</td>
<td>329±55</td>
<td>32±61</td>
<td>NS</td>
</tr>
</tbody>
</table>

LV, left ventricle; SMVT, sustained monomorphic ventricular tachycardia; LBBB/RBBB, left bundle branch block/right bundle branch block.

Diagnosis of SMVT
VT was diagnosed from the ECG recordings: (1) wide regular tachycardia (>100 beats/min), (2) P-QRS dissociation, and (3) fusion complexes between the basic QRS morphology and that of VT. SMVT showed a uniform QRS morphology in 12-lead ECG during tachycardia lasting more than 30 s. Actually, SMVT lasted more than 30 min after the onset of tachycardia in all because it developed out of the hospital and patients were then transferred to hospital.

All patients underwent EPS after admission and SMVT was confirmed from the induced tachycardia, which showed a QRS morphology identical to that of the clinical VT.18,19

Drug Administration
Before the administration of drugs, it was confirmed that the patients were alert and responsive and that the delivery of DC shock was available if required. Procainamide is the first-line drug used by us to terminate stable SMVT and it is given intravenously at 100 mg over 1–2 min. The endpoint was termination of tachycardia or the dosage reaching 150 mg.6,16

Both drugs were terminated when physicians observed no slowing of the tachycardia rate or a tendency of acceleration or signs of deteriorating consciousness. The 2 drugs were singly administered in a drip after termination of VT if needed.

Exclusion Criteria
Patients with acute chest pain, suggestive of acute myocardial infarction or angina pectoris, were excluded from the analysis. Acute myocardial ischemia on the ECG was also excluded after termination of SMVT. Subsequent coronary angiography was used to classify the underlying heart diseases as ischemic or non-ischemic.

Those who required DC shocks for unstable VT resulting in deterioration of consciousness or those who received pharmacological therapy or electrical intervention at another hospital before administration were also excluded, as were cases of spontaneous termination of SMVT before arrival at hospital. Patients who had no demonstrable heart disease and whose SMVT showed a right bundle branch block (RBBB) pattern with superior axis and were responsive to verapamil were also excluded from the present study.

Data Analysis
The clinical profiles of the 2 groups treated with procainamide or lidocaine were compared. The response of the cycle length (CL) of SMVT to the drug just prior to termination was determined, as well as the efficacy in terminating SMVT.

The clinical data and the dosages of the drugs were compared between the responders and nonresponders without termination of SMVT by either drug. In the procainamide-treated group, the cumulative efficacy of termination of tachycardia was plotted against the increments of dosage.

The numerical data are presented as mean±SD and comparisons between the 2 groups were made using the non-paired t-test. The prevalence or incidence was compared using the chi-square test. A P-value <0.05 was considered significant.

The protocol of the study was approved by the Ethics Committee of Niigata University School of Medicine.

Results

Clinical Characteristics
Previous myocardial infarction was found in 18/70 (25.7%) and 4/20 (20.0%) in the procainamide and lidocaine groups, respectively. Dilated or hypertrophic cardiomyopathy, arrhythmogenic right ventricular disease, and cardiac sarcoidosis were similarly found in both groups. Other heart diseases...
included arrhythmogenic left ventricular aneurysm unrelated to coronary heart disease and postoperative cases of tetralogy of Fallot or double-outlet right ventricle. SMVT of undetermined causes were also found in both groups (Table 1).

ECG after termination of SMVT showed sinus rhythm in all except 2 patients with atrial fibrillation and 1 with a pacemaker for atrioventricular block. Echocardiographic study was performed after admission and the left atrial dimension, end-diastolic dimension of the left ventricle and the ejection fraction did not differ between the groups treated with procainamide and lidocaine: 42±8 mm vs 40±8 mm for the left atrial dimension, 54±10 mm vs 54±8 mm for the left ventricular dimension and 50±16% vs 46±15% for the ejection fraction, respectively.

All patients were alert and responsive. Systolic blood pressure was 91±25 mmHg (range 40–150 mmHg). The CL of VT before drug administration was similar in both groups: 329±55 vs 324±61 ms for the procainamide- and lidocaine-treated groups, respectively (Table 2). The QRS morphology was RBBB/LBBB in 32/38 in the procainamide group and RBBB/LBBB in 8/12 in the lidocaine group.

