To the Editor:

Thank you for allowing me to respond to the letter by Ruiz-Santana and colleagues. His patient had pulmonary hypertension, which is one of the recognized risk factors for pulmonary artery rupture. It would be enlightening to know if the clinical event, hemoptysis and shock, developed while the catheter was being manipulated. The outcome in his patient is consistent with our findings that patients who experience free intrathoracic rupture with a hemotorax, which is shown in their fine illustrations, do not survive without surgery. As in this case, all of our patients who had a chest x-ray film taken demonstrated an abnormality.

Two of our patients had autopsies that revealed free perforations of the pulmonary artery with surrounding hematoma. A third patient had an immediate thoracotomy, which revealed liquid blood in the right chest and a perforation in the lung parenchyma of the lower lobe. She underwent lobectomy but succumbed several days later from cardiopulmonary failure.

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Outcomes of In-Hospital Cardiopulmonary Resuscitation

To the Editor:

It was interesting to read the article “Postdischarge Survival and Functional Status Following In-Hospital Cardiopulmonary Resuscitation” by Robinson and Hess (CHEST 1994; 105:991-96).

In this context, it may be of interest to recall some data from Buffalo General Hospital, New York, presented by me at the American College of Physicians’ Annual Session in 1974.1

Between July 1967 and January 1972, there were 1,269 patients with a definite diagnosis of acute myocardial infarction (AMI) admitted to the Coronary Care Unit of Buffalo General Hospital. Of these, 126 patients had 1 or more episodes of ventricular fibrillation (VF). Eighty-four patients did not survive to leave the hospital as a result of cardiogenic shock, progressive heart failure, extension of infarct, or some other terminal condition. The remaining 42 patients, who left the hospital, comprised the study group. These patients were followed up by their private physicians, who were kind enough to complete follow-up information for us. The follow-up period was from 9 months to 64 months (mean 34 months). There were 33 men and 9 women with mean ages 59.2 and 62.1 years, respectively. Twenty-six patients had primary VF and 16 secondary. Twenty-seven patients developed VF within the first 24 h. Relevant history included 50% with previous angina, 26% myocardial infarction, 29% hypertension, 17% diabetes, and 10% peripheral vascular disease (PVD).

During follow-up, 11 patients died, 5 as a result of further AMI; 4 patients died suddenly and unexpectedly, without preceding symptoms, presumably from malignant arrhythmia. Yearly survival was 88% for the first year, 83% for the second year, 84% for the third year, 65% for the fourth year, and 62% for the fifth year. Of the surviving patients, 24 were in the New York Heart Association functional class I or II, 6 were in class III or IV, and 1 patient was crippled by PVD.

At the time of reporting, 12 patients (10 men and 2 women) were gainfully employed; only 22 patients were under 65 years at the time of VF. Three men were unfit for work from cardiac disease or PVD; three patients had further AMI, and five had angina. Thirteen patients were off all cardiac drugs; eight patients were on diuretics, nine on digitalis, and six on quinidine or propranolol hydrochloride. Five patients had functionally deteriorated since their original admission.

It was concluded that the long-term prognosis of patients who had VF during AMI was not very different from that of the whole group of survivors of AMI.

Ventricular tachycardia or fibrillation occurred in 18 out of 24 patients (75%) who survived CPR in the study by Robinson and Hess. Their long-term outcome was broadly similar to that of our group. Two decades separated the two studies—a period which has been significant for explosion of revascularization procedures. It would be relevant to study the impact of such procedures on the subset of patients under discussion. This would only be possible from a large-scale study of the survivor population.

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REFERENCE


Erratum

In Table 3 of the ACCP Consensus Statement entitled “Primary Pulmonary Hypertension” (CHEST 1993; 104:245), the dose range for the drug prostaglandin E1 was stated incorrectly. The prostaglandin E1 dosages should have been listed in ng/kg/min instead of pg/kg/min. The correct table is reprinted for your reference.

Table 3—Dose Ranges, Route of Administration, and Half-Lives of Most Frequently Used Vasodilators

<table>
<thead>
<tr>
<th>Drug</th>
<th>Route</th>
<th>Dose Range</th>
<th>Half-Life</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nifedipine*</td>
<td>Oral</td>
<td>30-240 ng/day</td>
<td>2-5 h</td>
</tr>
<tr>
<td>Diltiazem*</td>
<td>Oral</td>
<td>120-900 ng/day</td>
<td>2-4.5 h</td>
</tr>
<tr>
<td>Prostacyclin†</td>
<td>Intravenous</td>
<td>2-24 ng/kg/min</td>
<td>3 min</td>
</tr>
<tr>
<td>Prostaglandin E1</td>
<td>Intravenous</td>
<td>5-30 ng/kg/min</td>
<td>2-4 min</td>
</tr>
</tbody>
</table>

*Sustained release preparations (Procardia XL and Cardizem CD) may be given once daily; half-life shown refers to conventional preparation.
†Dose range listed is for immediate infusions; dose requirements and tolerance in patients receiving long-term infusions have increased over time, often exceeding 50 to 100 ng/kg/min.