ELECTROPHYSIOLOGIC CHANGES BEFORE ONSET OF VENTRICULAR TACHYARRHYTHMIAS DURING PARTIAL REPERFUSION FOLLOWING SEVERE MYOCARDIAL ISCHEMIA IN DOGS

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We examined the electrophysiologic changes before an onset of ventricular tachyarrhythmia during partial reperfusion following severe myocardial ischemia. The left anterior descending coronary artery was occluded and cannulated below the occluded portion in 26 dogs. To deplete collateral flow into the ischemic myocardium, retrograde blood flow was induced for 20 min. Then, in all dogs except 7 with ventricular fibrillation during retrograde blood flow, partial reperfusion through collateral flow into the ischemic myocardium was produced by stopping the retrograde flow. Within 2 min of partial reperfusion, sustained ventricular tachycardia (VT) occurred in 7 dogs (group A) and non-sustained VT degenerating ventricular fibrillation occurred in 11 dogs (group B) of the remaining 12 dogs. In 6 dogs of group A and 9 of group B, epicardial conduction block appeared 5.0±2.2 and 3.5±1.3 min after ischemia. This was followed by fractionated electrical activities 15.2±3.2 and 11.7±3.3 min after ischemia. In group A, the fractionation had a slight change in configuration and a small increase in amplitude before the onset of VT during reperfusion; in group B, new deflections with large amplitude emerged before it. There was a significant difference in the amplitude (0.38±0.2 vs 0.67±0.3 mV, p<0.025) between the 2 groups, although there was no significant difference in the amplitude (0.33±0.2 vs 0.23±0.1 mV) of the fractionation just before reperfusion. Our results show that slight improvement in fractionation induces sustained VT, and new deflections induce non-sustained VT degenerating ventricular fibrillation, even during partial reperfusion. (Jpn Circ J 1992; 56: 1012-1021)

THE canine heart is widely used as an experimental model of the human heart although it has good collateral flow and relatively wide variation of flow compared to the human heart4-5. In dogs, partial release of occluded coronary arteries has been shown to suppress reperfusion ventricular tachyarrhythmias4,5 in contrast to full release6-10. Retrograde blood flow after coronary occlusion has been shown to deplete collat-

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eral flow into the ischemic myocardium, and to cause severe myocardial ischemia.\textsuperscript{11–16} Eng et al\textsuperscript{13} and our previous study\textsuperscript{14} demonstrated that myocardial blood flow during retrograde blood flow averaged 4.4 ml/min/100g in all layers and 4.7 ml/min/100g in the subepicardium. Using dogs with ischemia due to retrograde blood flow, we found that reperfusion ventricular fibrillation was not prevented by partial reperfusion when antecedent ischemia was severe.\textsuperscript{14} However, changes in conduction abnormalities before an onset of reperfusion ventricular fibrillation were not analysed, although maximal conduction delay during antecedent ischemia was examined.

In the present study, ventricular tachyarrhythmias occurred during partial reperfusion following 20-min ischemia due to retrograde blood flow in dogs. Sustained ventricular tachycardia (VT) in 7 of 19 dogs, and non-sustained ventricular tachycardia degenerating into fibrillation occurred in 11 of the remaining 12 dogs. The purpose of this study was to examine the difference in conduction abnormalities between the 2 types of ventricular tachyarrhythmia.

METHODS

\textit{Animal preparation}

Twenty-six mongrel dogs weighing 15–30 kg were anesthetized with intravenous sodium pentobarbital (30 mg/kg). Mechanical ventilation was provided using room air through a cuffed endotracheal tube with a Harvard dog ventilator. Thoracotomy was performed through the 5th left intercostal space, the pericardium was cut, and the heart was suspended in a pericardial cradle. The left anterior descending coronary artery (LAD) was dissected free from the surrounding tissue distal to the first diagonal branch.