**Drug Efficacy**

The mean doses were 358±50 mg for procainamide and 81±30 mg for lidocaine, and VT was terminated in 53/70 (75.7%) with procainamide (Figure 1) and 7/20 (35.0%) with lidocaine. Just prior to termination, the CL of SMVT was significantly prolonged with procainamide to 399±63 ms (P<0.001) but not with lidocaine to 333±55 ms (NS). The prolongation of the CL was greater with procainamide (19% on average) than with lidocaine (3% on average) (P<0.05). Of the cases of successful termination, SMVT was terminated in 80% with procainamide at <400 mg and an additional 20% at 800 mg (Figure 2). In 4 nonresponders to lidocaine, procainamide was given and terminated SMVT in 3. In 3 nonresponders to procainamide, lidocaine (2) or mexiletine (1) was given, but SMVT was terminated in only 1. DC shock was used in 16 nonresponders (22.9%) to procainamide and 10 nonresponders (50%) to lidocaine.

The QRS duration was prolonged with procainamide but not with lidocaine: 118±39 ms vs 93±17 ms (P=0.066) and the QTc intervals were 498±66 or 469±48 ms$^{1/2}$ when VT was terminated by procainamide or lidocaine, respectively. Blood pressure was 118±23 mmHg (systolic) and 73±14 mmHg (diastolic) when SMVT was terminated and heart rate was 67±13 beats/min.

**Comparison of Responders and Nonresponders**

In the responders to procainamide, SMVT was terminated at a mean dose of 346±190 mg and those without termination received a higher dose of procainamide: 580±264 mg (P=0.014). There were no differences between the responders and nonresponders to procainamide in the echocardiographic or other characteristics of SMVT.

In the responders to lidocaine, SMVT was terminated at a mean dose of 68±30 mg and those without termination received 82±28 mg (P=0.46). Again, there were no differences in the echocardiographic or other characteristics of SMVT between the responders and nonresponders.

If blood pressure was stable or rose upon slowing of the tachycardia rate by the drugs, higher doses were administered. However, the drugs were discontinued before reaching the maximal doses in cases of low blood pressure or signs of a deterioration in consciousness.
VT Termination With Procainamide and Lidocaine

Adverse Effects

The QRS duration was wider in the procainamide group compared with the lidocaine group: 118±38 ms vs 93±17 ms (P=0.066) when tachycardia was terminated by either drug.

When hemodynamics remained unstable or there was signs of deterioration in consciousness, the drug was discontinued and the patients underwent electrical cardioversion. No major side-effects were observed in any patient.

Discussion

In the present study, procainamide terminated 75.7% of SMVT whereas lidocaine was effective in only 35.0%. There were no differences between the 2 treatment groups in the underlying heart diseases or in the ejection fraction of the left ventricle. The results seem compatible with those reported earlier8–15 and summarized in Table 3, and could form the basis for recommending procainamide as the initial treatment of stable SMVT.

Since an early report by Wellens et al,7 many electrophysiologists have used procainamide to terminate SMVT and EPS-guided selection of antiarrhythmic drugs also suggests procainamide.22 This drug might facilitate the induction of SMVT that is non-inducible in the baseline EPS.22,23 Accordingly, we have been using procainamide as the drug of first choice in terminating SMVT since we started performing EPS.23,24

On the other hand, lidocaine also has some benefits: it is easy to administer and works rapidly,25 and it has been previously recommended as the drug of first choice to terminate VT.26 It is still recommended even now by some workers.25 However, comparative studies have shown that procainamide is superior to lidocaine in terminating SMVT and in the present study procainamide was effective in 75.7% of cases while the efficacy of lidocaine was only 35.0% (Table 3).

