ROLE OF ELECTROPHYSIOLOGIC TESTING AND CORONARY SPASM PROVOCATION TEST IN SURVIVORS OF CARDIAC ARREST

TOSHIYAMAGUCHI, M.D.

Twenty-one patients were successfully resuscitated from cardiac arrest. Electrocardiograms (ECG) during cardiac arrest were recorded in 14 patients with ventricular fibrillation in 7, ventricular tachycardia in 4, cardiac standstill in three, Torsade de Points in one and atrial fibrillation with rapid ventricular response in 1. Thirteen patients (group I) had structural heart disease or primary ECG abnormality and 8 patients (group II) had no apparent heart disease. Electrophysiologic study (EPS) was performed in 12 patients of group I and 5 of group II. In group I, ventricular tachycardia was induced in 7, and His-ventricular conduction disturbance was demonstrated in 2, and 2 patients with Wolff-Parkinson-White (WPW) syndrome had an effective refractory period of the antegrade accessory pathway <250 msec. No patients in group II showed abnormal EPS findings. Spasm provocation test was performed in 8 patients (2 in group I and 6 in group II). Coronary spasm was induced in 5 patients (1 in group I and 4 in group II). Two patients in group II had positive results of upright-tilt testing. During the follow-up period, 2 patients died suddenly in group I and 1 patient whose cause of cardiac arrest was unknown had a recurrence of cardiac arrest. In group II, all patients whose etiology could be demonstrated by serial examinations had good prognosis. In conclusion, EPS is useful in evaluation of the cause of cardiac arrest especially when patients have structural heart disease, and coronary spasm may be involved in patients with cardiac arrest without apparent heart disease.

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In western countries, survivors of cardiac arrest usually have structural heart diseases, especially coronary heart disease!–3 The role of electrophysiologic study (EPS) is well established in evaluating the cause of cardiac arrest5,6 since the most common cause of sudden cardiac arrest is ventricular tachyarrhythmias and these are induced in EPS2,4,6,8. The most common heart disease observed in such patients is chronic coronary artery disease2,4,6,8 and therapy is chosen on the basis of the results of EPS.

On the other hand, serious ventricular tachyarrhythmias or bradyarrhythmias have been confirmed during spontaneous attacks of vasospastic angina9–11 and 2% to 5% of patients with vasospastic angina may die suddenly during the follow-up period12–14. There are few reports which discuss the role of coronary spasm in cardiac arrest15,16. Since noninvasive examinations in patients with vasospastic angina usually result in normal findings, the provocation test of coronary spasm has been established as useful15,16. Using the provocation test of coronary spasm and EPS, we evaluated: 1)

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Sudden death

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The First Department of Internal Medicine, Niigata University School of medicine, Niigata, Japan
Mailing address: Toshio Yamaguchi, M.D., The First Department of Internal Medicine, Niigata University School of medicine, 1-754 Asahimachi-dori, Niigata 951, Japan

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the role of cardiac arrhythmias, 2) the role of coronary spasm, and 3) the prognosis of patients who have survived cardiac arrest.

SUBJECTS & METHODS

Patient population

Twenty-one consecutive patients (19 males, with a mean age 49 years, range 12 to 76 years) were included in the present study between January, 1988 and May, 1991. They had sudden unexpected collapse and unconsciousness, and cardiopulmonary resuscitation was required for the restoration of consciousness and pulse in the large arteries. The patients required emergency cardiopulmonary resuscitation and/or direct-current cardioversion. Patients in whom cardiac arrest was associated with acute myocardial infarction were excluded; none had infarction within one month of cardiac arrest. Patients who had irreversible brain damage because of unsuccessful cardiopulmonary resuscitation were excluded.

All patients underwent a physical examination, 12-lead electrocardiography, chest X-ray, routine laboratory testing, and two dimensional and color-flow doppler echocardiography. Left ventricular ejection fraction was determined by left ventriculography or echocardiography.

To determine the cause of cardiac arrest, EPS was usually performed first followed by a provocation test of coronary spasm. In a small number of patients, the spasm provocation test was performed first when patients had a history of angina or chest pain just prior to cardiac arrest. If the result of the first test, either the EPS or spasm provocation test, was positive and diagnostic, the other was not performed. When the cause of cardiac arrest was indeterminate following EPS and/or the provocation test of coronary spasm, upright-tilt testing was performed. All cardioactive and vasoactive agents were discontinued at least 5 half-lives before EPS and upright-tilt testing, and at least 24 h before the provocation test of coronary spasm. Informed consent was obtained before each study.