A unipolar epicardial electrogram (UEG) was recorded using a 3 mm diameter disc placed at the ischemic center, and Wilson's terminal was used as the indifferent electrode. One pair of bipolar electrodes was inserted into the right atrial appendage to judge ventricular arrhythmia. Eight pairs of epicardial bipolar electrodes (interelectrode distances 1.5 mm) were inserted 2.0 mm into the subepicardium within the ischemic zone to record bipolar epicardial electrograms (BEG). Four of the 8 BEGs were recorded because 4 channels of an 8 channel recorder were used to record other parameters, and the remaining 4 BEGs were recorded using a selector switch in an early phase of ischemia. Four of the 8 ECGs in which conduction abnormalities occurred early were selected, were then recorded continuously. In addition to the above electrograms and lead II ECG, aortic blood pressure was monitored using standard techniques in which a peripheral artery was cannulated. The UEG was amplified at a time constant of 2 sec, and BEG was amplified with frequency limits between 50 and 1000 Hz. The heart rate was maintained at a rate of 160 bpm with bipolar pacing electrodes sutured in the right atrial appendage. When the heart rate was more than 160 bpm, the sinus node was destroyed with an injection of 30% formalin. The pacing was performed using a programmed digital stimulator (San-ei Co.) that delivered rectangular impulses of 2 msec duration at an intensity of twice the diastolic threshold.

Recordings were made on an 8 channel medical recorder (Nihon Kohden Co.). The paper speed was 10 or 100 mm/sec. Recordings were also stored on a 7 channel magnetic tape recorder (Sony Co.) and replayed so that selected sections could be rerecorded at paper speeds of 25 to 200 mm/sec for detailed analysis.

\textit{Experimental protocol}

The LAD ligated in the dissected portion, and was immediately cannulated below the ligation with a metal cannula (2.4 and 2.0 mm, outer and inner diameters, respectively) which was connected to a polyethylene tube. The end of the tube was then vented to the atmosphere for 20 min so that collaterals could flow retrogradely (retrograde blood flow). The retrograde blood dripped into a beaker from the tube, and was injected into the left femoral vein using a roller pump set at the same speed as the retrograde flow (1.0 to 7.5 ml/min). Seven out of 26 dogs died of VF during the retrograde blood flow. In the 19 surviving dogs, partial reperfusion through collateral flow into the ischemic zone was achieved by stopping the retrograde blood flow, as in our previous

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### TABLE I ELECTROPHYSIOLOGIC CHANGES AFTER PARTIAL REPERFUSION

<table>
<thead>
<tr>
<th>Group</th>
<th>Onset (sec)</th>
<th>Maximal Heart Rate (bpm)</th>
<th>Amplitude of FEA (mV)</th>
<th>%increase (onset;RBF20°)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>RBF20°</td>
<td>onset of VT</td>
</tr>
<tr>
<td>A-1</td>
<td>90</td>
<td>412</td>
<td>0.30</td>
<td>0.37</td>
</tr>
<tr>
<td></td>
<td>228*</td>
<td>273</td>
<td>0.38</td>
<td>0.32</td>
</tr>
<tr>
<td>2</td>
<td>20</td>
<td>414</td>
<td>0.28</td>
<td>0.70</td>
</tr>
<tr>
<td>3</td>
<td>110</td>
<td>349</td>
<td>0.28</td>
<td>0.77</td>
</tr>
<tr>
<td>4</td>
<td>58</td>
<td>389</td>
<td>0.14</td>
<td>0.21</td>
</tr>
<tr>
<td>5</td>
<td>25</td>
<td>343</td>
<td>_**</td>
<td>_</td>
</tr>
<tr>
<td>6</td>
<td>4</td>
<td>405</td>
<td>0.27</td>
<td>0.30</td>
</tr>
<tr>
<td>7</td>
<td>12</td>
<td>380</td>
<td>0.26</td>
<td>0.28</td>
</tr>
<tr>
<td>Mean (SD)</td>
<td>46 (41)</td>
<td>346 (67)</td>
<td>0.33 (0.2)</td>
<td>0.38 (0.2)</td>
</tr>
<tr>
<td>B-1</td>
<td>39</td>
<td>400</td>
<td>0.10</td>
<td>0.42</td>
</tr>
<tr>
<td>2</td>
<td>24</td>
<td>243</td>
<td>0.37</td>
<td>0.88</td>
</tr>
<tr>
<td>3</td>
<td>37</td>
<td>406</td>
<td>0.11</td>
<td>0.43</td>
</tr>
<tr>
<td>4</td>
<td>16</td>
<td>369</td>
<td>0.34</td>
<td>0.96</td>
</tr>
<tr>
<td>5</td>
<td>92</td>
<td>300</td>
<td>0.26</td>
<td>0.90</td>
</tr>
<tr>
<td>6</td>
<td>51</td>
<td>245</td>
<td>_**</td>
<td>_</td>
</tr>
<tr>
<td>7</td>
<td>14</td>
<td>380</td>
<td>0.29</td>
<td>0.62</td>
</tr>
<tr>
<td>8</td>
<td>10</td>
<td>377</td>
<td>0.28</td>
<td>0.75</td>
</tr>
<tr>
<td>9</td>
<td>109</td>
<td>440</td>
<td>0.10</td>
<td>0.21</td>
</tr>
<tr>
<td>10</td>
<td>44</td>
<td>254</td>
<td>0.25</td>
<td>0.89</td>
</tr>
<tr>
<td>11</td>
<td>20</td>
<td>469</td>
<td>_**</td>
<td>_</td>
</tr>
<tr>
<td>Mean (SD)</td>
<td>41 (32)</td>
<td>353 (80)</td>
<td>0.23 (0.1)</td>
<td>0.67 (0.3)</td>
</tr>
</tbody>
</table>