The mechanism in most cases of SMVT is considered to be reentry, as suggested by the ability to induce or terminate tachycardia by programmed electrical stimulation.23,24 Demonstration of the phenomenon of transient entrainment is another strong piece of evidence of a reentrant mechanism.22,23,27,28 SMVT is usually unrelated to acute myocardial ischemia, but arises when there is scarring and residual myocardial tissue in which the myocardial cells are normally repolarized.29 Endocardial mapping may show fractionated or continuous electrograms at the site of origin of SMVT,30 and at the critical site, some cases of SMVT can be successfully ablated.31 Class I antiarrhythmic drugs preferentially depress the conduction within the area of slow conduction,32–34 and procainamide is considered to result in termination by increasing the refractoriness within that area.35,36 Caution needs to be paid to a fall in blood pressure when procainamide is given to patients with cardiac dysfunction.37 Furthermore, long-term use of procainamide is limited because of its lower efficacy in preventing recurrence of SMVT and its adverse extra-cardiac effects.38

The efficacy of lidocaine in terminating SMVT is limited and though rarely, it can cause hemodynamic deterioration.11,12,23 It is effective for tachyarrhythmia arising from ischemic myocardium because it binds preferentially to de-

Table 3. Efficacy of Procainamide and Lidocaine for Terminating Sustained Monomorphic Ventricular Tachycardia

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>No. of patients</th>
<th>Termination rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Procainamide</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wellens</td>
<td>1977</td>
<td>12</td>
<td>83%</td>
</tr>
<tr>
<td>Callan</td>
<td>1992</td>
<td>15</td>
<td>93%</td>
</tr>
<tr>
<td>Gorgels*</td>
<td>1996</td>
<td>15</td>
<td>80%</td>
</tr>
<tr>
<td>Present study</td>
<td>2009</td>
<td>70</td>
<td>76%</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>112</td>
<td>80%</td>
</tr>
<tr>
<td>Lidocaine</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Armengol</td>
<td>1989</td>
<td>20</td>
<td>19%</td>
</tr>
<tr>
<td>Griffith</td>
<td>1990</td>
<td>24</td>
<td>30%</td>
</tr>
<tr>
<td>Ho*</td>
<td>1994</td>
<td>33</td>
<td>18%</td>
</tr>
<tr>
<td>Somberg*</td>
<td>2002</td>
<td>11</td>
<td>27%</td>
</tr>
<tr>
<td>Marill</td>
<td>1997</td>
<td>35</td>
<td>29%</td>
</tr>
<tr>
<td>Present study</td>
<td>2009</td>
<td>20</td>
<td>35%</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>143</td>
<td>26%</td>
</tr>
</tbody>
</table>

*Randomized control study. The others are retrospective case series.
pressed fast channels and is effective in preventing VF in acute myocardial infarction. However, lidocaine might cause an acceleration of the tachycardia rate. Based on these findings, lidocaine is indicated as the second-line drug in recent guidelines.

In Japan, intravenous amiodarone has become available and is the first-line drug for unstable or refractory VT. Its efficacy for terminating stable SMVT has been reported as approximately 20–67%. Nifekarant, another class III drug, is indicated for refractory or unstable VT and is associated with prolongation of the QT interval.

**Study Limitations**

This was a retrospective study of a relatively small number of cases of SMVT. However, previous studies, mainly from foreign countries, also involved a limited number of patients and their results were comparable to ours and are evidence for the correct drug choices in the initial management of SMVT.

The study was not randomized and involved a small number of cases in the lidocaine-treated group because we have been using procainamide as the first-line therapy to terminate SMVT since we started performing EPS. Procainamide has been used in most SMVT cases, but some cardiologists, especially younger doctors, tend to choose lidocaine, depending on their knowledge of the literature. Because the use of lidocaine was related to physician preference, its low efficacy in terminating SMVT might not be related to the bias in patient selection.

Finally, the maximal doses of either drug were rarely given, because although the patients were alert and responsive, blood pressure was often very low. If there was no slowing of the tachycardia after a rise in blood pressure, or impending deterioration in consciousness was suggested, drug administration was stopped and the patients were prepared for DC cardioversion under general anesthesia.

In conclusion, procainamide, a relatively older drug, is effective in terminating SMVT associated with structural heart diseases and can be the drug of first choice. The efficacy of lidocaine was low and its use seems to be limited.

**Disclosure**

Conflict of Interest: none to declare.

**References**


