Pulmonary Embolism as Cause of Cardiac Arrest

Presentation and Outcome

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Background: Pulmonary embolism (PE) is a possible noncardiac cause of cardiac arrest. Mortality is very high, and often diagnosis is established only by autopsy.

Methods: In a retrospective study, we analyzed clinical presentation, diagnosis, therapy, and outcome of patients with cardiac arrest after PE admitted to the emergency department of an urban tertiary care hospital.

Results: Within 8 years, PE was found as the cause in 60 (4.8%) of 1246 cardiac arrest victims. The initial rhythm diagnosis was pulseless electrical activity in 38 (63%), asystole in 19 (32%), and ventricular fibrillation in 3 (5%) of the patients. Pronounced metabolic acidosis (median pH, 6.95, and lactate level, 16 mmol/L) was found in most patients. In 18 patients (30%), the diagnosis of PE was established only postmortem. In 42 (70%), the diagnosis was confirmed by echocardiography. In 21 patients, 100 mg of recombinant tissue-type plasminogen activator was administered as thrombolytic treatment, and 2 (10%) of these patients survived to hospital discharge. Comparison of patients of the thrombolysis group (n = 21) with those of the nonthrombolysis group (n = 21) showed a significantly higher rate of return of spontaneous circulation (81% vs 43%) in the thrombolysis group (P = .03).

Conclusions: Mortality related to cardiac arrest caused by PE is high. Echocardiography is supportive in determining PE as the cause of cardiac arrest. In view of the poor prognosis, thrombolysis should be attempted to achieve return of spontaneous circulation and probably better outcome.

Arch Intern Med. 2000;160:1529-1535

PULMONARY embolism (PE) is a possible noncardiac cause of cardiac arrest and has an extremely unfavorable prognosis. In fulminant PE, up to 90% of cardiac arrests occur within 1 to 2 hours after the onset of symptoms. Therefore, most of these patients will require cardiopulmonary resuscitation (CPR) before any attempt to diagnose the cause of cardiac arrest has been made, and in many cases the diagnosis will be established only by autopsy after unsuccessful CPR.

The mechanism of cardiac arrest caused by PE is based on pulmonary mainstream obstruction and liberation of vasoconstrictive mediators from the thrombi, leading to increased right ventricular afterload. As the right ventricle fails, right atrial pressure rises and cardiogenic shock ensues. Overload of the right ventricle results in a leftward shift of the ventricular septum, leading to decreased left ventricular diastolic filling and end-diastolic volume. Circulatory failure occurs through a profound decrease in left ventricular preload.

Therapies such as thrombolysis or surgical embolectomy are presumed to reduce mortality, and both methods have been performed even in ongoing CPR situations, either alone or in combination.

In a retrospective study, we analyzed the data of all patients with cardiac arrest caused by PE admitted to our emergency department, including clinical characteristics, diagnostic methods, therapeutic management, and outcome.

RESULTS

During an 8-year period, 1340 patients were admitted to the University Clinic of Emergency Medicine of the University of Vienna, Medical School after in-hospital or out-of-hospital cardiac arrest. Pulmonary embolism was identified as the immediate cause of cardiac arrest in 60 (4.5%) of these cases; 33 (55%) of them had suffered out-of-hospital cardiac arrest. On admission to our unit, 52 patients (87%) were already orotracheally intubated and undergoing mechanical ventilation, and 6 patients (10%) had to be intubated in our de-
PATIENTS AND METHODS

From July 1, 1991, to June 30, 1999, data for all patients admitted to the University Clinic of Emergency Medicine of the University of Vienna, Medical School, Vienna, Austria, after either in-hospital or out-of-hospital cardiac arrest were documented according to a specific protocol (Utstein style).16 The study procedures followed were in accord with the ethical standards of the responsible committee on human experimentation and with the Helsinki Declaration of 1975, as revised in 1983.