A vs B ns ns ns p<0.025 p<0.001

*Group A=sustained VT, group B=non-sustained VT degenerating ventricular fibrillation, Onset=onset of VT after partial reperfusion, *excluded from the mean, FEA=fractionated electrical activities, RBF20°=20 min after retrograde blood flow, **dogs without FEA.

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**Measurement parameters and definition**

ST segment elevation was measured 120 msec after the beginning of the QRS complex in the UEG. Conduction time was measured from the beginning of the QRS wave in the ECG to the end of the largest deflection in the BEG. Conduction block was defined as disappearance of the largest deflection in the BEG, and an onset of conduction block was defined as occurrence of 2 to 1 block preceding disappearance of the largest deflection. "Fractionated electrical activity" in the BEG which was recorded from the ischemic zone was defined as a signal with an amplitude >0.1 mV and total duration of at least 60 ms. Ventricular arrhythmias were observed for 5 min after partial reperfusion. Sustained VT was defined as a VT lasting for more than 30 sec and with a regular morphologic pattern. Non-sustained VT was defined as VT lasting for less than 30 sec and with a polymorphic pattern. According to the occurrence of VT after partial reperfusion, dogs were divided into 2 groups: Group A (7 dogs) had sustained VT; Group B (11 of the remaining 12 dogs) had non-sustained VT degenerating into fibrillation. Another 1 of the 19 dogs had non-sustained VT which did not degenerate into ventricular fibrillation.
Fig. 1. Spontaneous initiation of sustained ventricular tachycardia (VT).
Recordings consist of surface electrocardiogram (II), unipolar epicardial electrogram (UEG) from the ischemic zone, a right atrial electrogram (RA), and 4 bipolar epicardial electrograms from the ischemic zone (IZ1, IZ2, IZ3, IZ4). “A” shows a recording before retrograde blood flow. “B” was recorded 90 sec after partial reperfusion and shows spontaneous initiation of sustained VT. The first 10 ventricular cycles of sustained VT were characterized by slight variation in the cycle length and in the morphology of the QRS complexes. Fractionated electrical activities were detected in IZ1 and IZ3.

Fig. 2. Self-termination of sustained VT.
A: before retrograde blood flow. B: 30 sec after the onset of sustained VT, which occurred 20 sec after partial reperfusion. Fractionated electrical activities were detected in IZ1 and IZ2 consist of 3 deflections. C: self-termination 57 sec after the onset of sustained VT. During sustained VT, degeneration in the regularity of fractionated electrical activities in IZ3 was detected.

Statistical analysis
Data were expressed as mean ± SD, and were statistically analyzed using Student’s T-test. The incidence of conduction block was analyzed using Fisher’s exact test. A p value less than 0.05 was taken as significant.

