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).2–3 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 cm/s) 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.

Luigi La Vecchia, MD, FCCP
Department of Cardiology,
University of Verona,
Italy

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 hemothorax. 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.
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 hemothorax, 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 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.

Thomas J. Kearney, MD, Robert Wood Johnson Medical School, Piscataway, New Jersey

Outcome of In-Hospital Cardiopulmonary Resuscitation

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, 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/kg/day</td>
<td>2.5-5 h</td>
</tr>
<tr>
<td>Diltiazem*</td>
<td>Oral</td>
<td>120-900 ng/kg/day</td>
<td>2-4.5 h</td>
</tr>
<tr>
<td>Prostacyclin1</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.

1Dose 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.