According to the criteria of the American Heart Association, cardiac arrest was defined as sudden collapse, followed by loss of consciousness and absence of both spontaneous respiration and pulse, that required CPR.17,18 The following data concerning cardiac arrest and CPR were documented on arrival: sex and age of the patients; location of cardiac arrest (out of hospital vs in hospital); initial electrocardiogram (ECG) rhythm observed by any rescue worker, distinguishing between ventricular fibrillation, asystole, or pulseless electrical activity/electromechanical dissociation (PEA/EMD); and time until restoration of spontaneous circulation. For the interval from cardiac arrest to the beginning of basic and/or advanced life support, we presumed sufficient systemic blood flow to be absent (“no-flow time”). We considered no-flow time to be zero if the cardiac arrest occurred in the presence of medical personnel. The interval from the beginning of life support until return of spontaneous circulation we presumed to represent reduced systemic blood flow (“low-flow time”). Therapy for cardiac arrest included basic and advanced cardiac life support performed by the Vienna Ambulance Service and in-hospital emergency medical personnel in accord with international guidelines.17,18

On the patient’s admission to the emergency department, a physician stated the presumed cause of cardiac arrest on the basis of patient history, including previous physician reports, bystander information, preclinical run sheets, and ECG. Definitive causes of cardiac arrest in nonsurvivors were determined by autopsy. A tentative diagnosis of PE was based on history, symptoms, and ECG before cardiac arrest or after return of spontaneous circulation. The diagnosis of PE was verified in all cases by transthoracic echocardiography, transesophageal echocardiography, spiral computed tomography, ventilation-perfusion scan, and/or autopsy.

All patients received standard intensive care unit treatment with mechanical ventilation, sedation and analgesia. Two patients did not require mechanical ventilation. Characteristics of all patients (sex, age, ECG rhythm during circulatory arrest, laboratory measures, and estimated time of return of spontaneous circulation) are presented in Table 1.

In 42 (70%) of 60 patients the diagnosis was made while the patient was alive, while in the remaining 18 patients PE had clinically not been suspected and was found only at autopsy. In these 18 cases, the physicians stated the reason for the cardiac arrest to be of cardiac origin in 8 patients and undetermined in 10. In the 42 patients, the diagnosis of PE was already suspected clinically. Echocardiographic capability was available in 24 patients (57%), either transthoracic echocardiography (n=6) or transthoracic echocardiography (n=18). In 12 of those patients, emboli were present in proximal pulmonary arteries, while in other patients, indirect signs highly suggestive for PE, such as right ventricular dilatation, interventricular septal bulging, and tricuspid regurgitation, were detected. In another 5 patients the diagnosis was established by spiral computed tomography, and by ventilation-perfusion lung scan in 1 patient, either before cardiac arrest had occurred or following stabilization after successful resuscitation. In 12 patients, the clinical diagnosis of PE was tentative but was later confirmed by autopsy.

The leading symptoms before cardiac arrest are listed in Table 2. We found that there were 2 distinct groups...
Table 1. Factors at Time of Resuscitation*

<table>
<thead>
<tr>
<th>Factor</th>
<th>Finding (N = 60)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex, No. (%) F</td>
<td>37 (62)</td>
</tr>
<tr>
<td>Age, y</td>
<td>62 (50-73)</td>
</tr>
<tr>
<td>Out-of-hospital cardiac arrest, No. (%)</td>
<td>33 (55)</td>
</tr>
<tr>
<td>Witnessed cardiac arrest, No. (%)</td>
<td>58 (97)</td>
</tr>
<tr>
<td>ECG rhythm, No. (%)†</td>
<td>38 (63)</td>
</tr>
<tr>
<td>PEA/EMD</td>
<td>19 (32)</td>
</tr>
<tr>
<td>Asystole</td>
<td>3 (5)</td>
</tr>
<tr>
<td>Ventricular fibrillation</td>
<td>3 (5)</td>
</tr>
<tr>
<td>Lactate level, mmol/L</td>
<td>16 (11-20)</td>
</tr>
<tr>
<td>pH</td>
<td>6.95 (6.86-7.06)</td>
</tr>
<tr>
<td>ROSC, No. (%)</td>
<td>34 (57)</td>
</tr>
<tr>
<td>ROSC, min</td>
<td>14 (7-27)</td>
</tr>
<tr>
<td>Survivors, No. (%)</td>
<td>3 (5)</td>
</tr>
</tbody>
</table>

* Values are expressed as median (range) except as indicated otherwise. ECG indicates electrocardiogram; PEA/EMD, pulseless electrical activity/electromechanical dissociation; and ROSC, return of spontaneous circulation.
†First recorded during cardiac arrest.

of patients: 33 patients (55%) had lost consciousness suddenly, either as the initial symptom itself without any prodromal signs or almost immediately after onset of symptoms, whereas 27 patients (45%) developed cardiac arrest after sustained acute cardiogenic shock associated with pronounced bradycardia and hypotension.

