clearance of bupivacaine exhibited no significant change throughout the course of long-term infusion.

We conclude that continuous epidural infusion of bupivacaine is a safe and effective method of pain relief in children undergoing major lower extremity and genitourinary operations. It requires an anesthesiologist trained in regional anesthesia in children, repeated examination of the child, dependable infusion pumps and attention to measures which prevent infection.

The authors gratefully acknowledge the help of Roger Bell Laboratories for bupivacaine plasma level measurements and G. Rosine and E. Schmitt for secretarial assistance.

Anesthesiology

Influence of Balloon Inflation and Deflation on Location of Pulmonary Artery Catheter Tip

WILLIAM E. JOHNSTON, M.D.,* ROGER L. ROYSTER, M.D.,* JAKOB VINTEN-JOHANSEN, PH.D.,†
GLEN P. GRAVLEE, M.D.,* GEORGE HOWARD, M.S.P.H.,‡ STEPHEN A. MILLS, M.D.,†
WILLIAM Y. TUCKER, M.D.‡

Catheter perforation of the pulmonary artery represents a serious and sometimes lethal complication of invasive hemodynamic monitoring; it is seen predomin-

* Associate Professor in Anesthesiology.
† Assistant Professor in Surgery (Section on Cardiothoracic Surgery).
‡ Assistant Professor, Center for Prevention Research and Biometry.

Received from the Departments of Anesthesia and Surgery (Section on Cardiothoracic Surgery) and the Center for Prevention Research and Biometry, Wake Forest University Medical Center, Winston-Salem, North Carolina. Accepted for publication January 22, 1987. Presented in part at the 37th Annual Meeting of the American Society of Anesthesiologists, October 18–22, 1986, Las Vegas, Nevada.

Address reprint requests to Dr. Johnston: Department of Anesthesiology, Bowman Gray School of Medicine, 300 South Hawthorne Rd, Winston-Salem, North Carolina 27103.

Key words: Complications: pulmonary artery perforation. Monitoring: pulmonary artery. Surgery: cardiac.

nantly in patients with pulmonary hypertension.1,2 Two initiating mechanisms have been proposed: 1) eccentric balloon inflation, causing impingement of the catheter tip into the vessel wall;3–5 and 2) high intraballooon pressures, causing direct vessel disruption during balloon inflation.6,7 We believe that other forces acting on the catheter tip may contribute to causing this complication.

Several reports have described catheter-induced rupture through the pulmonary artery, with extension into the pleural cavity, causing massive hemothoraces.1,4,8,9 One possible explanation of the hemothoraces is the formation of a dissecting hematoma by the catheter perforating the pulmonary artery, with dissection into the pulmonary parenchyma and extension into the pleural space. Another explanation is direct penetration of the catheter tip through the pulmonary artery and parenchyma into the pleural space.8 In the latter circumstance, the hemodynamic forces causing this excessive

<table>
<thead>
<tr>
<th>Patient</th>
<th>Cm (mean ± SD) mg·ml⁻¹</th>
<th>GL ml·min⁻¹·kg⁻¹</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>.47 ± .07</td>
<td>7.09</td>
</tr>
<tr>
<td>2</td>
<td>.45 ± .12</td>
<td>7.40</td>
</tr>
<tr>
<td>3</td>
<td>.73 ± .07</td>
<td>4.56</td>
</tr>
<tr>
<td>4</td>
<td>.30 ± .11</td>
<td>11.11</td>
</tr>
<tr>
<td>5</td>
<td>.69 ± .14</td>
<td>4.83</td>
</tr>
<tr>
<td>6</td>
<td>.84 ± .29</td>
<td>3.96</td>
</tr>
</tbody>
</table>

REFERENCES


5. Hanson AL, Hanson B: Continuous mini-infusion of bupivacaine into the epidural space during labor: Experience from 1000 deliveries. Reg Anaesth 10:159–144, 1985


amount of catheter tip movement have not been explained.

Patients with pulmonary hypertension appear at risk for catheter-induced pulmonary artery rupture, due, in part, to structural differences in the vessel wall and to a larger pressure gradient across the inflated balloon which may force the tip to perforate the vessel.\textsuperscript{8,10} However, another factor causing the catheter tip to migrate distally may be the size of the catheter balloon.