Protocol of EPS

EPS was performed in 17 of 21 patients. The protocol of EPS has been previously described. Briefly, electrode catheters were positioned in the high right atrium, across the tricuspid valve to record a His-bundle ECG, and in the right or left ventricle.

Sinus node function and atrioventricular conduction were evaluated by means of right atrial rapid pacings and extrastimulus technique. Up to triple extrastimuli after 8 beats of ventricular stimuli at two basic cycle lengths (600 msec and 400 msec) and incremental pacings up to 210 bpm for 5–15 sec were given to induce ventricular tachyarrhythmias. When ventricular tachyarrhythmias were not induced, the same protocol was repeated after the administration of isoproterenol to increase the sinus rate by 20%. Stimulation was performed at 2 sites in the right ventricle and one site in the left ventricle. Patients with inducible ventricular tachyarrhythmias underwent serial EPS to select effective antiarrhythmic agents. Positive EPS test was defined as follows; 1) corrected sinus node recovery time ≥ 550 msec, 2) His-ventricular interval ≥ 70 msec or His-ventricular block, 3) effective refractory period of accessory pathway for antegrade conduction ≤ 250 msec, 4) induction of monomorphic ventricular tachycardia (VT) ≥ 6 consecutive complexes. Sustained VT was defined as VT that lasted ≥ 30 sec, or required immediate termination because of hemodynamic deterioration. Nonsustained VT was defined as VT which terminated spontaneously within 30 sec.

Antiarrhythmic agents were defined as effective if VT of 6 or more consecutive complexes were unable to be induced in EPS. If antiarrhythmic agents were not effective, surgical endocardial resection or electrical catheter ablation was performed in selected patients.

Provocation test of coronary spasm

A provocation test of coronary spasm was performed in 8 of 21 patients. The protocol of the test has been described previously. After control coronary angiography, methylergometrine was continuously infused into the coronary artery at a rate of 10 µg/min for up to 5 min. When ischemic change in ECG or chest pain appeared, coronary arteriography was performed immediately. Isosorbide dinitrate was then

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TABLE I PATIENTS WITH STRUCTURAL HEART DISEASE OR PRIMARY ECG ABNORMALITY (GROUP I)