In 40 patients an ECG had been obtained either before cardiac arrest or immediately after return of spontaneous circulation, which showed a right bundle-branch block in 28 patients (70%), S1Q3T3 pattern in 3 (8%), T-wave inversion in V1 to V3 in 2 (5%), and complete atrioventricular block in 1 (2%). In 6 cases (15%), the ECG showed unspecific changes.

On admission, blood was drawn from an arterial line in 51 patients (85%) immediately after arterial access had been achieved. Almost all patients had a pronounced metabolic acidosis, and median of serum lactate levels as well as of blood pH are shown in Table 1.

In 52 (87%) of all patients, central pulmonary emboli could be detected; in 22 of them emboli were detected in segmental regions as well, while in 8 patients (13%) emboli were found only in segmental regions. Deep vein thrombosis was found ante mortem or postmortem in 51 (85%) of 60 patients, but only 3 patients showed clinical signs of deep vein thrombosis.

We grouped the patients in 2 subcategories according to whether thrombolytic treatment had been administered. Of the 42 patients correctly diagnosed as having PE, 21 (50%) received thrombolytic therapy. The arrest was witnessed in all these patients. Initial rhythm, location of cardiac arrest, times of no and low flow, and relevant laboratory results and outcome of these patients are listed in Table 3. The diagnosis of PE was established or strongly suspected in 16 of these 21 patients by bedside echocardiography, in 2 patients by spiral computed tomography, and clinically in another 3 patients. Systemic thrombolytic treatment was instituted by the peripheral intravenous application of alteplase either during ongoing CPR (n=11) or shortly before (n=4) or after (n=6) cardiac arrest. In 10 patients the thrombolytic agent was given within 15 minutes as a 50-mg double bolus. Alternatively, 11 patients received a bolus dose of 15 mg, followed by continuous infusion of 85 mg over 90 minutes. Return of spontaneous circulation was achieved in 17 patients (81%). Of those, 2 patients survived to hospital discharge. The medical course of these 2 surviving patients was as follows.

Patient 17, a 67-year-old man, was admitted while he had already been undergoing CPR for 5 minutes and had received 2 mg of epinephrine and a bolus of 0.48 g of theophylline. His arterial blood pressure was 50/30 mm Hg, with a heart rate of 120 beats/min. The ECG showed a complete right bundle-branch block, and transthoracic echocardiography showed dilatation of the right ventricle. A bolus of 50 mg of alteplase was injected, followed by a second bolus of 50 mg after 15 minutes. Within 5 minutes the patient's condition stabilized and completely recovered, so that he was discharged from the hospital 48 days after cardiac arrest without neurological damage.

Patient 20, a 45-year-old man, had suffered a cardiac arrest shortly before arrival, and CPR had been performed for 3 minutes and was still going on when he was admitted. Transthoracic echocardiography showed a dilated right ventricle. Systemic thrombolysis with a 50-mg double bolus within 15 minutes achieved hemodynamic stabilisation. As the patient's hemodynamic condition deteriorated again, spiral computed tomography was performed and showed a large embolus in the main pulmonary artery. The patient underwent embolectomy and recovered without neurological damage, to be discharged from the hospital after 21 days.

In patient 1, systemic thrombolytic therapy failed and cardiopulmonary bypass was used to bridge to emergency surgery, but despite surgical embolectomy with a catheter device, no permanent spontaneous circulation could be achieved.

In 5 patients, bleeding complications were observed. Patient 13 developed a perirenal hematoma that was detected postmortem. In the other 4 patients, blood loss was detected by either abdominal sonography or computed tomography and required replacement. In patient 21 (rupture of liver) emergency abdominal surgery was performed. In patients 18 (rupture of liver) and 19 (mediastinal bleeding) emergency surgery was considered, but in both patients the bleeding stopped spontaneously. No patient died as a result of hemorrhage.

