Cardiac catheters for diagnosis and treatment of venous air embolism

A prospective study in man

ROBERT F. BEDFORD, M.D., WAYNE K. MARSHALL, M.D., ALBERT BUTLER, M.D., AND JOSEPH E. WELSH, M.D.

Departments of Anesthesiology and Neurological Surgery, University of Virginia Medical Center, Charlottesville, Virginia

Controversy continues to surround the use of right heart catheter monitoring for venous air embolism during neurosurgical operations performed in the seated position. Recovery of air from the right heart has been shown to be valuable in resuscitating patients from near fatal air embolism,7 and, although pulmonary artery pressure (PAP) changes clearly reflect the severity of air embolism and its successful treatment,4 the insertion of right heart catheters is not without hazard.8 Thus, some experienced clinicians doubt that the benefits from invasive monitoring outweigh the disadvantages.6 In an effort to address these issues, we have utilized Swan-Ganz right heart catheterization in conjunction with the noninvasive techniques of end-expiratory CO2 analysis and precordial Doppler ultrasound monitoring for venous air embolism in all patients undergoing neurosurgical procedures in the seated position at our institution.

Clinical Material and Methods

During a 3-year period from 1977 to 1980, 100 consecutive patients scheduled for elective cervical laminectomy or posterior fossa exploration in the seated position underwent percutaneous Swan-Ganz pulmonary artery catheterization* under pressure

* Swan-Ganz pulmonary artery catheter manufactured by Edwards Laboratories, 17221 Red Hill Avenue, Santa Ana, California.
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![Graph showing end-tidal CO₂ concentration (FETCO₂), right atrial (RAP), pulmonary arterial (PAP), and systemic arterial (SAP) pressures during an episode of air embolism with nitrous oxide (N₂O) challenge. No arterial hypotension occurred because the site of air entainment was rapidly occluded. Only small volumes of air were recovered from the right atrial and pulmonary arterial catheters, yet the effect of N₂O inhalation indicates that air bubbles are present in pulmonary arterioles.](image)

waveform control via either an internal jugular or antecubital venous approach. After the patients were positioned for operation, simultaneous measurements of right atrial and pulmonary capillary wedge (left atrial) pressures were performed. Additional monitors consisted of a radial arterial catheter, an end-tidal CO₂ (FETCO₂) analyzer, and a precordial ultrasonic Doppler device. The position of the Doppler monitor over the right atrium was verified by the change in Doppler signal caused by a 5-ml bolus injection of physiological saline solution through the right atrial port of the Swan-Ganz catheter. Cardiovascular pressures and FETCO₂ were recorded continuously with a Brush polygraph. General anesthesia was maintained with 60% to 70% nitrous oxide in oxygen and pancuronium, 0.1 mg/kg administered intravenously, and was supplemented with a variety of intravenous or inhalational agents as needed.

Whenever air embolism was diagnosed by an audible change in Doppler signal, an increase in PAP, or a decrease in FETCO₂ concentration, nitrous oxide was discontinued, the incision was packed with saline-soaked sponges, and vigorous attempts were made to recover air from both the pulmonary arterial and right atrial ports of the Swan-Ganz catheter. When arterial pressure fell by 20%, ephedrine (25 mg) was given intravenously. Repeated episodes of air embolism were treated by lowering the head of the operating table until no further air entrainment was detected.

When PAP and/or FETCO₂ values returned toward normal after air embolism, nitrous oxide was reintroduced into the anesthetic mixture (Fig. 1). If residual air in the pulmonary circulation was detected by an increase in PAP or decrease in FETCO₂, further operative manipulation was deferred until these values normalized. At the end of surgery, catheters were promptly removed and hemostasis was maintained by direct compression for 10 minutes.

Changes in measured parameters during air embolism were compared with pre-embolism values. Sta-

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† LB-2 end-tidal CO₂ analyzer made by Beckman Instruments, Inc., 2500 Harbor Boulevard, Fullerton, California, and Doppler ultrasonic device made by Parks Electronics, Beaverton, Oregon.