<table>
<thead>
<tr>
<th>Case</th>
<th>Age &amp; sex</th>
<th>Associated heart disease</th>
<th>ECG at cardiac arrest</th>
<th>Symptom</th>
<th>Other occasion</th>
<th>coronary angiography (%)</th>
<th>EF (%)</th>
<th>Result of EPS</th>
<th>Spasm induction</th>
<th>Effective drug</th>
<th>Other therapy</th>
<th>Prognosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>55 M</td>
<td>OMI</td>
<td>Vf</td>
<td>chest pain*</td>
<td>(−)</td>
<td>proximal LAD 75, Diagonal 100, distal RCA 90</td>
<td>32</td>
<td>SVT</td>
<td>(−)</td>
<td>(−)</td>
<td>sudden death</td>
<td>(7 M)</td>
</tr>
<tr>
<td>2</td>
<td>76 M</td>
<td>OMI</td>
<td>VT</td>
<td>chest pain*</td>
<td>(−)</td>
<td>proximal LAD 90</td>
<td>22</td>
<td>SVT</td>
<td>procaainamide</td>
<td>(−)</td>
<td>alive</td>
<td>(29 M)</td>
</tr>
<tr>
<td>3</td>
<td>67 M</td>
<td>OMI</td>
<td>Vf</td>
<td>epigastralgia</td>
<td>epigastralgia</td>
<td>proximal LAD 75, middle RCA 100</td>
<td>32</td>
<td>SVT</td>
<td>(−)</td>
<td>electrical ablation</td>
<td>alive</td>
<td>(6 M)</td>
</tr>
<tr>
<td>4</td>
<td>51 M</td>
<td>mitral stenosis</td>
<td>(−)</td>
<td>chest pain</td>
<td>(−)</td>
<td>normal</td>
<td>63</td>
<td>positive (proximal LAD 100)</td>
<td>diltiazem</td>
<td>(−)</td>
<td>alive</td>
<td>(17 M)</td>
</tr>
<tr>
<td>5</td>
<td>56 M</td>
<td>mitochondrial myopathy</td>
<td>Vf</td>
<td>chest pain</td>
<td>(−)</td>
<td>normal</td>
<td>49</td>
<td>prolonged HV</td>
<td>negative</td>
<td>pacemaker implantation</td>
<td>alive</td>
<td>(11 M)</td>
</tr>
<tr>
<td>6</td>
<td>57 M</td>
<td>DCM</td>
<td>VT</td>
<td>palpitation</td>
<td>syncope</td>
<td>normal</td>
<td>45</td>
<td>SVT</td>
<td>(−)</td>
<td>(−)</td>
<td>operative death</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>61 M</td>
<td>alcoholic cardiomyopathy</td>
<td>VT</td>
<td>chest pain</td>
<td>chest pain</td>
<td>proximal LAD 75</td>
<td>38</td>
<td>SVT</td>
<td>flecaainide</td>
<td>(−)</td>
<td>alive</td>
<td>(39 M)</td>
</tr>
<tr>
<td>8</td>
<td>67 M</td>
<td>idiopathic LV aneurysm</td>
<td>VT</td>
<td>chest pain</td>
<td>chest pain</td>
<td>proximal RCA 50</td>
<td>63</td>
<td>SVT</td>
<td>amiodarone</td>
<td>(−)</td>
<td>alive</td>
<td>(25 M)</td>
</tr>
<tr>
<td>9</td>
<td>34 F</td>
<td>HCM</td>
<td>Vf</td>
<td>(−)</td>
<td>palpitation</td>
<td>normal</td>
<td>73</td>
<td>NSVT</td>
<td>verapamil</td>
<td>(−)</td>
<td>alive</td>
<td>(43 M)</td>
</tr>
<tr>
<td>10</td>
<td>52 M</td>
<td>WPW</td>
<td>Vf</td>
<td>palpitation</td>
<td>palpitation</td>
<td>(−) (58) rapid-af (ERP&lt;250 msec)</td>
<td>−</td>
<td>(−)</td>
<td>surgery</td>
<td>(−)</td>
<td>alive</td>
<td>(40 M)</td>
</tr>
<tr>
<td>11</td>
<td>42 M</td>
<td>WPW</td>
<td>rapid-af</td>
<td>palpitation</td>
<td>palpitation</td>
<td>(−) (64) rapid-af (ERP=230 msec)</td>
<td>−</td>
<td>(−)</td>
<td>electrical ablation</td>
<td>(−)</td>
<td>alive</td>
<td>(12 M)</td>
</tr>
<tr>
<td>12</td>
<td>14 M</td>
<td>long QT</td>
<td>TdP</td>
<td>palpitation</td>
<td>palpitation syncope</td>
<td>(−) (71) negative</td>
<td>−</td>
<td>propranolol</td>
<td>(−)</td>
<td>(−)</td>
<td>alive</td>
<td>(27 M)</td>
</tr>
<tr>
<td>13</td>
<td>29 M</td>
<td>cardiac amyloidosis</td>
<td>cardiac standstill</td>
<td>chest discomfort</td>
<td>syncope</td>
<td>normal</td>
<td>41</td>
<td>prolonged HV</td>
<td>(−)</td>
<td>pacemaker implantation</td>
<td>sudden death</td>
<td>(2 M)</td>
</tr>
</tbody>
</table>

* = symptom only in acute myocardial infarction, (EF) = ejection fraction assessed by echocardiography
ECG = electrocardiogram, EF = ejection fraction, EPS = electrophysiologic study, M = male or month, OMI = old myocardial infarction, LAD = left anterior descending coronary artery, RCA = right coronary artery, SVT = sustained ventricular tachycardia, VT = ventricular tachycardia, Vf = ventricular fibrillation, HV = His-ventricular interval, DCM = dilated cardiomyopathy, LV = left ventricular, HCM = hypertrophic cardiomyopathy, NSVT = nonsustained ventricular tachycardia, WPW = Wolff-Parkinson-White syndrome, ERP = effective refractory period of accessory pathway for antegrade conduction, af = atrial fibrillation, TdP = Torsade de Pointes.
### TABLE II PATIENTS WITHOUT APPARENT HEART DISEASE (GROUP II)