Although the characteristics of catheter balloon inflation have been described,\textsuperscript{11} the properties of balloon deflation and the relationship between balloon deflation and catheter tip movement, particularly in the presence of pulmonary hypertension, have not been studied. We have observed that balloon deflation differs from inflation, and believe the smallest balloon volume occurs during deflation. Consequently, the most distal catheter migration may occur during deflation, rather than during inflation. In support of this theory, Haapaniemi et al. reported a case where distal tip migration occurred during balloon deflation.\textsuperscript{7} For these reasons, we tested three hypotheses: 1) that distal migration of the catheter tip is greater during balloon deflation than during balloon inflation, 2) that the amount of tip migration during deflation is greater in the presence of elevated mean pulmonary artery pressure (PAP) than when PAP is normal, and 3) that characteristics of balloon deflation differ from those of balloon inflation.

METHODS

To test the first two hypotheses, we studied catheter migration in 14 men and four women scheduled for elective coronary artery bypass surgery. Patients were excluded from the study if emergency surgery was required or if they had any form of cardiac dysrhythmia. Eleven of the 18 patients (group 1) had PAP < 20 mmHg; seven patients (group 2) had pulmonary hypertension (PAP \textgeq 20 mmHg). The study was approved by the Institutional Research Practices Committee and required patient consent, which was obtained.

By the Seldinger technique, we advanced a 7.5 F quadruple-lumen pulmonary artery catheter (American Edwards Laboratories, Santa Ana, CA) from the right internal jugular vein into the pulmonary artery until a pulmonary artery occlusion pressure tracing was obtained. The catheter was positioned so that inflating the balloon with 1.5 ml air, the volume recommended by the manufacturer, caused the catheter to wedge within two cardiac cycles. We noted the catheter length at the point of insertion into the introducer sheath, and secured the end of the catheter to the patient’s forehead to prevent accidental catheter migration. Anesthesia consisted of diazepam, fentanyl, and pancuronium iv.

An intravenous infusion of nitroglycerin at 50 \textmu g/min was started before induction and maintained throughout the study period.

Following induction of anesthesia and endotracheal intubation, we used fluoroscopy to determine the location of the catheter tip and recorded that location relative to a zero point defined as the radiographic intersection of the catheter in the pulmonary artery with the catheter in the superior vena cava (fig. 1).\textsuperscript{12} Catheter tip location to the patient’s right of the zero point was designated positive (forward) migration; to the left as negative (backward) migration. A coin placed on the chest wall served as a reference standard to correct for image magnification.

Under fluoroscopy, after temporarily disconnecting the endotracheal tube from the ventilator, we inflated the catheter balloon with 1.5 ml of air from the syringe, identifying the wedge position by the appearance of a typical pulmonary artery occlusion pressure tracing. We
CLINICAL REPORTS

TABLE 1. Hemodynamic Variables in Group 1 and 2 Patients

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group 1 (n = 11)</th>
<th>Group 2 (n = 7)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean arterial pressure (mmHg)</td>
<td>82 ± 4</td>
<td>85 ± 7</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>65 ± 3</td>
<td>75 ± 7</td>
</tr>
<tr>
<td>Pulmonary artery pressure (mmHg)</td>
<td>16.2 ± 0.7</td>
<td>25.1 ± 2.2*</td>
</tr>
<tr>
<td>Pulmonary artery occlusion</td>
<td>10.2 ± 0.7</td>
<td>16.7 ± 1.4*</td>
</tr>
<tr>
<td>pressure (mmHg)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Central venous pressure (mmHg)</td>
<td>8.5 ± 0.9</td>
<td>11.7 ± 1.3*</td>
</tr>
<tr>
<td>Cardiac output (l/min)</td>
<td>5.7 ± 0.6</td>
<td>5.3 ± 0.7</td>
</tr>
<tr>
<td>Catheter insertion distance (cm)</td>
<td>50.2 ± 0.6</td>
<td>54.7 ± 0.8*</td>
</tr>
</tbody>
</table>

* P < 0.05 compared to group 1.

Ana, CA) to quantitate catheter balloon inflation and deflation characteristics in vitro. We tested each catheter by injecting 1.5 ml of air before the study to insure that inflation was symmetrical by visual inspection. We measured intraballoon pressure with a transducer and intraballoon volume by water displacement in a 37° C water bath. Using the 1.5 ml syringe, we slowly injected 0.1 ml increments into the balloon until it was fully inflated; the balloon was then deflated in 0.1-ml decrements. At each 0.1-ml interval, we waited 20–30 s for the pressure to stabilize and then measured intraballoon volume. The inflation-deflation procedure was performed three times, and the respective values were averaged.

We analyzed the data, expressed as mean ± standard error (SE), by paired t test for intragroup comparisons and the in vitro study, and by t test for independent samples for intergroup comparisons. A P value ≤ 0.05 was considered significant.