RESULTS
1. Characteristics of sustained VTs and nonsustained VTs degenerating ventricular fibrillation
Table I shows the characteristics of these VTs. In group A, 8 sustained VTs occurred
in the 7 dogs within 228 sec after partial reperfusion. An example of initiation of sustained VT is shown in Fig. 1. Six of these 8 sustained VTs had self-termination (Fig. 2), while the other 2 (A-6 and A-7) degenerated into ventricular fibrillation by a fall in blood pressure during sustained VT. The duration of 8 sustained VTs averaged 159±62.8 sec (range 38 to 660 sec). In group B, 11 nonsustained VTs degenerating into ventricular fibrillation occurred within 109 sec after partial reperfusion. There were no significant differences in the average onset time of VT and the average maximal heart rates between the 2 groups. The morphological pattern of sustained VT in group A was monomorphic in 6 (Fig. 1 and 2) and was bimorphic in the other 2 (Fig. 3).

2. Electrophysiologic changes during both retrograde blood flow and partial reperfusion

Table II shows data from the 8 BEG with the greatest conduction abnormality in the 2 groups during both retrograde blood flow (ischemia) and partial reperfusion. There was no significant difference in the incidence of conduction block, which appeared during ischemia, between groups A and B (6/7 vs 9/11). In addition, the average onset time of conduction block after ischemia was not different between groups A and B (5.0±2.2 vs 3.5±1.3 min). In the dogs with conduction block, fractionated electrical activities appeared at 15.2±3.2 min after ischemia in group A and at 11.7±3.3 min in group B (not significant). Ventricular arrhythmias decreased from a few min after the onset of conduction block to the onset of the fractionated electrical activities, and then increased during retrograde blood flow. The fractionated electrical activities were only slightly changed by 20 min of ischemia.

Typical recordings (A-1) in group A are shown in Fig. 4. An epicardial bipolar electrogram within the ischemic zone (IZI) showed a conduction time of 120 msec at the beginning of the conduction block, 3 min after ischemia (B). Fractionated electrical activities suddenly appeared at 18 min after ischemia, and had not changed by 20 min of ischemia. Twenty min after ischemia (C), the fractionated electrical activities comprised 3 deflections (arrow). Sixty sec after partial reperfusion (D), the configuration of the fractionated electrical activities suddenly changed and their amplitude increased. That
Fig. 4. Typical bipolar epicardial electrogram changes in group A.

Fig. 5. Typical bipolar epicardial electrogram changes in group B.

is, the 3rd deflection (arrow) appeared earlier and its amplitude (0.37 mV) was greater than it had been (0.3 mV) just before partial reperfusion. During an occurrence of sustained VT (E) which occurred 90 sec after partial reperfusion, the 1st deflection seemed to combine with the 2nd, and the 3rd deflection appeared later and its amplitude was lower than that recorded 30 sec earlier (D). Similar findings were also observed in the
TABLE III  RISK REGION, RETROGRADE FLOW AND SYSTOLIC BLOOD PRESSURE

<table>
<thead>
<tr>
<th></th>
<th>Risk Region (% whole heart)</th>
<th>Retrograde Flow (ml/min)</th>
<th>Systolic Blood Pressure (mmHg) before RBF</th>
<th>20 min after RBF</th>
</tr>
</thead>
<tbody>
<tr>
<td>A (n=7)</td>
<td>30.4±5.8</td>
<td>3.2±1.8</td>
<td>140±11.5</td>
<td>137±10.8</td>
</tr>
<tr>
<td>B (n=11)</td>
<td>32.7±5.4</td>
<td>2.9±1.6</td>
<td>143±12.3</td>
<td>140±11.6</td>
</tr>
<tr>
<td>p</td>
<td>ns</td>
<td>ns</td>
<td>ns</td>
<td>ns</td>
</tr>
</tbody>
</table>

other 5 dogs with conduction block (Table I).

Typical recordings (B-4) in group B are shown in Fig. 5. An epicardial bipolar electrogram within the ischemic zone (IZ2) showed a conduction time of 120 msec at the beginning of the conduction block, 4 min after ischemia (B). Fractionated electrical activities suddenly appeared after 15 min of ischemia, and were very slightly changed by 20 min of ischemia. Twenty min after ischemia (C), the fractionated electrical activities comprised 2 deflections (arrow). Sixteen sec after partial reperfusion (D), the new deflections (arrow) suddenly emerged on a downhill slope of the 2nd deflection of the fractionated electrical activities. The amplitude of the new deflections was large and the duration was long. The amplitude (0.96 mV) of the new deflections was markedly higher than that (0.34 mV) of the fractionated electrical activities just before partial reperfusion.