In 21 patients in whom PE had been diagnosed, no thrombolytic treatment was performed. The arrest was
witnessed in all these patients. Initial rhythm, location of cardiac arrest, times of no and low flow, and relevant laboratory results and outcome of these patients are listed in Table 4. In 1 patient, thrombolytic therapy was withheld because of verified hematothorax after chest compressions during external cardiac massage, and he was discharged after 48 days. The remaining 20 patients were not considered for thrombolytic therapy because of known underlying severe morbidity and expected neurological damage after prolonged resuscitation efforts. Although return of spontaneous circulation was achieved in 7 of these patients, all 20 patients died of multiple-organ failure, their underlying malignant disease, or severe cerebral damage.

When we compared patients in whom thrombolysis had been administered (n = 21) with patients in whom this treatment had been withheld (n = 21) regarding various measures, we did not observe a statistically significant difference.
cant difference between the 2 groups except for the occurrence of return of spontaneous circulation, which was significantly higher in the thrombolysis group (Table 5) (P = .03).

### COMMENT

Our study supports the experience that PE as one possible cause of cardiac arrest has an exceedingly poor prognosis. Diagnosis of PE in cases of cardiac arrest is often difficult to establish. Although more than half of our patients were admitted after a prehospital cardiac arrest, we established PE as the cause of cardiac arrest in two thirds of the cases. Systemic thrombolysis and surgical embolectomy are useful, but in our study, despite these interventions, overall mortality remained high.

Previous studies have reported on the difficulties involved in diagnosing PE. Hauch et al demonstrated that fatal PE was an unexpected autopsy finding in 10 of 16 postoperative cases. In another study, PE was responsible for 8 (10%) of 80 cardiac arrests where a cardiac origin had been primarily suspected. Bedell and Fulton showed that the 2 diseases most frequently undetected clinically were ischemic bowel disease and PE. In another autopsy study, Karwinski and Svendsen reported that the clinical diagnosis of PE as the immediate cause of death was missed in 84% of all cases of patients with death of unknown reason.

We believe that clinical suspicion of PE as a cause of cardiac arrest remains the key in timely diagnosis and treatment. In our study, the most suggestive reported symptoms of PE were sudden dyspnea and syncope. Therefore, these symptoms should increase and reinforce the clinical suspicion of acute PE, especially in the presence of predisposing conditions. This is different from a cardiac cause such as myocardial infarction or dissecting thoracic aortic aneurysm, where the leading symptom is chest pain, which was reported in only a quarter of our patients. Deep vein thrombosis is known to be an important risk factor for PE, but clinical signs of deep vein thrombosis are rare and nonspecific. While leg pain or swelling were reported before cardiac arrest in only a few cases, we have detected deep vein thrombosis in 85% of our patients at autopsy. This suggests that lack of signs of deep vein thrombosis is not as helpful in acute decision making as previously believed.

Care must be taken in analyzing ECGs before cardiac arrest or after successful CPR in patients with PE. We found that right bundle-branch block was present in 67% of these cases, and this should induce a high suspicion for massive PE as cause of cardiac arrest. In addition, it is known that, while ventricular fibrillation is the most common rhythm in cardiac arrest of cardiac origin, PE is significantly associated with PEA/EMD or asystole, and both of these rhythms are notoriously associated with poor outcome. We found PEA/EMD or asystole in about 95% of our patients initially. Another probable predictor of poor outcome is the level of metabolic disturbance at admission. We found that a pronounced metabolic acidosis was present in almost all of the patients.

For in vivo diagnosis of PE, several standard diagnostic approaches routinely used in uncompromised patients are available. Spiral computed tomography and ventilation-perfusion lung scan are noninvasive, and in massive PE they are both highly sensitive and specific. However, these methods have serious limitations, as they are time consuming and require the patient to be transported to the radiology or nuclear medicine department, which is almost impossible in acute resuscitation situations. In our group of patients, spiral computed tomography was used for initially establishing PE as the diagnosis in 5 patients, and ventilation-perfusion scan was used in 1. Pulmonary artery angiography is invasive, expensive, time and personnel consuming, and not available at all hospitals. Therefore, for clinical decision making in patients with cardiac arrest caused by PE, it is no more helpful than spiral computed tomography.