<table>
<thead>
<tr>
<th>Case</th>
<th>Age &amp; sex</th>
<th>ECG at cardiac arrest</th>
<th>Symptom</th>
<th>coronary angiography (%)</th>
<th>EF (%)</th>
<th>Result of EPS</th>
<th>Spasm induction</th>
<th>Effective drug</th>
<th>Prognosis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>at cardiac arrest</td>
<td>other occasion</td>
<td>normal</td>
<td>74</td>
<td>negative</td>
<td>positive (proximal LAD 99)</td>
<td>nifedipine</td>
<td>alive (31 M)</td>
</tr>
<tr>
<td>1</td>
<td>51 M</td>
<td>(-)</td>
<td>(-)</td>
<td>normal</td>
<td>54</td>
<td>-</td>
<td>positive (proximal LAD 100)</td>
<td>nifedipine</td>
<td>alive (20 M)</td>
</tr>
<tr>
<td>2</td>
<td>35 M</td>
<td>(-)</td>
<td>chest pain</td>
<td>normal</td>
<td>75</td>
<td>-</td>
<td>positive (proximal CX 100)</td>
<td>diltiazem</td>
<td>alive (35 M)</td>
</tr>
<tr>
<td>3</td>
<td>56 M</td>
<td>(-)</td>
<td>chest pain</td>
<td>normal</td>
<td>52</td>
<td>-</td>
<td>positive (proximal RCA 100)</td>
<td>nifedipine</td>
<td>alive (29 M)</td>
</tr>
<tr>
<td>4</td>
<td>60 M cardiac standstill</td>
<td>(-)</td>
<td>syncope neack pain</td>
<td>proximal RCA 50</td>
<td>69</td>
<td>negative</td>
<td>negative (proximal RCA 100)</td>
<td>midodrine</td>
<td>alive (13 M)</td>
</tr>
<tr>
<td>5</td>
<td>55 F</td>
<td>(-)</td>
<td>(-)</td>
<td>normal</td>
<td>68</td>
<td>negative</td>
<td>negative (proximal RCA 100)</td>
<td>propranolol</td>
<td>alive (42 M)</td>
</tr>
<tr>
<td>6</td>
<td>43 M cardiac standstill</td>
<td>(-)</td>
<td>syncope</td>
<td>normal</td>
<td>76</td>
<td>negative</td>
<td>negative (proximal RCA 100)</td>
<td>(-)</td>
<td>recurrence (14 M)</td>
</tr>
<tr>
<td>7</td>
<td>12 M</td>
<td>Vf</td>
<td>(-)</td>
<td>syncope</td>
<td>(-)</td>
<td>negative</td>
<td>negative (proximal RCA 100)</td>
<td>(-)</td>
<td>alive (8 M)</td>
</tr>
<tr>
<td>8</td>
<td>50 M</td>
<td>Vf</td>
<td>(-)</td>
<td>normal</td>
<td>74</td>
<td>negative</td>
<td>negative (proximal RCA 100)</td>
<td>(-)</td>
<td>alive (31 M)</td>
</tr>
</tbody>
</table>

EF = ejection fraction, EPS = electrophysiologic study, LAD = left anterior descending coronary artery, CX = left circumflex coronary artery, RCA = right coronary artery, Vf = ventricular fibrillation.

**Clinical characteristics of the patients**

Clinical features are summarized in Table I and II. Structural heart disease was recognized in primary (group I) of 21 patients (old myocardial infarction in 3, WPW syndrome in 2, mitral stenosis, mitral insufficiency in 2, and dilated cardiomyopathy in 1, respectively). The other patients were followed up every 1-6 months. Sudden death was defined as death within one hour after collapse.

**RESULTS**

**Follow-up**

After hospital discharge, all patients were followed up every 1-6 months. Sudden death was defined as death within one hour after collapse.

**Upright tilt testing**

The tilt table consisted of an electrically-motorized bed with a foot board support. A cannula was inserted into the radial artery to record heart rate and pressure. Blood pressure was continuously monitored and recorded. After 10 minutes of rest in the supine position, patients were tilted head-up to 60 degrees for 60 minutes. A positive tilting test was defined as a development of syncope; hypotension in association with bradycardia; hypotension in association with a fall in heart rate and pressure; or syncope. The spasm provocation test was positive if focal vasoconstriction \( \geq 75\% \) in the other coronary artery when coronary spasm was not induced in one coronary artery. The same procedure was performed in the other coronary artery. At the same time, the provocation test was negative. Caution was then administered to such patients in a suitable dose to prevent coronary vasospasm.

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other 8 patients (group II) had no detectable structural heart diseases, nor primary ECG abnormalities except for mild coronary atherosclerosis (Table II).

In group I, ECG was documented during cardiac arrest in all but one patient. Ventricular fibrillation was documented in 5 patients, and VT in 4 patients. Atrial fibrillation with rapid ventricular response, Torsade de Pointes, and cardiac standstill were each found in one patient, respectively (Table I). On the contrary, in group II, only 4 patients (50%) had documented ECG during cardiac arrest. Ventricular fibrillation was seen in 2 and cardiac standstill in 2 patients (Table II). Drug free ambulatory ECG monitoring was performed in 6 of group II, but only occasional premature ventricular depolarization was recorded.