Similar findings were also observed in the other 8 dogs with conduction block.

As shown in Table I, there was a significant difference in the amplitudes of the deflections before VT during partial reperfusion between the 2 groups (0.38±0.2 mV, the fractionated electrical activities in group A vs 0.67±0.3 mV, the new deflection in group B), although there was no significant difference in the amplitudes of the fractionated electrical activities 20 min after retrograde blood flow between the 2 groups (0.33±0.2 mV vs 0.23±0.1 mV). There was also a significant difference in % increase (before an onset of VT/20 min after retrograde blood flow) in the amplitude of the deflections between the 2 groups (120±15% vs 303±78%, p<0.001).

Fig. 6 shows the mean ST segment elevation. There was no significant difference between groups A and B during either ischemia or partial reperfusion. In comparison to
the ST segment elevation just after 20 min of ischemia, the value measured before the onset of VT during partial reperfusion was slightly lower in group A (23.1 ± 7.6 vs 21.3 ± 7.8 mV, p < 0.025) but was markedly lower in group B (25.7 ± 8.3 vs 19.7 ± 7.7 mV, p < 0.005). The degree of the improvement in ST segment elevation during partial reperfusion was greater in group B than in group A (p < 0.05).

3. Systolic blood pressure, risk region and retrograde blood flow

As shown in Table III, there were no significant differences in systolic blood pressure between the 2 groups either just before ischemia or 20 min after ischemia. Furthermore, there were no significant differences between the 2 groups in either the risk region (% whole heart) or the retrograde blood flow.

DISCUSSION

Most reperfusion ventricular tachyarrhythmias are induced by complete release of an occluded coronary artery\(^{5-10}\) but few of them are induced by partial reperfusion\(^5,5,14\). Sheehan and Epstein\(^4\) showed that 11 of 20 dogs developed ventricular fibrillation during partial reperfusion. However, the degree of their partial reperfusion was mild because mean peak flow during reperfusion was 163% of baseline in those 11 dogs. Kabell et al\(^5\) also showed that ventricular fibrillation did not occur in any of their 15 dogs undergoing 2-stage reperfusion, while VT occurred within 2 min after reperfusion in 7 of those 15. In addition, our previous study\(^{14}\) showed that ventricular fibrillation occurred (9 of 13 dogs) during partial reperfusion despite a small amount of reperfusion flow.

In the present study, as in our previous study\(^{14}\), partial reperfusion through collateral flow into the ischemic myocardium was produced by stopping the retrograde blood flow. This can be regarded as partial reperfusion, because ST segment elevation decreased for a short period after reperfusion. Moreover, in our previous study\(^{14}\) we showed that the myocardial blood flow during partial reperfusion was greater than that just prior to it, although the duration of retrograde blood flow was only 10 min. As a result, the partial reperfusion in this study caused an improvement in severe conduction abnormalities and ST segment elevation.

During partial reperfusion in the present study, sustained VT occurred in 7 of 19 dogs, and non-sustained VT degenerating into ventricular fibrillation occurred in 11 of the remaining 12 dogs. There was a slight change in configuration and a small increase in the amplitude of fractionated electrical activities for a short time after partial reperfusion and before sustained VT in 6 of 7 dogs in group A. The increase in the amplitude of fractionated electrical activities was observed in the late component, and this component moved toward the early component as reperfusion progressed. This indicates that the ischemia-induced conduction abnormalities improved mildly during partial reperfusion. This mild improvement was induced by a gradual progression of reperfusion, as shown by a slight improvement in ST segment elevation. On the other hand, before non-sustained VT degenerating ventricular fibrillation, the new deflections with large amplitude and long duration emerged on the fractionated electrical activities for a short time after partial reperfusion in 9 of 11 dogs in group B. Murdock et al\(^9\) observed that a new fractionated electrical activity emerged, associated with an occurrence of ventricular arrhythmias, during full reperfusion. They regarded this finding as an improvement in conduction delay induced by full reperfusion, despite a reemergence of fractionated electrical activities. Thus, in group B, as in group A, the ischemia-induced conduction abnormalities also improved during partial reperfusion, and the degree was marked because of an abrupt progression of reperfusion, as shown by a great improvement in ST segment elevation. Gardner et al\(^{16}\) have shown that different components of the fractionated electrical activities coincide with activation of different adjacent regions. Therefore, it is suggested that heterogenous activation occurred within the different adjacent regions in which the bipolar electrograms were recorded. The degree of the heterogeneity is thought to be mild in group A and marked in group B, resulting in sustained VT in the former and non-sustained VT degenerating into ventricular fibrillation in the latter.