‡ Brush polygraph (Model 440) manufactured by Gould, Inc., Instrument Systems Division, 3631 Perkins Avenue, Cleveland, Ohio.
TABLE 1
Detection of air emboli in 100 operations in a seated position

<table>
<thead>
<tr>
<th>Monitor</th>
<th>Episodes Detected</th>
<th>No. of Operations</th>
<th>Mean Change</th>
</tr>
</thead>
<tbody>
<tr>
<td>precordial Doppler</td>
<td>80</td>
<td>35</td>
<td>—</td>
</tr>
<tr>
<td>pulmonary artery</td>
<td>36</td>
<td>19</td>
<td>11.4 torr ± 1.9 SEM</td>
</tr>
<tr>
<td>pressure</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>end-tidal CO₂</td>
<td>30</td>
<td>16</td>
<td>1.2% ± 0.1 SEM</td>
</tr>
</tbody>
</table>

Statistical analysis was performed using Student’s t-test for paired data with p < 0.05 regarded as significant.

Results

Precordial Doppler monitoring detected a total of 80 episodes of air embolism in this series, whereas PAP changes occurred in 36 episodes, and FET\textsubscript{CO₂} changed in only 30 (Table 1). No systemic arterial hypotension or cardiac arrhythmia occurred, however, unless both PAP and FET\textsubscript{CO₂} changed. Because of the extreme sensitivity of the Doppler monitor, then, a considerable amount of operative time was expended treating episodes of air embolism which were of no hemodynamic consequence. Furthermore, on only three occasions did the Doppler device’s sensitivity permit detection of air embolism more than 30 seconds before PAP or FET\textsubscript{CO₂} changes developed, and none of these episodes was associated with systemic arterial pressure changes.

In addition to problems with the Doppler monitor’s extreme sensitivity to very small air emboli, we also observed occasional problems with false-negative diagnosis. In three instances, the operating room staff failed to detect Doppler “air sounds” while the diagnosis of air embolism was being made from obvious changes in PAP, FET\textsubscript{CO₂}, and aspiration of air from the Swan-Ganz catheter. Failure of the Doppler monitor to detect air embolism may have been caused by slippage of the ultrasound transceiver during long operations, or perhaps was due to entrainment of such fine streams of air bubbles that there was no audible increase in the reflected ultrasound signal.

During air embolism, we observed a definite correlation between the hemodynamic consequences of the embolus on systemic blood pressure and the magnitude of the changes in PAP and FET\textsubscript{CO₂}. Although six small episodes of air embolism caused small PAP changes without altering FET\textsubscript{CO₂}, as the severity of the emboli increased these two modalities agreed well for clinical purposes (Fig. 2). Furthermore, both PAP and FET\textsubscript{CO₂} were quite sensitive to nitrous oxide challenge following air embolism, and thus were equally useful not only in detecting the severity of the embolus, but also in indicating when it was resolving and when it was reasonable to continue the operation (Fig. 1).

Recovery of air from the Swan-Ganz catheter proved to be disappointing. Only small volumes (2 to 5 ml) of air could be recovered from either the right atrium or the pulmonary artery during embolism which caused increased PAP. Even in episodes with systemic hypotension, a maximum of only 20 ml of air could be retrieved. Despite limited recovery of air, hemodynamic parameters improved rapidly with packing of the incision, ephedrine therapy, and discontinuation of nitrous oxide.

An unexpected finding in this series was that in 29 of the 100 patients placed in the seated position, right atrial pressure exceeded pulmonary capillary wedge pressure, and, presumably, left atrial pressure (6.9 torr ± 0.5 SE versus 3.5 torr ± 0.5 SE, p < 0.001). These patients thus were at risk for developing paradoxical air embolism if they had a probe-patent foramen ovale. In fact, one patient did go on to sustain a dense hemiparesis with multiple cortical infarcts following a relatively modest episode of intraoperative venous air embolism.