Symptoms prior to cardiac arrest included chest pain, syncope or palpitation in 12 patients in group I, and in 5 patients in group II (Table I, II). Seven patients in group I had an ejection fraction of 50% or lower, but all group II patients had normal ejection fraction (Table I, II).

Results of EPS
EPS was performed in 12 patients of group I, and in 5 of group II. All 5 patients in group II had negative findings on EPS. In group I, 9 patients with structural heart disease had positive results of EPS. Sustained VT was induced in six, and nonsustained VT in one, and His-ventricular conduction disturbance was found in 2 (Table I, II). The other 3 patients had only primary ECG abnormality. Two patients had WPW syndrome and inducible atrial fibrillation. Their effective refractory period for antegrade conduction was shorter than 250 msec. One patient had long QT syndrome and Torsade de Pointes was induced on exercise, but he had no abnormal findings in EPS. Polymorphic VT was not inducible using our protocol in all subjects who underwent EPS, including the patient with long QT syndrome.

Results of provocation test of coronary spasm
A provocation test of coronary spasm was performed in two patients of group I and six of group II. All of these patients showed normal findings in the other examinations. In group I, coronary spasm was induced in one patient. In group II, coronary spasm was induced in four of six patients (Table I, II). All five patients with positive results of the spasm provocation test had both chest pain and ST change on ECG at the time of induction of spasm in a major coronary artery. In one patient of group II (case 4 in Table II), cardiac standstill which had progressed from complete atroventricular block was also documented on ECG at the time of induction of spasm in the proximal right coronary artery. Coronary spasm was relieved by intracoronary infusion of isosorbide dinitrate and none of the 5 patients had any complications associated with the spasm provocation test.

Results of head-up tilt test
The head-up tilt test was performed in 2 patients of group II (case 5 and 6 in Table II). Of these, the results of EPS and/or spasm provocation test were negative, but bradycardia-hypotension with syncope was induced. They recovered consciousness, and blood pressure and heart rate returned to the control state soon after relief of tilting. They also underwent a serial tilting test to investigate drug efficacy, and syncope was prevented by administration of an alpha-sympathetic agent and β-blocker in each
patient.

Clinical outcomes

Using this systematic approach, the cause of cardiac arrest was established in 19 patients (13 in group I and 6 in group II). Among nine patients with structural heart disease, 7 had inducible VT, and the efficacy of antiarrhythmic agents was evaluated by serial EPS. Four patients were treated with effective drugs guided by EPS. Case 2 was treated with procainamide (2000 mg/day), Case 7 with flecainide (300 mg/day), Case 8 with amiodarone (200 mg/day), and Case 9 with verapamil (320 mg/day) (Table I). All these patients had a favorable outcome during the long-term follow-up period (Fig I). Of the other 3 patients without EPS-guided drug therapy, one patient was successfully treated by electrical catheter ablation and his course was uneventful during follow-up period of 6 months. One patient died suddenly during the follow-up period, and one patient died after an operation because of complications due to infection (Fig. 1). Two patients with His-ventricular conduction disturbance were implanted with a permanent pacemaker but the one with cardiac amyloidosis died suddenly 2 months later (Fig. 1). Patients with WPW syndrome underwent either successful catheter ablation, or surgical division of the accessory pathway. All patients with successful therapy had no recurrence of cardiac arrest. Five patients with inducible coronary spasm were treated with Ca-antagonist and had no recurrence of sudden cardiac arrest. Two patients with vasovagal syncope were treated with β-blocker or alpha-sympathetic agent and had no recurrence (Table II). In the 2 patients with ventricular fibrillation (with negative results of EPS and the spasm provocation test), 1 patient had recurrence of cardiac arrest within 1 month after EPS (Table II).

DISCUSSION

Role of EPS in survivors of cardiac arrest

Survivors of cardiac arrest who are discharged without EPS-guided treatment, or with empiric antiarrhythmic therapy are known to have a high risk of recurrence and sudden death. The role of EPS in patients with cardiac arrest has been well demonstrated in Western countries, and abnormal EPS findings are found in 57% to 76% of survivors of cardiac arrest. Such findings have been confirmed to be unrelated to acute myocardial infarction. The most common inducible arrhythmia is VT and the majority of such patients have old myocardial infarction.