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The mechanism for our VT is not clear because 8 electrograms are not adequate in number to evaluate conduction in the ischemic zone. In canine myocardial infarction models, sustained VT has been shown to be related to fractionated electrical activities which provide the conditions necessary for reentry. In our model, fractionated electrical activities also continued to exist during reperfusion, although there was a qualitative difference in the improvement in fractionated electrical activities between the 2 groups. Moreover, the improvement in fractionated electrical activities suggests that the heterogeneous activation, which is responsible for reentry, occurred during reperfusion. Therefore, reentry may have been responsible for our VT. In addition, the site of reentry in our VT is thought to include the regions in the subepicardium which were recorded by the bipolar electrograms. In a recent study, Pogwizd and Corr showed, using multiple electrodes, that 75% of non-sustained VT during full reperfusion resulted from a non-reentrant mechanism because continuous activation was not apparent and the time from the end of sinus beat to the beginning of VT was not associated with intervening depolarization. This also indicates that an absence of continuous activation is related to a non-reentrant mechanism.

In contrast, Kabell et al proposed that enhanced automaticity rather than reentry was the mechanism for VT during 2-stage reperfusion, because this VT, which lasted for several min, was slow and sensitive to lidocaine, and because they found no fractionation in regional electrograms during reperfusion. However, their reperfusion was performed as a 2-stage procedure consisting of release to a flow-limited stenosis for 2 min followed by complete reperfusion. Therefore, their VT was not the one which occurred during partial reperfusion (first stage), but was the one which occurred during full reperfusion (second stage).

The myocardial blood flow during ischemia with retrograde blood flow is about 50% of that during ischemia without it. In addition, in the canine heart, collaterals have been shown to be located mainly in the epicardial layer. This indicates that retrograde blood flow causes severe conduction abnormalities in the epicardial layer. In our model, at least one of the 8 epicardial bipolar electrograms within the ischemic zone showed severe conduction abnormalities, including conduction block, in 6 of 7 dogs in group A and 9 of 11 dogs in group B. In the present study, there were no significant differences between the 2 groups in the onset times of either conduction block or fractionated electrical activities during ischemia. During ischemia, non-sustained VT occurred, but sustained VT did not occur in either group. In dogs in which collateral blood flow was interrupted by latex embolization, sustained VT was also not observed, although there was severe epicardial embolization delay. On the contrary, Euler et al have shown, in ovine hearts, that sustained VT frequently occur after occlusion of the circumflex coronary artery. They observed that the epicardial conduction delay in the ischemic zone electrograms peaked within the first few min of ischemia and then gradually became less severe over the next 5 to 10 min. They also observed that sustained VT did not occur following severe conduction delay, but occurred following subsequent improvement in the conduction delay. In our 2 groups, slight changes in the configuration and amplitude of fractionated electrical activities occurred over a long period, from the onset of fractionated electrical activities until 20 min of ischemia. It seems that such an improvement develops too slowly to induce sustained VT.

The 20 min of retrograde blood flow in the present model may correspond to an early phase of myocardial infarction in humans with poor collateral circulation, and partial reperfusion may correspond to partial clot lysis which occurs spontaneously. Therefore, just as severe ventricular arrhythmia such as sustained VT or non-sustained VT developing into ventricular fibrillation occurred during partial reperfusion following severe myocardial ischemia in our canine model, it probably also occurs during partial clot lysis in an early phase of myocardial infarction in humans.

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