RESULTS

All patients were well oxygenated, with P_{aO2's} between 349 and 516 mmHg and P_{aCO2's} between 32 and 43 mmHg. Their temperatures varied from 35.3–36.6° C. PAP averaged 16.2 ± 0.7 mmHg (range 11.3–18.7 mmHg) in group 1 and 25.1 ± 2.2 mmHg (range 20.0–36.0 mmHg) in group 2 (P < 0.01). Pulmonary artery occlusion pressure and CVP were higher in group 2 than in group 1 patients (table 1). Other hemodynamic variables were not significantly different between the groups.

In all patients, the catheter tips lay in the right pulmonary artery. The catheters required a greater insertion distance before a satisfactory wedge position was obtained in group 2 patients than in group 1 patients (P < 0.05, table 1). No patient in either group had evidence of pulmonary artery perforation during the study period.

At baseline, group 1 catheter tips were located 1.0 ± 0.6 cm to the right of the zero point in diastole and 1.9 ± 0.6 cm to the right of the zero point in systole (P < 0.01 comparing systole with diastole, fig. 2). Balloon inflation caused the catheter tips to migrate 1.4 ± 0.3 cm toward the periphery of the lung from systolic baseline values (P < 0.01). With passive balloon deflation, catheter tips migrated an additional 0.4 ± 0.2 cm over a range of 0–1.8 cm (P < 0.05 compared to inflation values). The total excursion distance of the catheter tip in group 1 patients from baseline at diastole through balloon deflation was 2.6 ± 0.5 cm (P < 0.001).

In group 2 patients, catheter tips were located to the patient's right of the zero point by 2.1 ± 0.7 cm during diastole and 3.3 ± 0.6 cm during systole (P < 0.02, fig.

![Fig. 2. Catheter tip locations at the four measurement intervals in group 1 patients with PAP < 20 mmHg and in group 2 patients with PAP ≥ 20 mmHg. All distances are referenced to the zero point (see text).](image-url)
Balloon inflation caused the catheter tips to move minimally to 5.2 ± 0.4 cm, a location very similar to that in group 1 patients (3.3 ± 0.4 cm). With balloon deflation, catheter tips in group 2 patients migrated an additional 1.6 ± 0.3 cm toward the lung periphery over a range of 0.3–2.6 cm (P < 0.01 compared to inflation values). The total excursion distance of the catheter tip from baseline at diastole through balloon deflation was 2.7 ± 0.7 cm (P < 0.01), a distance similar to group 1. However, during balloon deflation alone, the amount of catheter tip migration was greater and the actual tip location was more distal in group 2 than in group 1 patients (P < 0.05, respectively, fig. 2). The proportion of tip migration occurring during balloon deflation relative to the overall excursion distance was 13% in group 1 patients in contrast to 62% in group 2 patients. Catheter tips in both patient groups spontaneously returned to baseline position following each inflation-deflation sequence. Anteroposterior radiographs from a group 2 patient taken at baseline during diastole and during balloon deflation are shown in figure 3. The actual distances of catheter tip migration during inflation and deflation for each patient are shown in figure 4.

In the in vitro studies of six catheters, instantaneous balloon inflation from zero to 0.41 ± 0.1 ml occurred after injecting 1.1 ± 0.1 ml of air. Intraballooon pressure was 294 ± 34 mmHg at this point; it decreased slightly with further inflation, indicating balloon expansion, as previously noted by McDonald et al.11 On the other hand, incremental deflation appeared to be more gradual, with the smallest balloon volume before zero being 0.22 ± 0.05 ml (P < 0.001 compared with the initial inflating volume). A representative pressure-volume tracing of one catheter balloon is shown in figure 5.

**DISCUSSION**

We believe our technique of measuring catheter tip distance11 is accurate and justified. As assessed by computed tomographic examinations of the thorax, the distance between the superior vena cava and right pulmonary artery is small. For that reason, and because there was no rotation of the patient's body and no ventilation during fluoroscopy, we believe that parallax was so minimal that it did not influence our results (R. H. Choplin, personal communication). Although a bony landmark could have been used, the catheter intersec-

**Fig. 4.** Peripheral movement of the catheter tip during balloon inflation and deflation. Group 1 patients tended to center near the solid line (line of identity). In contrast, group 2 patients were shifted rightward, indicating greater tip movement during balloon deflation than inflation.
Most suggested causes of catheter tip perforation of the pulmonary artery have implicated balloon inflation. According to one theory, eccentric inflation of the balloon forces the catheter tip into the vessel wall, damaging the wall and causing eventual perforation. However, it is more likely that the location of the catheter tip influences the configuration of the balloon during inflation. Shin et al. found that the greatest incidence of eccentric balloon inflation occurred when the catheter tip was located in the lung periphery, while concentric balloon inflation occurred if the tip was more proximally placed. Further studies are needed to determine whether catheter balloons tend to inflate concentrically or eccentrically in patients with pulmonary hypertension.