Complications resulting from Swan-Ganz monitoring were infrequent and minor. Except for small hematoma formation at the site of cannulation, the
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only other problems were one asymptomatic carotid artery puncture with a No. 16 introducing needle and one episode of antecubital vein thrombophlebitis that resolved spontaneously. There were, however, five patients in whom we elected to forego Swan-Ganz cannulation after several attempts at large-vein catheterization proved unsuccessful. None of these patients subsequently developed evidence of air embolism.

Discussion

Several recent studies have demonstrated that the primary pathophysiological event in venous air embolism is not "air lock" in the right heart, but, rather, intense vasoconstriction of the pulmonary circulation which results in a marked increase in pulmonary vascular resistance.\(^1\) Small volumes of air produce increased PAP and ventilation-perfusion mismatch which is reflected in a reduced \(FET_{CO_2}\) and/or increased venous admixture. Since most of the air causing hemodynamic impairment is distributed to the periphery of the lung, it is understandable that only minimal amounts of air could be recovered from either right atrial or pulmonary arterial catheters, particularly since the diagnosis was made and air entrainment was stopped before the capacity of the pulmonary vasculature had been exceeded.

Our results indicate that a pulmonary artery catheter is of no greater value for the diagnosis of air embolism than noninvasive \(FET_{CO_2}\) monitoring, since these two modalities agree well during air embolism that causes systemic hypotension and both also promptly indicate the severity of the embolus and when treatment has been successful. As a route for recovery of air, a Swan-Ganz catheter appears to be no more effective than a right atrial catheter, although in this series recovery of air was never a major factor in normalizing hemodynamics. Other series, in which large volumes of air have been aspirated from the right heart, have relied on Doppler monitoring alone and may have missed episodes of "silent" air embolism such as the three we diagnosed with PAP and \(FET_{CO_2}\) monitoring when there was no audible change in Doppler signal.

We have found two unique uses for the Swan-Ganz catheter during seated neurosurgical procedures. In the case of hemodynamic changes induced by brainstem or spinal cord manipulation (Fig. 3), it is possible to rule out air embolism as a causative factor because PAP does not increase in this circumstance, even though both blood pressure and \(FET_{CO_2}\) may fall rapidly. Second, the ability to measure both right and left atrial pressures permits identification of those patients who may be at risk for developing paradoxical (systemic) air embolism through a probe-patent foramen ovale if an air embolus should occur. Since a probe-patent foramen ovale is present in 20% to 30% of the general population,\(^3\) it seems prudent to avoid the hazards of paradoxical air embolism by utilizing the prone position in those patients whose right atrial pressure is higher than pulmonary capillary wedge pressure when they are placed in the seated position.

Use of Swan-Ganz catheter monitoring for neurosurgical procedures performed with the patient seated will probably remain controversial for the foreseeable future. Not only does insertion require more time and/or personnel than may be available during a busy surgical schedule, but the list of complications unique to use of the Swan-Ganz catheter continues to grow. Ventricular fibrillation,\(^2\) knotting of the catheter, rupture of the pulmonary artery, pulmonary infarction,\(^5\) cardiac valve damage,\(^9\) and perforation of the heart\(^8\) have all been reported, even though in our hands we have found the technique to be reasonably safe and no more difficult than central venous cannulation under electrocardiographic control.

Although precordial Doppler monitoring for air embolism is quite sensitive, it is nonquantitative and occasionally in error. Our data indicate, however, that when Doppler monitoring is combined with continuous \(FET_{CO_2}\) analysis and/or PAP monitoring, an accurate diagnosis of venous air embolism can be made and corrective surgical measures can be taken before

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**Fig. 3.** Record of arterial hypotension and decreased end-tidal CO\(_2\) (\(FET_{CO_2}\)) concentration due to depressed cardiac output from brain-stem manipulation. The absence of a change in pulmonary artery pressure (PAP) rules out the possibility of air embolism as a cause for the hypotension. RAP = right atrial pressure; SAP = systemic arterial pressure.
the right heart and/or pulmonary outflow tract fill with air. Furthermore, despite the invasive nature of Swan-Ganz monitoring, we have found it to be valuable in identifying patients at risk for developing paradoxical air embolism and for differentiating hemodynamic changes caused by brain-stem manipulation from those caused by air embolism.

References