sue of permanent pacing in post-radiation atrioventricular (A-V) block. In 1992 our group described in a non-English medical journal a similar case, that I will summarize briefly.

A 54-year-old woman developed complete A-V block with syncope 22 years after radiation therapy of the mediastinal area for Hodgkin’s lymphoma. The most peculiar feature of this case was that a few months after implantation of a single chamber pacemaker (VVI), the patient started to complain of peripheral edema, dyspnea, and fatigue. These symptoms were interpreted as the so-called “pacemaker syndrome.” Interestingly enough, this patient had normal baseline left ventricular function (estimated ejection fraction of 64%), and no evidence of pericardial involvement, while diffuse thickening of both mitral leaflets and discrete thickening of septal tricuspid leaflet were observed during echocardiographic examination. An echo-Doppler study was performed during VVI and temporary sequential A-V pacing (programmed A-V interval of 150 ms). The evaluation of mitral inflow pattern showed that the latter modality of pacing was associated with a 36% increase in the time velocity integral (from 12.6 to 17.2 cm²) and in stroke volume (from 50.4 to 68.8 mL), which is consistent with a marked improvement of cardiac output. These findings suggested that sequential A-V pacing might be of benefit for this patient also in the long term. We were lucky enough to confirm this impression soon after implantation of a physiologic dual chamber device (DDD). For the following 2 years, the patient has been clinically well, without the need for diuretics or other cardiovascular drugs.

The case reported by Knight and Sutton seems to confirm our observation. In fact, the patient described developed progressive right heart failure after VVI pacing, which was reverted when sequential A-V mode was instituted. It is possible that a functional Doppler evaluation during temporary A-V pacing might have allowed the patient to avoid the experience of tricuspid valve surgery, which was, as the authors state, of minimal clinical benefit.

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REFERENCES

Pulmonary Artery Rupture Illustrations

To the Editor:

We have read with great interest the excellent article by Kearney and Shabot in the November 1995 issue (CHEST 1995;108:1349-52) in which the authors reviewed their own experience and 65 cases collected from the literature with Swan-Ganz-associated pulmonary artery rupture. However, they do not show any illustrations.

We wish to report a patient with severe pulmonary hypertension (105/70 mm Hg), secondary to mitral stenosis, which developed into massive hemoptysis and shock due to pulmonary artery rupture after right-side catheterization. A chest radiograph (Fig 1) performed immediately after the rupture demonstrated a right lower lobe infiltrate and a right pleural effusion, which was documented to be a hemathoma. A thoracotomy could not be performed, and, like the seven patients in the study by Kearney and Shabot, our patient did not survive. At autopsy, a ruptured right lower lobe pulmonary artery and associated pulmonary hematoma were found (Fig 2).

Figure 1. Chest radiograph showing a right lower lobe infiltrate and a right pleural effusion.

Figure 2. Autopsy revealed a ruptured right lower lobe pulmonary artery (arrow) and associated pulmonary hematoma.
Outcome 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 1,206 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 procainamide 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 prostanoglandin E₁ is 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>Diltazein*</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.