Pulmonary hypertension is thought to play a primary causative role in catheter-induced pulmonary artery perforation. In the present study, we found that catheters in patients with elevated PAP required a 4.5 ± 0.7 cm greater insertion distance to obtain a pulmonary artery occlusion pressure, and that they tended to have a more peripheral location at baseline than catheters in patients with normal PAP. The effect of severe pulmonary hypertension on catheter tip migration during balloon deflation with respect to vessel caliber remains unknown. The role of pulmonary hypertension as an etiologic mechanism of catheter-induced pulmonary artery perforation may be one of causing the catheter tip to be located in the lung periphery, as suggested by Lemen et al.

We believe that the characteristics of balloon inflation and deflation represent a primary factor causing distal tip migration into the pulmonary artery. We found that the most distal catheter tip location occurs during balloon deflation in all patients. Of greater significance is the observation that the catheter tip migrates further toward the lung periphery during deflation in patients with elevated PAP. However, since our group 2 patients had only moderate pulmonary hypertension, and since catheter-induced perforation of the pulmonary artery was not seen in any patient, our comments remain speculative.

Observing that, during rapid balloon inflation, the catheter tip acted like a "spear" against the vessel wall, Haapama et al. recommended gradual balloon inflation to eliminate this problem. However, since both a study by McDonald and Zaidan and the in vitro portion of our study showed instantaneous balloon inflation despite the gradual incremental injection of air, we do not regard slow balloon inflation as protective against catheter perforation of the pulmonary artery.

Because the discrepancies between instantaneous inflation and gradual deflation of the catheter balloon are
most likely to occur in vivo, we believe these discrepancies account for the distal catheter tip migration found during passive balloon deflation. Gradual balloon deflation would cause smaller balloon volumes and allow the catheter tip to be propelled into the lung periphery. The spearing effect discussed by Haapaniemi et al. may occur more often with balloon deflation rather than inflation, and appears more likely to occur when PAP is elevated. Since the balloon no longer covers the catheter tip during deflation, the exposed tip could directly traumatize smaller pulmonary vessels. As can be seen in figure 3A, in a patient with pulmonary hypertension, the catheter tip seen on a radiograph obtained before balloon inflation may be located medially. However, as can be seen in figure 3B, the catheter tip protrudes into the lung periphery during deflation, which we believe enhances the risk for perforation.

The size of the right ventricular cavity represents another potential influence upon the extent of distal tip migration during balloon deflation. It seems likely that, despite similar baseline locations of the catheter tips in both groups, patients in group 2 had larger catheter loops in the right ventricle, since they required greater catheter insertion lengths. In addition, right atrial pressure was higher in group 2 than in group 1 patients. Consequently, we do not know whether distal tip migration in group 2 patients during balloon deflation depended solely on pulmonary artery pressure, or on shortening of a large right ventricular catheter loop. In both. Nevertheless, the catheter tips in both groups returned to baseline position with respect to the zero point following balloon deflation. If shortening of the ventricular catheter loop is important, maintaining the smallest possible catheter loop in the right ventricle may minimize the amount of tip migration during deflation. Catheter repositioning may be required before obtaining a wedge pressure recording. This could be accomplished in a sterile fashion using a plastic protective shield. In addition, active deflation of the balloon or disconnecting the syringe may affect the amount of tip migration. Rapid deflation could minimize the time of smaller balloon volumes and allow equilibration of pressures on either side of the balloon to occur as quickly as possible. Both suggestions remain speculative and warrant further study. It should be kept in mind that rapid balloon deflation might cause excessive balloon trauma and rupture.

To summarize, we found that the pulmonary artery catheter tip locates more distally in the lung periphery during balloon deflation than during balloon inflation. Moreover, the distal tip migrates further during deflation in patients with elevated PAP than in patients with normal PAP. These findings may be important to an understanding of the cause of catheter perforation of the pulmonary artery.

The authors express their appreciation to Robert H. Choplin, M.D., for the use of fluoroscopic and radiographic equipment; to Ellen Tommasi, B.A., for her technical assistance; and to Betty McDonald for her secretarial skills.

REFERENCES