In the present study, structural heart disease was found in 10 of 21 patients and primary ECG abnormality such as WPW syndrome, or long QT syndrome was found in three patients. In 10 patients with structural heart disease, VT was induced in seven, and His-ventricular conduction disturbance was found in two patients. These results were similar to previous studies. EPS should be performed if survivors of cardiac arrest have structural heart disease, but the role of EPS in patients without apparent heart disease seems to be limited.

Role of spasm provocation test without apparent heart disease

In 2% to 12% of patients who experience cardiac arrest, no apparent heart disease can be identified. However, in the present study, eight (38%) of 21 patients had no apparent heart disease, and of these, 5 patients who underwent EPS had normal EPS findings. On the contrary, 4 patients without apparent heart disease had inducible coronary spasm. Coronary spasm, is suggested to be a cause of cardiac arrest, because serious ventricular tachyarrhythmias or bradyarrhythmias often complicate attacks of coronary vasospasm. However, the incidence of coronary spasm in survivors of cardiac arrest has been reported to be only 1% to 3%. In the present study, coronary spasm was induced in five (25%) of 21 patients. The prevalence of coronary spasm, as the cause of sudden cardiac arrest is considerably higher in the present study than in previous reports from Western countries. This may be explained by the difference in the prevalence of coronary heart disease between Japan and Western countries. Coronary heart disease is more frequent in Western countries than in Japan, but vasospastic angina seems to be more frequent in our country.

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Role of tilting test

When fatal arrhythmias and coronary spasm were excluded as the cause of unexplained syncope, vasovagal reaction may be involved\(^{21,22}\) although vasovagal syncope is generally thought to be a self-limited benign disorder. However, cases with long-lasting cardiac standstill have also been reported and cardiopulmonary resuscitation was required\(^{32-34}\) In the present study, bradycardia-hypotension was induced by the head-up tilt test in 2 patients who received cardiopulmonary resuscitation.

Idiopathic ventricular fibrillation

In patients with ventricular fibrillation, the role of EPS was limited when patients have no apparent heart disease. Viskin and Belhassen\(^{26}\) reported 54 patients with "idiopathic" ventricular fibrillation described in the literature between 1948 and 1989. The mechanism of "idiopathic" ventricular fibrillation has not yet been known, and only some potential factors such as mental stress, latent myocarditis, latent preexcitation or coronary spasm were listed\(^{26}\) A rational and effective procedure needs to be established in order to diagnose such patients.

Follow-up of patients with aborted sudden-cardiac death

Previous studies have shown that patients on empiric antiarrhythmic therapy are still at high risk for recurrence and sudden death\(^{23,24}\) and it is essential to choose therapy according to the results of EPS\(^{35,36}\) In the present study, 7 patients with inducible VT underwent serial EPS to assess the efficacy of antiarrhythmic drugs. Four patients who underwent EPS-guided drug therapy had a favorable outcome. On the contrary, 1 patient without an effective therapy died suddenly.

When coronary spasm is provoked in patients with aborted sudden cardiac death, the cause of cardiac arrest may be due to spasm. Spasm can be effectively treated by drugs, with a favorable outcome for the patients. One patient with "idiopathic" ventricular fibrillation had a recurrence of cardiac arrest during the follow-up period.

Limitation of the study

Because we did not evaluate all patients with both EPS and spasm provocation test, some patients with coronary spasm might have positive EPS results, or some patients with positive EPS results might also have positive results of spasm provocation test. However, in previous studies, survivors of cardiac arrest have never had positive results in both tests\(^{35,37}\) The present study suggests that coronary spasm seems to be a more frequent cause of cardiac arrest in survivors of cardiac arrest especially without apparent heart disease, and that in such instance, the spasm provocation might be the first step.

In conclusion, 1) EPS, provocation test of coronary spasm, and the tilting test are useful for evaluation of the cause of cardiac arrest and for selection of effective therapies in survivors of cardiac arrest. Clinical outcome of survivors of cardiac arrest is favorable if effective therapies are chosen after such studies, but unfavorable without effective therapies. 2) Coronary vasospasm seems to be one of the most important causes of cardiac arrest especially in patients without apparent heart disease: the spasm provocation test should be performed in these patients. 3) The role of EPS or spasm provocation test might be limited in "idiopathic" ventricular fibrillation. Further studies are necessary from the standpoints of epidemiologic, clinical, and electrophysiologic characteristics, as well as the clinical course and treatment in idiopathic ventricular fibrillation.

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