As bedside diagnostic methods, both transthoracic and transesophageal echocardiography are readily available. Especially in critically ill patients in whom other diagnostic techniques are high risk or technically difficult, echocardiography is feasible, low risk, and inexpensive. Therefore, it is the method of choice for substantiating the diagnosis of PE in patients during ongoing CPR. Findings of transthoracic echocardiography in patients suffering from PE include dilatation of the right ventricle, paradoxical septal movement (bulging), and tricuspid regurgitation, but because of thorax deformity, lung emphysema, or adiposity, the method is limited. When transesophageal echocardiography is used, findings include location and size of emboli, and this was possible in 12 of 24 patients. Additionally, real-time differential diagnosis of cardiac tamponade caused by rupture of the aortic aneurysm or perforation of a ventricle after myocardial infarction, which can also mimic the symptoms of PE, is possible. The ready availability of an echocardiographic system and trained personnel is the key to efficient patient treatment in such a situation. Varriale and Maldonado reported that the average time from initiation of CPR to a suitable echocardiographic image of the

| Table 5. Comparison of Thrombolysis and Nonthrombolysis Groups for Common Determinators* |
|---------------------------------|---------------------------------|---------------------------------|---------------------------------|---------------------------------|---------------------------------|---------------------------------|---------------------------------|
|                                | Thrombolysis Group (n = 21)     | Nonthrombolysis Group (n = 21)  | P                               |
|                                | Sex, No. (%)                   | Age, y                         | Out-of-hospital cardiac arrest, No. (%) | Witnessed cardiac arrest, No. (%) | ECG rhythm, No. (%) | Lactate level, mmol/L | pH | ROSC, No. (%) | Survivors, No. (%) |
|                                | F (13) (62)                    | 60 (43-73)                     | 12 (57)                         | 21 (100)                        | 15 (71)              | 18 (11-22)             | 6.90 (6.85-6.98) | 17 (81) | 2 (10) |
|                                | (14) (67)                      | (66-52-73)                     | (8 (38)                         | (21 (100)                       | (15 (71)             | (14 (9-17)             | (7.00 (6.88-7.09) | (9 (43) | (1 (5) |
|                                | .75                            | .28                            | .35                             | .99                             | .58                 | .06                   | .14                      | .03             | .99                   |
|                                |                                |                                |                                 |                                 |                     |                       |                           |                  |                      |

* Values are expressed as median (range) except as indicated otherwise. See the first footnote to Table 1 for explanation of abbreviations.
heart was 3.9 minutes (range, 17 seconds to 10 minutes), and this allows for timely therapeutic intervention. In our group of patients, transthoracic echocardiography detected right ventricular loading in 12 patients and visualized an embolus in another 12 patients.

Since the 1990s, some clinical studies have retrospectively attempted to evaluate different therapeutic strategies in patients with cardiac arrest caused by PE to determine whether early institution of specific therapy can reduce mortality. However, selection bias cannot be excluded, as anecdotal reports about unsuccessful thrombolytic treatment during CPR may not have been published as often as successful interventions. Scholz et al reported that 7 of 17 patients survived after thrombolytic treatment, a mortality rate of 59%. Böttiger et al cited mainly case reports with successful thrombolytic treatment for in-hospital resuscitations. Hopf et al described survival of 5 of 6 postoperative patients after systemic thrombolysis with rt-PA. We also noted that there is no consensus on drug and dosage of thrombolytic treatment, as in the same study patients have been treated with urokinase, streptokinase, and rt-PA in individual dosage regimens.

Thrombolytic treatment with rt-PA was administered in 21 patients. Although in 17 of them spontaneous circulation returned, only 2 survived, resulting in a mortality rate of 90%. Of the 21 patients who received thrombolytic treatment, 8 were admitted under ongoing CPR. The surviving patients belonging to this group both had central PE and were admitted after out-of-hospital cardiac arrest, after a short time of basic life support. That these patients survived without neurological damage is probably because of the short no-flow time and the immediate application of thrombolytic therapy. In the nonthrombolysis group, only 1 patient survived after segmental PE. Although mean age was not significantly different in both groups, the patients in the nonthrombolysis group were older (mean age, 66 years vs 60 years), which indicates that comorbidity may have been higher and possibly also explains why return of spontaneous circulation occurred less often.

Bleeding, in particular cerebral bleeding with fatal or disabling outcome, represents the main hazard of thrombolytic therapy. The most severe complications encountered in our emergency department were rupture of the liver in 2 cases and mediastinal bleeding in 1 patient. These were almost certainly caused by the resuscitation efforts but were aggravated by thrombolysis. Laboratory studies and ultrasound examinations should be performed in short intervals to recognize bleeding complications early. Our data may suggest that the application of a higher rt-PA bolus dose is associated with better outcome. In the literature, however, there is no randomized clinical trial that substantiates this assumption. The recommendations of the fifth American College of Chest Physicians Consensus Conference are to administer either 100 mg of alteplase over 2 hours or a bolus of 250,000 U of streptokinase followed by 100,000 U/h over 24 hours. The Continuous Infusion vs Double Bolus Administration of Alteplase (COBALT) study failed to show any superiority of the application of a double bolus of rt-PA compared with accelerated administration of rt-PA over 90 minutes in coronary thrombolysis; in addition, it is possible that this may be associated with a higher incidence of intracranial hemorrhage.

Surgical embolectomy for the treatment of cardiac arrest caused by PE is useful as an alternative method, especially after failure of thrombolysis or when this is contraindicated, eg, in case of previous major trauma and/or recent surgery. Some authors report that surgical embolectomy in patients having an episode of cardiac arrest after PE is associated with mortality rates of between 50% and 74% (Table 6). In our study, in only 1 patient was embolectomy performed because of hemodynamic instability. In 1 case we used extracorporeal membrane oxygenation for bridging to emergency surgery after unsuccessful thrombolysis. Although embolectomy was performed, the patient died. Despite this unfavorable outcome, we suggest that, in the case of cardiac arrest caused by PE, this method may be beneficial as a last-resort procedure in some patients where no spontaneous circulation can be achieved by conventional resuscitation efforts and systemic thrombolysis.

Catheter thrombectomy is an alternative method of managing PE, but none of our patients underwent this intervention.

Comparison of our results with the outcome data of previous studies is difficult, as important information such as location of cardiac arrest or long-term results such as hospital discharge and survival at 6 months are not presented in some of these retrospective analyses. More than half of our patients were initially resuscitated successfully, but most of them died early during the hospital stay, either in intractable shock or from severe cerebral damage. In addition, other authors reported on only selected patients, while we present data for all patients suffering cardiac arrest after PE. Moreover, some authors made their observations during a period of 9 to 25 years, a time span that makes a sound scientific view difficult (Table 6).

CONCLUSIONS

Despite systemic thrombolysis, the mortality related to cardiac arrest caused by PE is extremely high. Because of its bedside diagnostic capabilities and lack of interference with resuscitation efforts, echocardiography should be performed in all cases to establish the diagnosis of PE as the cause of a cardiac arrest. Because thrombolytic agents are easily and rapidly available in almost all hospitals, thrombolysis as the last-resort treatment to

Table 6. Pulmonary Embolectomy After Cardiopulmonary Resuscitation

<table>
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<tr>
<th>Source</th>
<th>Study Duration, y</th>
<th>No. of Patients</th>
<th>No. of Survivors</th>
<th>Mortality Rate, %</th>
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<tr>
<td>Meyer et al16</td>
<td>20</td>
<td>24</td>
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<td>Doerge et al11</td>
<td>16</td>
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<td>Schmid et al22</td>
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<td>50</td>
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<tr>
<td>Gray et al13</td>
<td>23</td>
<td>25</td>
<td>9</td>
<td>64</td>
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<tr>
<td>Clarke and Abrams14</td>
<td>25</td>
<td>19</td>
<td>5</td>
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<td>9</td>
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<td>9</td>
<td>19</td>
<td>7</td>
<td>63</td>
</tr>
</tbody>
</table>
achieve return of spontaneous circulation in cases where no obvious contraindications exist and good neurological outcome is expected should be further evaluated.

Accepted for publication November 4, 1999.

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REFERENCES